THE RELATION OF DUST TO "COAL MINERS LUNG".

by

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INTRODUCTION.

This work was carried out in an industrial practice on the North-East coast of England over a period of three years.

This district is essentially a coal mining area and the coal mined is of the Gas and Steam types. Normally it was shipped to Baltic Ports or Italy. Since the war, however, the coal has been usually shipped to London.

The population has been established here for over a century and is of mixed Durham and Cornish stock, the Cornish element having arrived some seventy years ago when there was a slump in the Cornish Tin Mines. They are nearly all third or fourth generations of the original inhabitants. The same name recurs again and again amongst them and relationship and marriage ties bind the whole neighbourhood closely together. There has, I think, been some in-breeding to a certain limited extent. The people are conservative in their outlook and are rather inclined to resent changes; most of them have worked in the same coal mine from their youth up. Thus their occupational and family histories are easy to elicit and the influence of the local mine dust can be estimated fairly accurately.

This work, therefore, is an attempt to assess firstly, the damage, if any, done to the lungs of coal miners by the mixed dusts and fumes encountered during the course of their work, and secondly, the relation, if any, between coal miners lung and pulmonary tuberculosis. The cases

were all those met with in the course of daily practice.

There has been much speculation as to the effect produced by the continued inhalation of dust and fumes in bituminous coal mines on the workers' lungs and whether or not the dust so inhaled leads to pulmonary fibrosis of the Silicotic type. Some workers maintain that exposure to such dust leads to a definite pulmonary fibrosis and therefore damage to the lung, whilst others maintain that not only is the dust harmless but actually beneficial by exerting a protective influence against the tubercle bacillus.

I may state at the outset it is my belief that continued exposure to this dust does lead to a type of pulmonary fibrosis, and also, that it causes a chronic Bronchitis, Emphysema and eventually even a Bronchiectasis.

There are few coal miners of over 40 years of age who can show a clear lung field on radiological examination and the usual X-Ray report of pulmonary fibrosis or peri-bronchial infiltration indicates the first step in a series of changes which, once initiated, progresses slowly to a terminal condition of extensive fibrosis or Bronchiectasis with resulting strain and damage to the right side of the heart.

It is also my belief that the long defunct "Miners Asthma" of 60 or more years ago is returning. My reasons for believing this will be related later.

Coal is the chief mineral wealth of this country and

and its importance will be still more apparent after hostilities cease. The bye-products which can be extracted from coal during its distillation are manifold, and these are the very products which have hitherto largely been wasted. Petroleum, drugs and plastics to mention only a few, are some of the products of coal. It therefore seems to me to be of some importance, that any of the hazards of coal mining should be thoroughly ventilated and brought to light. The dust hazard is one of these especially in its relation to pulmonary disease. A great deal of work has been done in this connection by the Medical Research Council which has published its findings in two volumes entitled - Chronic Pulmonary Disease in South Wales Coal Miners 1943 - H.M.Stationery Office. This work has, however, been entirely in connection with Anthracite Miners in whom the incidence of pulmonary disease is extraordinarily high. This thesis, is, on the other hand, entirely in connection with Bituminous Coal Miners and is my own personal experience after nearly 10 years practice amongst them.

In each of the cases which I studied, a clinical examination was made and occupational and medical histories taken. The sputum also was examined and then the clinical examination was correlated with a radiological examination - if such was possible. An analysis was also taken of the stone dust used in the mine and an examination made of the part, if any, played by nitrous fumes on the worker's lungs. These last are present in the coal mine after shot-firing has been taking place.

The diagnosis of Pneumoconiosis of coal workers was only made after :-

(1) an occupational history which indicated that the man had been exposed to both coal and stone dust in the course of his work over many years. Such occupations were Coal Hewers, Coal Cutters and Drillers, also Fillers, Firemen or Deputies and Stone-men or Stone Workers. This last is a worker who widens and heightens the roadways so that the coal tubs can pass through. He is thus exposed to a greater extent to stone dust in addition to coal dust.

(2) a careful clinical examination for symptoms and physical signs such as breathlessness, cough, tachycardia, emphysema, decreased air entry or prolonged expiration. Other signs such as clubbing of the fingers was also noted.

(3) a Sputum Examination.

(4) Radiological examination when possible.

In a recent article in the Journal of Industrial Medicine 1942 pp 470-473, Flinn Seifert and Brinton examined 507 coal workers in a bituminous coal mine and found 16 cases of Anthraco-silicosis amongst them. These cases were not advanced and were not seriously disabled. Only one case of moderately advanced Anthraco-silicosis was found. Personally I found amongst my own cases that the men affected were definitely seriously disabled, so much so, that they were unable to follow any occupation at all.

They were disabled by factors which could be ascribed to the dust hazard. Only one of my own series could be described as a moderately advanced case and he developed Pulmonary Tuberculosis with a positive sputum. I have described him as suffering from Pneumoconiosis of coal workers, as before he developed a positive sputum at the age of 63 he had previously been X-Rayed and the report was "extensive reticulation of the lung field". Although this seems to indicate a relationship between dust reticulation and pulmonary tuberculosis, yet this relationship was not seen in subsequent cases.

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List of References:

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HISTORICAL SURVEY.

When I was an undergraduate student of clinical medicine, the term Pneumoconiosis was used to lump all the dust diseases together and they were ascribed to any and every form of injurious dust of which pulmonary fibrosis sums up the pathological findings and tuberculosis the morbid result. Silicosis was considered the dangerous disease that it is, while Anthracosis was considered to be harmless and benign.. When, however, I became a general practioner in a mining district (in partnership with my late father) I found that Anthracosis was not so benign and that many miners suffered from disabling pulmonary complaints including Emphysema, Bronchitis, Asthma, Bronchiectasis and Silicosis itself. I propose, therefore, to discuss the relation of these diseases to the dust or dusts inhaled by the coal miner in the course of his employment.

The history of dust diseases goes back almost to the dawn of history - to the Father of Medicine himself, for Hippocrates in his Epidemics speaks of the metal digger (metalliferous miner) who he says "has his right hypochondrium bent, a large spleen and a costive belly, he breathes with difficulty, is of a pale wan complexion, and is liable to swellings in his left knee". There are several points of interest here - the constipation (costive belly) for which the person exposed to dust inhalation takes aperients, also the shortness of breath which is often the first and maybe the only sign and finally the swelling of the left knee, which is in all probability

a prepatellar bursitis - what we in Durham call a "beat knee".

Again Johannes Agricola in his work De Metallien (1557) speaks of the necessity of ventilating the mines and states that when the dust is dry it ulcerates the lungs and produces consumption. He states that in the Carpathians there are women who have married seven husbands all of whom have died of the disease (Silicosis and Tuberculosis). Agricola makes the difference between these cases and the combers of silk cakes and sifters of corn in whom it dries up the throat and lines the pulmonary vessels with dusty matter that causes a dry and obstinate cough "- all who live by this trade are short winded and seldom live to be old. They are seized with orthopnoea and at the last with dropsy." A good description indeed of Bronchitis and Emphysema with terminal myocardial failure. In these last two occupations, corn sifting and silk cake combing, note that he makes no mention of consumption . This is noteworthy because even to-day, as already mentioned, pneumoconiosis is ascribed to exposure to any and every form of injurious dust. These generalisations may have resulted from the publicity given to Professor Alison, who, when speaking of that modification of phthisis which occurred in middle and advanced life in those workmen who are much exposed to irritation of the lungs by fine powders says "I have reason to believe that there is hardly an instance of a mason regularly employed in Edinburgh living free from phthisical symptoms to the age of 50." The newer parts of Edinburgh were then being

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built of Craigleith sandstone. This industrial disease was shown by Gulland to persist among Edinburgh masons in this century. (Collis)

Thackrah noted that among Lead Miners the men are injured by working ore in sandstone but not when the ore is in limestone.

In connection with Alison's remarks on "that modification of phthisis which occurs in middle and later life" I would draw attention to the fact that coal miners are exposed to an excessive degree of a dust composed of Calcium Carbonate, Calcium Sulphate with 1% of Silica Dioxide. This dust is deliberately thrown down at the coal face and the compressed air machines stir it up as also do the men's feet and the passage of tubs etc. This operation of "dusting" is done to preclude any possibility of an explosion after a "shot" is fired. The shot-firing also disturbs this dust and creates more, and to it is added the nitrous fumes after the explosion and the great concentration of coal dust which is also set up. The dust used for this "stone-dusting" is frequently obtained from stone sent out of the mine to the surface where it is crushed for both brick making and "dusting"purposes. Consider this and the fact that the miners are undergoing physical exertion and therefore have their mouths open, and one can realise that chronic pulmonary disease is excessively common in coal miners.

The stone dust used is fine and floury and one can understand how it is that a person such as I have described

in Case 1. can do nearly 50 years work underground and then develope extensive reticulation of both lungs and be found to have a sputum positive to the tubercle bacillus. The danger of this type of case working in a confined space underground can also be imagined. As before mentioned, this dust is fine and floury and below five microns in size. It can be conveyed by air and can be inhaled into the air passages which are specially constructed to impede the inhalation of dust, but this dust is so fine that it gains entrance even to the alveoli. The result in some cases is Asthma, and it appears to me to be real spasmodic Asthma.

That the inhalation of dust causes Asthma is well known. Agricola mentions it as occurring as the result of dust inhalation among Carpathian miners; Diembrock mentions its occurrence among stone masons. Greenhow also mentions it as occurring in Cornish Tin Miners, who he states -"for the most part become asthmatical about the age of 40."

A point of some interest is that these cases seem to be worse on a Monday morning or after a day or two off work. They are "tight in the chest" the next morning but that it gradually works free as the day goes on. The extra strain on the lung usually causes Emphysema.

About three generations ago, Miners Asthma was very common among coal miners. "Then in every little mining village" writes the Secretary of a Miners' Association," "there was a contingent of old miners past work on account of difficulty in breathing" - a stage reached usually between the ages of forty and fifty.

This is confirmed by Greenhow, who tells of the ravages wrought in his day among coal miners in South Staffordshire by Asthma - "some miners retain their health till an advanced period of life but the greater number suffer more or less from asthmatical symptoms before attaining the age of fifty, and many break down or are disabled at forty to fifty years of age -- a miner is usually an old man at fifty." The same observer mentions the prevalence of the disease in the South Wales coal-field and states that the miners blamed the fumes from explosives. He contrasts the condition with that seen among lead miners and tin miners as one with Emphysema more frequent with less pneumonia and while associated with chronic bronchitis not associated at an early period of life with slight dyspnoea.

In America Drs. Wainwright and Nichols report that in 1894 - 1904 Asthma accounted for 7% of all deaths among coal miners as compared with 1.6% amongst all occupied males. They further report that the condition as seen amongst coal miners is one of Chronic Bronchitis and Emphysema.

I would remark, however, that while Chronic Bronchitis and Emphysema are both very common pulmonary complaints amoungst coal miners, yet there are several cases of undoubted spasmodic Asthma which I can only conclude are cases of this long defunct Miners Asthma returning again. Case 4. is a case in point - a coal cutter on the arc-wall where dust is everywhere, both stone and coal, and conditions are very much enclosed. He worked thus for about fourteen years and then had to give it up and come to the surface

on account of these frequently recurring attacks of spasmodic asthma. He has been skin tested and was found to be sensitive to mixed inhalants. He has been desensitized with mixed inhalants Bencards, with peptones also but with little improvement.

Collis in his Milroy Lectures states "synchronous with improved ventilation in the mines the disease has disappeared in this country. The opportunity of ascertaining what was the determining cause has gone, and also of obtaining a good clinical description of the condition, but the following note sent me by Dr.J.Taylor of Chester-le-Street, suggests that coal miners asthma was true spasmodic asthma. Dr.Taylor writes - "One case only do I recollect - the breathing was carried on by the respiratory muscles of the chest, and the diaphragm did not enter much into the mechanism of inhalation. The patient suffered at all times, but a windy day always seemed to initiate an attack."

If the supposition is correct that coal miners asthma was true asthma, possibly the miners were correct in ascribing it to fumes, especially as Oliver has pointed out they would be nitrous fumes, and in that case the disease should not be classed as a true pneumoconiosis. The disease, however, common as it was, has passed from our midst and conjecture as to its cause is idle."

I have quoted Collis at some length, chiefly because it is my contention that Miners Asthma is returning, but I would like to comment on his beautiful command of English.

That Miners Asthma is returning to the coalfields is

not my opinion only, it is shared by many others. At a recent meeting in Newcastle-on-Tyne which I attended it was noted there too by the Emeritus Professor of Medicine in Durham University Dr.Hume. At this meeting which was only a small gathering and was addressed by Dr.C.L.Sutherland, Chief Medical Officer to the Silicosis Board, it was noted that there had been a return, or a threatened return of the old Miners Asthma.

The reasons for the return of this condition have been gone into later - it would suffice to say that probably the increased employment of mechanical compressed air contrivances may play a part.

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REVIEW OF PREVIOUS PUBLISHED WORK.

Thackrah 1831 in his book upon occupational ill health, recognised that colliers were subject to asthma. It should be pointed out, however, that the term Asthma was used to designate any form of pulmonary disease attended by dyspnoea arising from their occupation. In 1836 a number of fatal pulmonary cases were described by Thomson. He stated that the chief symptoms were dyspnoea and cough with sputum at first mucoid and later black - the characteristic 'black spit' The progress was slower, emaciation less severe and absence of hectic fever differentiated it from Tuberculous disease. This description could be applied to several cases that I have seen here, including two which have eventually died of Tuberculosis.

Again Cox 1857 writing on diseases of coal miners divided their complaints into (a) Asthma (dyspnoea) (b) Bronchitis present with the asthma, (c) black spit and (d) Tuberculosis consumption. From these extracts it is evident that chronic pulmonary disease was present in coal miners in the first half of the last century. It is not mentioned whether one clinical entity was accepted or whether there was a group of entities. Most writers seem to have agreed,however, that coal dust or coal dust and stone dust were the immediate causes.

Greenhow stated that 'miners asthma' was a chronic bronchitis analogous to that of Cornish miners but with emphysema more frequent, and pneumonia, followed by breaking down of lung tissue, less common.

I would like to add at this point, that frequent attacks of pleurisy and atypical pneumonia are a common feature in the history of the cases that I have examined myself. Also, on the infrequent occasions of an autopsy in an old retired miner, one almost always finds a very thickened pleura - so much thickened, indeed, as to cause a dull percussion note on examination.

Later, however, towards the end of the last century, a curiously complacent attitude seems to have been uniform amongst all observers. For example, in Allbutt and Rollestons System of Medicine (1909) Sir Thomas Oliver states that although there was a high incidence of pulmonary trouble among coal miners in the past, that now conditions had so much improved, that coal mining was a healthy occupation. He further stated that 'Coal Miners Phthisis' or Anthracosis is on the decline in this country.

Further, in 1914, in the Milroy Lectures on the Hygienic Aspects of Coal Mining, Shufflebotham stated that prior to the Coal Mines Regulation Act 1911, which, among other things, required improved ventilation and control of shot-firing, one of the principal diseases from which coal miners suffered had been fibrosis of the lungs or miners phthisis as described by Greenhow, but that at the present time in Great Britain, fibrosis of the lungs among miners could be said to be practically non-existent. He quoted certain evidence given by physicians before the Departmental Committee on Industrial Diseases in 1906. He further stated, however, that infiltration of the lung with coal

dust was still prevalent although it did not seem to entail disablement. Shufflebotham called this infiltration 'Anthracosis' using the term differently from the earlier writers. Indeed he seems to have used it in the sense which I think is correct, if I may so presume, for although like all other students of my time I was brought up to believe that Anthracosis was a harmless disease, I have since had reason to revise that opinion. Anthracosis is a disabling disease and it is only recently, that out of the confusion of radiological reports such as 'peribronchial fibrosis' 'peribronchial infiltration' diffused mottling etc, the condition of 'dust reticulation' has emerged about which more later. It does seem worth emphasizing, however, that Shufflebotham was correct in describing this infiltration of the lungs with coal dust as Anthracosis, although, I think he was wrong in stating that it did not cause any disablement, for I think that it causes a fibrosis along the peribronchial lymph channels which, in turn, causes a Bronchiectasis or bronchial dilatation with symptoms of dyspnoea.

In 1922 Mavrogordato stated similar views and in the following terms "It is well known now that colliers do not develope 'miners phthisis' and that they are very well placed with regard to respiratory diseases in general... .. The collier was not always free from chest trouble; colliers asthma was a by-word and old established pathological museums have specimens of miners lungs showing fibrosis."

My experience has been that most miners lungs show

evidence of fibrosis - again we come upon such terms as peribronchial fibrosis, in other words the coal dust acting as an irritant causes a fibrosis in men who have been exposed to it for many years.

More recently a working miner himself has written of "our greatest fear Silicosis and its black twin Anthracosis." This again is using the term Anthracosis in its malignant sense as opposed to the earlier benign idea of Anthracosis.

Dr.J.S. Haldane's views have held great weight since they were published some forty years ago. He had previously associated the excessive death rate from 'miners phthisis' among Cornish Tin Miners with the inhalation of stone dust by the rock drillers (Haldane, Martin and Thomas 1904). Also in 1909 he had brought to the notice of the Royal Commission on Health and Safety in Coal Mines the possible danger of similar rock work in coal mines. Haldane also noticed that there was a relative excess of deaths among the older age group of miners from bronchitis as a whole.. He suggested that this relative excess might be due to physical exertion and consequent over breathing and that the much greater amount of bronchitis some decades previously had been due to worse ventilation which caused Co_2 and increased respiration. This strikes a personal note with me as my own grandfather had been subject to attacks of Bronchitis and Asthma from the age of fifty and finally died of this condition. I should add that he had been Manager of a coal mine all his life and he attributed his condition to defective ventilation in his early years in the mines.

Haldane,however, adduced evidence from which he argued the present relative excess, of Bronchitis, among coal miners was not due to dust inhalation as he stated "there is no real statistical evidence of harm resulting from the inhalation of coal or shale dust in the quantities breathed by miners and a strong presumption that the dust they breathe protects them from serious dangers." He recapitulates "coal mining in this country is a very dusty occupation but there is no evidence in the mortality returns for either young or old miners, that dust inhalation has hitherto caused appreciable danger to life in British coal mining."

It should be borne in mind, however, that'Bronchitis' is a crude heading and that many cases (before 1929) would be returned as Bronchitis which, to-day, would be diagnosed and certified by the practitioner as Pneumoconiosis or Silicosis, for even just prior to 1929 X-Rays of the chest were not as lightly taken as to-day. In addition there has been a gradual change from the heading of Bronchitis to that of Chronic Interstitial Pneumonia. This latter contains all the occupational lung diseases returned as such. A second criticism might be that overbreathing due to increased CO_2 or to physical exertion might result not only in overstretching the lungs but also to increased inhalation of dust.

Later in 1931, Haldane held to his original thesis that "it seems practically certain that inhalation of coal dust or shale dust must cause Bronchitis." He still held that the mortality among older miners from Bronchitis was connected with muscular exertion and that even if no dust

inhalation accompanied coal mining the mortality from Bronchitis would probably still be as high." He further affirmed that there was no evidence apart from very exceptional conditions that coal miners suffered from Silicosis. He did suggest, however, that in a lung previously damaged by Bronchitis, Emphysema or Phthisis, in which the elimination of inspired dust would be interfered with so that local accumulation of dust might occur, which in turn, might produce a scattered fibrosis giving an X-Ray appearance similar to Silicosis.

To the end of his life Haldane maintained that many cases officially diagnosed as Silicosis in coal miners were previously cases of either Bronchitis or ordinary Phthisis - the collections of dust with such extra fibrosis as is found in X-Ray examinations being due to paralysis by bronchitis of the normal process of dust elimination.

Kettle, however, held that there was no evidence that chronic bronchitis interfered with the elimination of dust inhaled into the lungs.

Cummins also made the point that Bronchitis was often a label of convenience obscuring a case of Anthraco-silicosis or Pneumoconiosis and that deductions from mortality statistics of 'Bronchitis' might therefore be fallacious.

This last, is in my opinion, a point of some practical importance, as one often comes across cases of what appear to be chronic Bronchitis and who manage to keep going on some form of light work. Yet when investigated, these cases reveal evidence of Pneumoconiosis. If these cases are off

work they are labelled Chronic Bronchitis for if Pneumoconiosis was put on their certificates, endless examinations and a prolonged period off work would be the result, so that the patient is frequently loathe for any other diagnosis to be mentioned. Added which the diagnosis of Pneumoconiosis cannot lightly be made - certainly not without X-Ray evidence, and clearly patients are frequently unwilling to go to the trouble of X-Ray examinations if they feel fairly well and can manage light work.

Thus by the end of the War of 1914-1918, Anthracosis had come to be considered by many as harmless and a relic of departed pathological conditions associated with faulty mining conditions of the past. But it was about this time that radiography began to have an increasing use in the diagnosis of chest diseases. Thus in 1925, attention was drawn by X-Ray examinations to serious lung disease among coal workers in the Somerset coal mines - a condition similar to Silicosis was diagnosed. At the same time radiological and clinical examinations were carried out on one hundred and thirty workers in the South Wales coal-field. In these cases marked alterations were found as the X-Ray films included mottling and diffuse shadows. These were very similar to those found in cases of Silicosis. The incidence was found to increase with length of employment. It was found that many of the cases were quite able to carry out their employment but they were considered to represent a definite impairment of lung structure and to involve an increasing respiratory disability, for example, by shortness of breath. This last is always a feature and is present in

all my series of cases. It is also a frequent complaint among coal miners who do not show any other sign or symptom and it seems to me that dyspnoea is the first symptom and it is caused by an Emphysema (usually basal in origin) caused by fibrosis and sustained high intra-alveolar tension by men undergoing strenuous physical exertion, as it is amongst the piece-workers that these conditions are commonest - or such is my experience.

Enid Williams 1933 examined one hundred miners - half anthracite and half non-anthracite of the older age groups., and found that though their physique was good, the great majority of them had chest symptoms (mainly dyspnoea, cough and sputum).

Similarly Jones (1936) differentiated between the classical Silicosis of rock workers which ran a rapid course, and the more common cases among miners which were difficult to diagnose without X-Ray examination. Many of these cases were formerly probably called Bronchitis and the chief cause of absenteeism among miners is Bronchitis with probably Lumbar Fibrositis a close second.

Symptoms of Pneumoconiosis:- The intial step in the process in all my cases seems to have been s state of breathlessness on exertion. This would appear in a hard-working man who had always previously been healthy. He would find that he was unable to keep up with his fellows, and had to stop and get his breath. They frequently complained that the dust was bothering them more than usual. The onset appears to be fairly acute, although it

also appears to take many years of exposure to produce it. The miner would then develope the sense of constriction in the chest with dyspnoea and slight cough. In most cases the man would be a Coal-cutter, Filler or Stone-man. Frequently, however, they were Shot-firers or Deputy-overmen. All these types of coal workers are exposed to great concentrations of dust. It is difficult to generalise on the dangerous types of work underground, however, for the men have changed their work so often. Sometimes they boast of having done every kind of work there is underground so that it is difficult to place the appropriate blame.

Reason for dyspnoea in coal miners over forty years of age:- This, as already noted, is exceedingly frequent in mining districts. A man may be of good proportions, well developed and well nourished and yet be exceedingly breathless. It is worthy of comment as one never sees it in text-books. All one finds on physical examination, is possibly a slight degree of Emphysema. It is rather remarkable that the men themselves do not appear to take undue notice of it, putting it down to repeated 'colds in the chest', to war service i.e. war gasses, or to accidents.

This dyspnoea is quite genuine. I have watched them in the street and when they were in ignorance of being observed, and they never varied.

They frequently have had an accident to the chest, usually a crushing accident, or they have an Influenzal attack or a 'cold' after which they do not seem to regain their strength. Eventually they find their way to the

Tuberculosis Dispensary, and on radiological examination, they are found to have some such condition as 'peri-bronchial fibrosis', 'peribronchial infiltration' 'diffuse mottling' or the reticulation stage of Silicosis.

Thus, as I repeat, the first sign is dyspnoea and the onset may be rapid although insidious in its initial stages. The incidence of these cases is rising and has risen considerably of late.

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COMPOSITION OF DUST IN COAL MINES.

Stone dust is used as a sort of prophylactic against explosions in coal mines, being sprinkled copiously all over the coal face and around the neighbourhood, so that the floor is about 2" deep with this stone dust and even the miners themselves are covered with it. Their feet kick it up and when the 'shot' is fired this stone dust is well mixed with coal dust. One has already said that if coal dust is innocuous, then coal dust admixed with stone dust is not, and produces the pulmonary disease from which miners suffer. It is always, it seems to me, the men who are employed in connection with shot-firing, such as Stone-men, Coal-cutters, Deputies and Fillers who develope these chronic pulmonary diseases and especially Anthraco-silicosis.

After shot-firing, while the air is still thick with this coal dust cum stone dust mixture, the men have to work clearing up the results of the explosion - their mouths are open, their intra alveolar tension is raised as they work, their respiratory rate is increased so that a heavy concentration of the mixed dust is inhaled.

Having obtained a sample of this dust which is used as above described, I had it analysed and the composition is as follows: Average Analysis.

	%
Calcium Carbonate	97.72
Calcium Sulphate	00.10
Magnesium Carbonate	00.70
Ferric Oxide	00.08
Aluminium Oxide	00.15

	%
Silica	01.05
Moisture	00.20

Thus the Silica concentration is just over 1%. It may be remarked that this is too small a concentration to be harmful, but several points must be remembered. In the first place, as I have already stated it is, in my opinion, the coal dust plus stone dust that is harmful, and the effect of the above mixture plus coal dust on the lung is unknown precisely. It is only for about eight years that this prophylactic stone dusting has been used, and, let it be noted, it is only recently that the return of the long defunct 'Miners Asthma' has been noted also. Whether the above mixture has any effect chemically on the human lung as Silica Dioxide has, is unknown. It may be that the mixture has a faculty for dissolving elastic tissue and replacing it by fibrous tissue thus producing the dyspnoea and pulmonary fibrosis so often met with in coal miners.

Another factor is, that although the human lung is specially designed for getting rid of inhaled foreign matter such as dust, yet there is a limit to this, and the continuous inhalation of dust over a period of many years must produce an embarrassment of the cleansing system of the lung with the lymphatic system packed with mixed dust, so that possibly both a chemical and mechanical irritation are set up producing reticulation of the lung fields.

Lastly one should remember that Coal-cutters either cutting through stone or Drillers boring through stone may have much heavier concentrations of Silica thrown back

in their faces from the exhaust of the compressed air operated machines. In the case of electrically operated machines, this is not such an important factor. They may bore through Silica rock and have large concentrations of Silica dust around them. Such men may of course develope a classical Silicosis and it is not about such cases that I am here concerned but rather with the coal miner who developes chronic pulmonary disease, whether it may be Emphysema, Bronchitis, Pulmonary Fibrosis, Bronchietasis or the reticulation stage of Silicosis itself.

In connection, however, with the coal miner who developes Silicosis and later Tuberculosis, it is frequently only the Tuberculosis which is diagnosed. The result is the man does not get any compensation and it is with the early diagnosis that Iam interested. It is in this connection that the Ministry of Fuel and Power have announced the publication of a report of the advisory committee on the treatment and rehabilitation of miners in the Wales regionnsuffering from Pneumoconiosis.

I should add that according to the Coal Mines Acts (Precautions against Coal Dust) Modification Order,1939 - the incombustible dust used in the stone dusting operation should be :- (a) of such fineness that, of the dry dust which passes through a 60 mesh sieve, not less than 50% by weight and except with the permission in writing of the Board of Trade, not more than 75% by weight shall pass through a 240 mesh sieve, and (b) of such character that it is readily dispersable into the air and, when in use in places when it is not directly wetted by water from the

strata, does not cake, but is dispersed into the air when blown upon with the mouth or a suitable appliance.

In this Act quoted above, it is stated that no dust shall be used for the purpose of complying with these Regulations of a kind which may be prohibited by the Board of Trade on the ground that it is likely to be injurious to the health of persons working in a mine. It further lays down that samples of the dust shall be systematically collected and analysed, and that the number of such samples shall not be less than ten to the mile. These samples are supposed to be collected monthly, except in the case of certain roads where it is only collected once in three months.

Two kinds of dust are inhaled by coal miners. One is the coal dust itself and the other is stone dust from strata above or below the coal seam. Usually the latter consists of shale, limestone or fireclay and which form stone dust especially during coal-cutting operations. Frequently the stone dust consists of silicates or limestone. In this district the percentage of stone dust is high, which probably accounts for the fact that in all its one hundred years history, this mine has never had a serious explosion.

In coal mining, each district seems to have a different mortality from respiratory diseases. Thus in the mine in which I am especially interested, the incidence of respiratory disability is relatively low, whilst in another mine not four miles away it is high, and in this latter mine, numerous cases occur of reticulation, nodulation, coarse mottling

and massive shadows.

The diminished incidence of pulmonary tuberculosis in coal miners has persisted for generations. In old statistics viz.1849 - 1853 it is no less marked than now (J.S.Haldane). Thus one can only conclude that the inhalation of coal dust seems to prevent tuberculosis. When dusty air is inhaled much of it is deposited on the moist walls of the nose and respiratory passages and only a very small part of it in the finer bronchi and alveoli. Thus expired air is practically free from dust. A fair proportion of the inspired air however, does reach the alveoli and is deposited there.

All the air passages of the bronchi are lined with ciliated epithelium and the action of the ciliary stream sweeps the dust out of the air passages until it can either be swallowed or expectorated and thus eliminated. Even the finer dust which reaches the alveoli must be eventually eliminated or otherwise the alveoli would finally become choked with solid particles of dust. In coal miners the elimination of coal dust goes on for weeks even after they have ceased working - as shewn by their sputum remaining black.

Microscopical examination shows that the dust particles are largely engulfed by certain large cells in the alveolar walls known as phagocytes. These cells have the property of taking up and destroying living bacteria. Many of these dust cells are found in the sputum and in the bronchial secretions so that they evidently wander out of the alveoli and up the bronchi. Again others find their way into the

lymphatic vessels and are found in lymphatic tissue around the bronchi. Many are carried on to the lymphatic glands about the root of the lung, so that in time the glands become highly charged with dust. It is not known what definitely happens to the dust particles in the lymphatic glands - possibly they pass into the blood stream or are excreted via the alimentary canal at some point. Incidentally if the dust particles do gain access to the blood stream, would this cause those serious attacks of what appears to be spasmodic asthma that one sees so frequently in coal miners?.

With certain kinds of dust, the elimination mechanism seems to fail and these are, of course, the dangerous kinds of dust.

In the case of some dusts - coal dust for example, when a miner is away from the dust for a long time the lung cleanses itself, but with the dangerous kinds of dust, part only is eliminated. The remainder is collected in dust cells which are found in groups on the alveolar walls. At the same time other changes have begun, of which one is the developement of fibrous tissue with consequent distortion of the.lung structure and loss of elasticity in the lung (J.S.Haldane)

This last is a factor of great importance,in my opinion, as it explains the almost universal occurrence of Emphysema in coal miners. One cannot examine a coal miner of over forty years of age without finding some evidence of Emphysema and in some individuals there are gross findings. The packing of the lymphatics with coal dust seems to initiate a foreign body reaction or fibrosis which causes a

loss of elasticity in the lung and consequent fibrosis. This causes great dyspnoea and distress as in the second case of my own series - besides others.

When large quantities of a harmless dust are inhaled inflammatory reaction and permanent damage to the lungs is caused, so that the difference in the different effects of inhaled dusts is only one of degree.

I think that in recent years the coal mines have become more dusty due to mechanisation and that the lungs of the present day miners are overloaded with coal dust and to a limited extent with certain kinds of stone dust also.

One may infer that it is only because the amount of dust inhaled is in actual practice moderate that ill effects do not in practice arise from the inhalation of what are usually regarded as 'harmless' dusts.

One may ask why it is that certain dusts are relatively harmless, whilst others are definitely harmful, and the answer is that the harmful dusts are only eliminated from the lungs with great difficulty.

When the writer was an undergraduate, it was considered that silica and quartz, because of the sharpness and angularity of their crystals produced a mechanical change in the lungs which caused a fibosis. It is of course well known that dusts which are harmful to humans break up into sharp fragments. One would therefore expect that all hard stone when ground up would be harmful, and that quartz, sandstone etc. would be equally harmful if inhaled in a relatively pure

form and when mixed with other dusts. Such, however, is not the case - only quartz in an almost pure form yields dust which is dangerous and this is found as millstone grit, ganister, flint and sandstone. The only exception to this is true granite which consists of quartz, felspar and mica, and many men engaged in dressing this stone suffer from Silicosis. There is another type of granite which is worked in quarries in England and is of quite a different character as it does not produce a'dangerous dust'.

In the Cripple Creek gold mines of Colorado, the gold occurs as an intrusion through granite and this rock contains over 70% Silica free and combined and yet gives a dust which is relatively harmless. The dust of ganister which is almost pure Silica is very dangerous, but Collis has published an instance where ganister dust used with fireclay to make ganister bricks causes no trouble. The reason for this would appear to be that along with the quartz there is inhaled a quantity of amorphous dust derived from the local 'country' dust. (J.S.Haldane - Effects of dust inhalation in Mines).

In the evidence of the Royal Commission 1864, it is stated that in Cornish mines where the lode passed through a slaty rock known locally as 'Killas' the miners did not suffer from phthisis, but that only when the lode passed through granite did they contract Silicosis and Phthisis.

The original theory of mechanical irritation is on the whole, difficult to believe. Minute particles of fine dust one might think could hardly do a great deal of damage although by packing the lymphatics they may produce a

foreign body reaction or fibrosis. Apart from this, however, it has been found that precipitated Silica remains in the lungs and causes changes similar to those produced by quartz dust, (Carleton)

In the production of Bronchitis, however, it is likely that mechanical irritation is a point of considerable importance.

Gye and Kettle found that soluble colloidal silica when introduced into the body, has injurious local and general reactions, also that silica dust introduced locally is both injurious and renders a tissue more liable to tuberculous infection. They suggested, therefore, that the injurious action of Silica is due to it going into solution gradually and as a result poisoning the phagocytic cells in the lungs,or so effecting them, that they are unable to resist tuberculous infections. If this were so, however, one would expect that such dusts as shale, which contains 50% Silica, would cause serious effects which is, however, not borne out in practice. Also the behaviour of other types of stone dust which are harmless in practice although they contain 70% Silica, would be difficult to explain.

One must conclude, therefore, that dusts which produce fibrosis or phthisis are harmful in proportion as they are physiologically inert. One formulates the theory that a dust particle engulfed by a phagocyte simply remains in the cell and interferes with its vital processes and if enough particles of dust are engulfed the cell is eventually killed not be any direct toxic action but simply by interfering

with the physiology of the cell.

Coal is a very insoluble substance but it has the property of taking up or absorbing large quantities of nitrous fumes, methane or other gas, and giving them off again in a current of air. Possibly it is the result of inhaled coal dust giving off other substances which it has absorbed that accounts for the fact that inhaled coal dust stimulates the process of dust excretion. Other non-crystalline dusts besides coal dust possess the property of absorbing substances which are again given off to water not containing them and it is for this reason that other dusts are capable of stimulating the process of dust elimination.

From this one can deduce that Silica is not dangerous because it is Silica, but because it is physiologically inert and incapable of exciting the process of dust elimination. This then would apply to dry crystalline and completely insoluble dust and Arnold and Beattie found that in animals which had inhaled emery powder, results similar to Silicosis were produced in the lungs.

Recently it has been stated (Brit.Med.J.Nov.4th 1944) that Silica exerts its injurious effect upon animal tissue through a slow transformation into Silica Acid and further goes on to say that Denny Robson and Irwin(Canad.Med.Assoc. J.1937. 1) thought that if the solubility of Silicious material retained in the lungs could be sufficiently reduced, the usual fibrotic response could be diminished or prevented.

This was the beginning of the modern treatment of

Silicosis with Aluminium powder which, when inhaled, rendered Silicious material insoluble in vitro by coating the silica particle with a thin film of gelatinous hydrated alumina, which, on drying, formed crystalline β-aluminium monohydrate.

Attempts have therefore been made to prevent Silicosis by mixing quartz with metallic Aluminium dust and this is at present on trial.

Before the practice of preventing explosions in coal mines by adding shale dust to the coal dust normally present was introduced, Haldane was asked to make enquiries into the experience of men who had been exposed for many years to the inhalation of shale dust. Also to enquire into the experience of men exposed to the dust from coal cutting machines when cutting through rock or strata adjacent to the coal seam. He, Haldane, stated that no ill effects were noted from this and concludes by saying " there are, therefore, no grounds for apprehending any ill effects from the increased percentages of shale dust which are now everywhere being applied underground in coal mines, although this shale dust contains over 50% of free and combined Silica including about 35% of free Silica or quartz".

Such were J.S.Haldane's views on this stone-dusting as it is called, but I would call attention to the fact that this dust reticulation of the lungs,or pneumoconiosis, as now found in so many coal miners and which leads to much incapacity, was not then recognised as qualifying for compensation under the Workmens Compensation Acts.

The flood of cases which this recognition has released has revealed very clearly that the shale dust and other stone dusts used to prevent explosions in coal mines, has a very deleterious effect on the miners lungs. It is a practice which, when persisted in, leads to dust reticulation of the lungs and even massive shadows on radiological examination.

Haldane does qualify his remarks by stating that before stone dust of any kind is used neither human experience or experiments on animals should place it under any suspicion of belonging to a harmful class of dust and he further remarks that "even the most harmless kinds of dust may become harmful if inhaled for long in large quantities".

It is my opinion that we are now reaping the result of the long continued inhalation of shale dust which has been so liberally used in coal mines to prevent explosions. The coal dust in it may stimulate the process of dust elimination from the lungs, but not all the Silica will be eliminated and it is this stone dust which is causing the X-Ray picture seen so frequently to-day and which is recognised as dust reticulation of coal miners.

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List of References:

Canadian Med.Assoc. J. 1937: 1.
Ibid. 1939-40: 213.
Indust. Med. 1939: 8, 133.

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THE RELATION BETWEEN DUST INHALATION AND RESPIRATORY DISEASE.

To establish this relation among coal miners some reference to the nature of the dust to which these men are exposed is necessary.

Shufflebotham remarked that fibroid lung is rare amongst coal miners and anthracosis does not seem to entail disablement, but he pointed out that coal miners are exposed not only to coal dust, but also to dust of the strata in which the coal is found. Also, Oliver has remarked that when a coal miner's lung shows marked fibrosis it is an indication that the individual has worked in a coal seam that contained a quantity of stone - it is the stone dust in the coal, not the coal dust itself, which is the cause of the fibrosis. As Belt has remarked, the coal miners lung is, in a very real sense, his occupational log book - it retains a quantitative and indelible record of the mineral particles breathed during life and after death, as Cummins,1935, says, it constitutes a sort of palimpsest of the industrial history.

Probably the prevalence of the respiratory disease amongst coal miners is the character and the amount of the stone dust present.

The influence of coal dust itself has been investigated in America by Drs.Wainwright and Nicholls. These workers having first previously agreed from statistical and other evidence that coal miners are singularly free from phthisis,

exposed guinea pigs for about two months to the inhalation of coal dust and then injected a culture of tubercle bacilli into them. Such anthracosed animals developed extensive tuberculosis of the abdominal viscera and of the glands around the tracheal injection but the lungs were free whilst control animals developed extensive tuberculosis of the lungs and abdominal viscera.

Such facts are evidence that coal dust protects in some way from phthisis.

At the outset, therefore, the form of respiratory disease which follows on dust inhalation is found to vary and Bronchitis which occurs in excess whenever there is exposure to injurious dust appears to be par excellence the chief of the pneumoconioses.

The importance of Bronchitis as an indication of the injurious properties of a given dust has been over-looked somewhat for two main reasons viz:

(1) The excessive mortality from phthisis in some dusty industries has overshadowed it.

(2) Bronchitis isacomplaint which advances slowly and though causing incapacity does not cause much mortality during the working period of life.

Leaving aside bacterial infection, the cause of Bronchitis is stimulation of the Bronchial mucosa. This causes congestion and inflammatory hyperaemia of the walls of the air passages so that an excessive exudation of mucous results. Dust is inhaled and rests on the walls of the bronchi and to be removed must be mixed with mucous and

be wafted back by the ciliated epithelium and expectorated as mucous. If a patient, therefore, has a black spit, it can almost be taken for certain that his ciliated epithelium is working well and that his mechanism for the removal of dust is functioning satisfactorily.

To bring about the removal of the dust, however, a large excess of mucous has to be produced and this has to be increased with the amount of dust present. Obviously, therefore, if this dust is increased beyond physiological amounts and degeneration and destruction of the ciliary mucosa will occur and at the same time by frequent coughing a ballooning of the alveoli is produced, causing Emphysema and by dilating the bronchi whose epithelium is degenerated, a condition of Bronchiectasis could be produced. This could be the etiology in Cases 2,3,6,13,15 and 21.

Coal dust alone does not cause Bronchitis, but coal dust with an admixture of stone dust can and does.

In addition to the irritation caused by the inhalation of dust in coal miners, one must also remember the irritation by fumes from explosives and the variation in temperature in different parts of the mine and different humidities.

There are various methods of estimating the concentration of dust in a given sample of air - the commonest method being by means of the konimeter which is simply a piston working in a cylinder of about 2.5 cu.ins. The piston is depressed and the cylinder filled with air drawn in from outside - the dust impinges on a plate made sticky with gelatine. This is the method used for sampling dust concentrations

on the Rand Mines in South Africa. By means of this instrument a 'snap' sample can be taken.

It should be noted that in the case of these dusts that chemical and physical activity are both increased by fine division. Very large dust particles are probably of little pathological significance and in taking dust samples the particle count (that is the number of dust particles in a given microscopic field) seems to give the most valuable information.

Small particles are more injurious than large and the smaller the particle the more injurious it is. To be pathological the particles must be three microns or less in diameter.

The foregoing remarks apply particularly to the Silica content but also to any dust that is inhaled.

It would appear that the size frequency of the dust particles in the lung sections are not unlike the average size distribution of the dust in the air in coal mines. Few particles over three microns are observed in the air, and few particles over that size are found in the lungs. Mavrogordato,1940, regards particles of five microns or less as dust of fibrosis producing size.

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THE PROBLEM OF CHRONIC PULMONARY DISEASE IN COAL MINERS.

Chronic pulmonary disease is a problem among all mining communities and is a well known clinical condition to all practitioners in mining districts. In 1934 it was officially recognised as an incapacitating disease and compensation was paid under the Workmens Compensation Acts. This, however, had the effect of releasing a flood of cases so that the number of cases receiving compensation has mounted rapidly until pulmonary disease in coal miners has become a question of national importance. The number of cases in the coal mining industry has risen from 229 in 1933 to 1,279 in 1938. This rise has, I believe, continued since, and it is my own impression that it would be higher still if practitioners were more aware of the condition and especially of the earlier stages of it. As so often happens, however, the condition is labelled Chronic Bronchitis and left at that, and the men themselves seem curiously complacent about it. It has surprised and startled me in going over this ground for the purpose of this Thesis how many cases one can find if one is on the look-out for them.

That the miner can, and does, die from this pulmonary condition is evident by the frequency of deaths attributed to Silicosis in the different industries as supplied by Bridge(1938) in an analysis based upon data from death certificates. In this analysis, 41.2% of the deaths attributed to Silicosis in England and Wales in 1938 were assigned to the coal mining industry.

The rise of numbers of new cases of Silicosis for the coal mining industry between 1933 and 1938 probably is not so much due to an increased number of new cases as to the recognition of disease already present.

The total amount of compensation paid out to Silicosis cases in 1933 was £24,000 out of £109,000 paid for the whole country. In 1938, however, the corresponding amount was £138,000 out of a total of £231,000.

One must bear in mind in connection with chronic pulmonary disease in coal miners that only cases which satisfy certain limited diagnostic and legal criteria can be officially regarded as cases of Silicosis and it is my impression, that a condition peculiar to coal miners exists which is now called Pneumoconiosis, because the condition is due to the inhalation over long periods of time of coal dust and to a lesser extent of stone dust. This condition of Pneumoconiosis is characterised by two main symptoms which are cough and dyspnoea. The main features found on examination are Emphysema (especially basal emphysema) and pulmonary fibrosis found on radiological examination.

In all the cases which I have studied for the purpose of this paper the diagnosis has almost invariably been Chronic Bronchitis and Emphysema and it is only when further radiological examination has been carried out that a condition of pulmonary fibrosis analogous to early Silicosis has been found.

It is my impression that these cases are only found in men who are exposed to coal dust, silica dust and other mineral dusts for considerable periods. Thus a man who commences

work in the mine at fourteen years may only exhibit symptoms twenty, thirty or more years later - a fact which rather tends to mask the condition as the patient is not likely to blame his working conditions when he has been exposed to these conditions maybe for thirty years. Another factor of importance is that the condition is very insidious and steals upon the patient very gradually so that it is only when he finds he cannot carry on the work he is accustomed to, that he seeks advice. Even then it is frequently only to invoke the aid of the practitioner to secure lighter work for him without much mention of the disabling factor.

I have frequently been struck by the frequency of occurrence of a man previously non-complaining and robust who suddenly discovers that he has become incapable of carrying on his work by reason of dyspnoea. When kept at rest and especially in the open air, he makes an apparent quick recovery and may return to work and carry on for a while, but he is never again capable of carrying on his previous occupation and becomes a frequent industrial 'absentee' or 'casualty'. If he persists in attempting to carry on work underground, and I may mention that in my experience miners are curiously conservative in their outlook, he quickly becomes completely dyspnoec and finally bed-ridden and dies from congestive cardiac failure.

These are all ones own personal impressions and therefore, not of much importance, but they coincide in many respects with those of my late father who was also in practice here for forty years, and had mentioned to me that in his early

years 'miners asthma' had been a common complaint but that it had then disappeared but now appeared to be returning. Since his death, some eight years ago, I have kept his observations in mind, and have come to the conclusion that the so called 'miners asthma' is returning.

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THE RELATION OF SHOT-FIRING TO PULMONARY DISEASE IN COAL MINERS.

In order to loosen and bring down coal from the coal face, various methods have been used. Hitherto the commonest method has been to use an explosive charge. This is known as shot-firing and the usual procedure is for the deputy overman to bore a hole in the coal face by a pneumatic or electric drill. This hole is generally about three feet deep, and having completed the drilling, the explosive is them inserted in cartridge form. The drill hole is then 'stemmed' with clay or sand in order to compress the charge into a small space, so as to have the maximum effect in breaking up the coal. Next, the coal face is liberally sprinkled with stone dust, the composition of which has already been mentioned, and then when everyone has retired to s safe distance the charge is detonated by the 'deputy'. Within a very few minutes of the explosiom, the men return to the coal face and break up the coal and load it into tubs or on to a conveyor belt from whence it is removed from the working place. As can well be imagined, this is a very dusty period in the mine the explosion causing the air to be charged with fine stone and coal dust in addition to the fumes from the explosion.

The question has been raised as to the relation between these fumes and pulmonary disease of coal miners.

Oliver has pointed out that these fumes are nitrous fumes and they consist of nitrous and nitric oxides,nitrogen

dioxide, nitrogen tetroxide and nitrous and nitric acids. The most important of these in relation to pulmonary disease would be Nitrous Oxide (NO), Nitrogen Dioxide (NO_2) and Nitrogen Tetroxide (N_2O_4).

Animal experiments have shown that concentrations of nitrous fumes greater than two hundred parts per million are definitely harmful (Lehman and Hasegawa 1913). The effect of nitrous fumes when inhaled is to cause irritation of the larynx and cough when over forty parts per million. Therefore, it appears possible that repeated exposure to concentrations much less then forty parts per million may have a harmful effect on miners, as one of the most prominent symptoms seen in practice is a short frequent cough in cases of pneumoconiosis of coal workers. Amongst my own series of cases this was the first symptom that brought them to seek advice.

Haldane and Graham (1935) published a report on the amount of nitrous fumes produced during shot-firing and found concentrations of up to ninety parts per million, although as they pointed out, exposure to these concentrations could only be for a minute or so, as ventilation would rapidly dissipate the fumes.

Robson, Irwin and King of the Banting Institute, Toronto, published a report which is very significant in its purport, which is the effect upon rabbits of nitrogen dioxide either in conjunction with silica dust, or alone. They stated that rabbits which had been exposed to atmospheres containing as little as 0.01%, that is one hundred parts per million by

volume of Nitrogen Dioxide for two hours per day along with silica dust, developed classical Silicosis after about twelve weeks, whilst when exposed to Silica dust alone, the time required for the developement of Silicosis was ten to eleven months.

In view of this interesting report, it seems quite possible, therefore, that repeated exposures to much less concentrations than one hundred parts per million might produce chronic pulmonary disease in coal miners who might then go on to develope Pneumoconiosis or even Silicosis.

The Committtee on Industrial Pulmonary disease have carried out an investigation on the production of nitrous fumes in coal mines. In this they estimated the nitrous fumes in the 'returns' that is the return airways from a working district and found that the average production of nitrous fumes was between 0.8 and 5.1 parts per million. These figures seem low, but it should be remembered that nitrous fumes are very readily absorbed by coal debris and dust at the coal face. The fumes are absorbed by the coal dust and then slowly given off again in a current of air. When the coal dust and debris brought down by the shot-firing are removed, the concentration of nitrous fumes drops considerably. Thus it is that appreciable quantities of nitrous fumes are present in shifts in which no shot firing has occurred.

When the ventilation is good, the concentration of nitrous fumes even within a few minutes of a shot being fired, is less than five parts per million.

Whether exposure over long periods to such low concentrations, in conjunction with a dusty atmosphere, will definitely cause chronic pulmonary disease or bring about a condition of Pneumoconiosis of coal miners is not known. The experimental work of Robson, Irwin and King of the Banting Institute is, however, very suggestive to my mind. These experiments, as already mentioned, showed that extensive damage could be done to the lungs of rabbits exposed two hours a day for two weeks, i.e. in 28 hours, to a concentration of one hundred parts per million of nitrogen dioxide.

Is it not possible, therefore, that exposure of humans to a concentration of five parts per million for much longer periods, could produce pulmonary damage?. In some 560 hours working time, a coal miner is exposed to the same hazards as the rabbits in the Toronto experiments.

Vigdortschik 1937, showed that 127 men working for three to five years in concentrations of 2.5 parts per million of Nitrogen Dioxide showed evidence of chronic poisoning due to the fumes.

It is, however, possible that the sixteen hours daily that a coal miner is not in contact with the fumes may give the lungs time to recover from the eight hours or so that he is exposed to them.

Taking this all in all, it appears to me especially desirable to avoid any unnecessary exposure to nitrous fumes as the Toronto experiments seem to indicate a very real relation between exposure to nitrous fumes and the developement

of Silicosis or other chronic pulmonary disease to which the coal miner is subject.

Methods of obviating, or minimising, the exposure of coal miners to nitrous fumes:- The objects here would be to either

(a) abolish shot-firing and use some other method of bringing down the coal.

(b) Do all the shot-firing in the back (second) shift and leave the coal for some hours before the coal fillers come to remove it.

(c) Some high pressure ventilation scheme for use immediately after shot-firing.

Recently a hydraulic method of breaking up the coal at the coal face has been evolved, but it is not yet in general use. The most practicable method at present would be the use of an extractor fan to remove the fumes after shot-firing has been taking place, and to forbid men from returning to the place where shot-firing has occurred for some hours after it has taken place.

In the Rand mines, four hours have to elapse before the miners return to the district where the shot-firing occurred. In some collieries in this country, shot-firing is only permitted in the 'back' shift. It should, however, be noted, that coal dust and coal debris absorb the nitrous fumes and then slowly give them off again in a current of air, so that a small percentage of nitrous fumes are constantly present.

There is one other method which is beginning to be used and that is by wetting the coal by means of a water spray.

By this method both dust and fumes are kept down to a minimum. The Mist projector, as it is called, is slowly being brought into more general use. This is simply a device to provide a fine 'mist' of water all over the coal face and can be maintained by means of a pump from a tank of water, if compressed air is available.(Environmental Studies)

It is stated that a very dilute solution of Ammonium Carbonate is better than water especially in reducing the nitrous fumes, as well as the dust. This method is not practicable while the men are working but only a short time is necessary before the shot is fired for the mist projector to be in action.

By the above methods, or one or all of them, both nitrous fumes and dust can be reduced to a minimum which will not be harmful to men working at the coal face.

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Anthracosis. In the current edition (1943) of Boyds Pathology, the condition,of Anthracosis is described as the commonest but least harmful of the dust diseases. It is found in coal miners it is stated, but a varying amount of coal dust is found in every lung at autopsy and no sharp line can be drawn as to the amount of coal dust that constitutes Anthracosis. It does not cause much irritation as it is insoluble and a lung may be loaded with it and yet show little fibrosis. The carbon particles are taken up by mononuclear phagocytes and deposited in the interlobular septa the deep layers of the pleura and the bronchial lymph nodes. All these structures acquire a coal black colour. Anthracosis does not predispose to Tuberculosis indeed coal

miners are singularly free from that disease.

The above extract is from Boyds Text Book of Pathology and after nearly ten years in practice in a mining district, I would like to differ in many points from this. A pure Anthracosis is an unknown quantity as one cannot very well acquire a pure Anthracosis in industrial employment without at the same time acquiring a degree of Silicosis from the rock that the miner has to drive through, and it is, in my opinion, a clinical entity that the coal miner acquires - an Anthraco-silicosis one might call it or Pneumoconiosis of coal miners as it is now known.

It has long been known - at least since the investigations of a Royal Commission in 1861, that dust is one of the deadliest enemies of the miners health. Yet scientific study of the causes of miners lung diseases was long and tragically delayed in Britain. Only in 1918, twenty-one years after the first Workmens Compensation Act was passed did Silicosis become a 'scheduled' disease for which compensation was payable; only in 1934 was the Silicosis compensation scheme extended to include all workers engaged 'in any operation underground in any coal mine'.

In February of last year an Act was passed to bring into the scope of workmens compensation all cases of Pneumoconiosis which is defined as 'fibrosis of the lungs due to Silica dust, Asbestos dust, or other dust' and includes the condition of the lungs known as 'dust-reticulation'. The way for this legislation was prepared by six years of pains-taking scientific investigation in the pits among the

miners of Wales (as it happens) conducted by a special committee of the Medical Research Council which published its medical findings in June 1942 and its studies of environmental conditions in August 1943.

These reports resulted in an Order giving the Chief Inspector of Mines power to compel the adoption in any mine in South Wales and Monmouthshire of a variety of measures to reduce the exposure of men to dust, and to the appointment by the Minister of Fuel and Power of an Advisory Committee on the treatment and rehabilitation of miners suffering from Pneumoconiosis. This committee has now decided, however, that not enough is yet known about the disease for the recommendation of large scale measures of treatment. It therefore recommends the creation of a centre for clinical study and research and the introduction of periodic medical and X-Ray examinations of miners. The carrying out of this proposal as soon as circumstances permit will form another contribution towards the long overdue comprehensive programme for protecting and improving the health of miners. (extract from the 'Times')

Now all dwellers in towns have black lungs and only in dwellers in remote places does one find pink lungs. The degree of this blackening varies from the greyish lungs of a country dweller to the coal black lungs of a coal miner and the various gradations of these to the condition of Anthracosis when one finds a black area in the lung surrounded with a rind like an orange and which contains pure carbon pigment and which is officially recognised by

the Silicosis Board as Silicosis for the simple reason that if this were not recognised as such, no reasonable diagnosis could be advanced. All gradations are so found and little mention is made of them in the Textbooks of Pathology. Anthracosis was first described as such by a young Scottish graduate named Thomas Stratton over a century ago. He described the black lungs of coal miners and coined the term Anthrocosis.

Thus Anthracosis can be applied equally to the lungs of a town dweller and those of a man who has worked for many years in a coal mine, but as already pointed out, there are many gradations and variations in this.

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CAUSES FOR THE RISE OF INCIDENCE IN CHRONIC PULMONARY DISEASE.

The men themselves believe that the cause of the increased number of cases is the increased use of mechanical contrivances in the mine as compared with previous years. Such mechanical contrivances as the 'windy pick' which is a type of pneumatic drill similar to the drill one sees in use in city streets to dig up underground cables. Recently American coal-cutters have been introduced to step-up the output - these are combined cutters, loaders and conveyors.

There may be some truth in their idea of these machines causing an increase in the number of pulmonary abnormalities as these machines have to operate in very confined spaces, often only about two feet high, and the exhaust from the machine throws up a continuous column of dust in the operator's face. Also the machines have to cut through stone sometimes, and in such a confined space, the air will be thick with stone dust. The danger of the patient with latent pulmonary tuberculosis in these confined spaces will also be appreciated and I should add that although the incidence of pulmonary tuberculosis in this district is not high, yet there has been an increased number of cases of men over fifty dying of pulmonary tuberculosis recently. The generation before this one knew of such conditions as Miners Asthma which has been already mentioned. This has not been present for some fifty or more years in any numbers. Now, however, the condition is again being seen.

It may be asked what it is that is bringing it back. What reason there is for the return of the old miners asthma - the 'scourge of the mine' as dramatically described by authors of fiction - is difficult to state, but my own impression is that it is the latest mechanical contrivances used in the coal mines these days, which are usually operated by compressed air and which cut through coal and stone alike. They also raise a considerable dust from the exhaust and so desseminate all around the working place a mixture of coal and stone dust which the miner has perforce to inhale. Long continued inhalation of this over a period of many years produces a characteristic change in the lung fields as seen radiographically and frequently, though not always, clinically also.

Characteristics of dust reticulation:- One might say that reticulation is both an X-Ray and a clinical diagnosis, and the one may be present without the other. Radiologically it is seen (as in Casel.) as a'graining' of the lung field passing from the hilum out to the periphery. This network or reticulum is as marked at the periphery of the fields as it is nearer the hila. Other terms which are used in addition to reticulation are granularity and arborisation.

The modern X-Ray plant will show up a reticulation stage while an X-Ray plant older than say 1928 will not. A technically good radiographic film has varied in its standard since 1919 when one relied on glass plates. Up till about 1928, X-Ray plants were not capable of showing very definitely the condition we now call dust reticulation.

The cause of this radiological appearance -

Reticulation is presumed to be an intense concentration of coal dust associated with peribronchial thickening and with an area of Emphysema around it. It is a 'foreign body reaction' and is caused by an inability of the lung to rid itself of inhaled particles. Under ordinary conditions the lung has resources for cleaning itself which are most surprisingly effective, even when, as in these cases, dust is inhaled in quantity. Take Case 28, who states that when he comes out of the mine, his sputum is black and remains so for an hour or two, but that after that it becomes clear again. The lining mucosa of the upper respiratory air passages extracts dust from incoming air, prevents it from reaching the lungs and sweeps it by ciliary action to the mouth and throat and throws it off as sputum. In this connection, however, it should be remembered that men doing hard manual work are frequently breathing through their mouths, and have their mouths wide open so that they will get a larger quantity of inhaled dust than another person who is breathing quietly. I have talked to miners, and they say that while the younger men tie handkerchiefs over their mouths, the older ones chew tobacco which ensures them keeping their mouths shut.

The carbon particles that do reach the lungs are taken up by macrophages, or dust cells, and deposited in the interlobular septa, the deep layers of the pleura (- does this cause the thickened pleura so often seen in miners at autopsy?) and the bronchial lymph nodes. The dust that enters the macrophages, passes into the lymph channels and thence into the lymph nodes and it is this choking of the

lymph channels with dust which produces the reticulum seen by the X-Ray and which is known as dust reticulation.

There seems to be good evidence to show that dust reticulation in coal miners is due more to silicates than to coal, and the effect of this is that the dust cells are gradually immobilised so that they tend to accumulate in the lung, and secondly, they also tend to produce reticulum fibres. As Belt says neither of these effects can be produced by coal dust alone. Thus it is that coal dust which ordinarily would be excreted from the lung and bronchi is not excreted when it is mixed with a proportion of silica and instead the above changes take place which produce the condition known as dust reticulation.

Thus coal dust by itself may be relatively harmless but when mixed with silica, it is very far from being harmless. In the mine the air at the coal face is thickly impregnated with both coal and stone dust and the men are breathing it. I have found by an analysis of the stone dust used to prevent explosions at the coal face, that it consists of 1.05% Silica Dioxide.

Clinically reticulation is a cardio-respiratory syndrome with dyspnoea as the important symptom and tachycardia as the important sign. The tachycardia has been found by electrocardiographic methods to be a physiological tachycardia.

Prior to July 1st 1943, reticulation of the lung field was not admitted by the Silicosis Board to be a cause

for certification under the Workmens Compensation Acts. It was found, however, that coal miners were suffering from an incapacitating degree of dyspnoea with cough, tachycardia and other symptoms which came on fairly acutely and was a permanent condition. Under these circumstances on radiographic examination, the lung was seen to have a fine grained appearance all over it with obliteration of the hilar shadow and the term reticulation was introduced. From the above date reticulation was admitted as an incapacitating industrial disease.

Is there any degree of recovery from this condition? - Possibility of functional recovery. These are controversial questions, but my own impression is that they do not make any degree of permanent recovery and that they become chronic invalids, linger on for a variable length of time, and finally die - usually of right heart failure or associated intercurrent bronchial affection.

Prevention of reticulation and therefore of early Silicosis: - it is difficult to get the miners to wear a mask and few would do so. Various attempts have been made to spray water about and a mist projector has been produced which keeps up an appropriate amount of water vapour in the atmosphere at the working place. The miner, however, conservative as ever, protests that he is contracting rheumatism by this means.

It is of interest to me to recall that my grandfather, himself a mining engineer, suffered from Chromic Bronchitis and Asthma from the age of fifty, which he attributed to

defective ventilation in the mines in his early years. He was aware of the coal dust hazard to miners and patented a device which sprayed the coal tubs with water before their removal from the coal face.

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ANATOMY AND PHYSIOLOGY OF THE RESPIRATORY ORGANS IN RELATION TO PNEUMOCONIOSIS.

The nasal air passages and the bronchi and lungs are especially designed to prevent the inhalation of dust.

In the nasal area the fine hairs around the nostril arrest the inhalation of dust, and in the nose itself, the nasal mucous membrane is composed of columnar ciliated epithelium among which is interspersed many goblet or mucous cells. Thus dust becoming entangled in the mucous is eliminated by means of the current established by the cilia all waving in the same direction.

The air from there enters the organs of respiration proper which are the larynx, trachea bronchi and lungs.

The larynx opens above into the lower part of the pharynx and the air which passes in and out traverses the larynx, pharynx, nasal cavity and the oral cavity too, if the mouth is open, as it so often is in hard manual labour.

The cavity of the larynx is lined with mucous membrane under which in certain localities are masses of mucous glands.

The lungs:- Each lung, in health, lies free within the right or left pleural cavity and is attached only by its root and the pulmonary ligament. It should be remarked however, that healthy lungs are uncommon in coal miners, at least in my experience from post mortems, in which the pleura is frequently thickened and there are frequently adhesions between the parietal and the pulmonary layers of

of the pleura.

The lungs are not exactly alike, the right lung being slightly larger than the left. It is also shorter and wider. This difference between the lungs is due partly to the great bulk of the right lobe of the liver which forces up the right dome of the diaphragm to a higher level than the left dome, and partly also to the heart and mediastinum projecting more to the left than to the right thus diminishing the width of the left lung.

The healthy lung is light, soft (feathery almost) to the touch and crepitates when pressed upon. When placed in water the lungs should float.

When the chest cavity is opened, the atmospheric pressure and the intra pleural pressure are equalised and the lungs collapse to about one third of their size.

The left lung is divided into two lobes by an interlobar fissure. In the right lung there are two interlobar fissures which subdivide it into three lobes.

The apex of the lung is blunt and rounded and rises above the level of the first costal arch to the full height of the cupola pleurae. It therefore protrudes above, through the superior aperture of the thorax into the root of the neck, and in cases of Emphysema, the 'fullness' and distension in the supraclavicular fossae are very apparent.

The surface of the adult lung is mottled in appearance and in persons not following an unduly dusty occupation

is slaty-blue in colour but scattered over this there are numerous dark patches and fine intersecting lines of a dark colour.

In early childhood the lung is rosy pink and the darker colour and mottling which later appear are due to the lung tissue, and particularly its interstitial tissue, becoming impregnated with atmospheric dust and minute particles of soot.

At every breath, foreign matter is inhaled, but only a small proportion ever reaches the lung. The greater part becomes entangled in the mucous which coats the mucous membrane of the nose, pharynx and larger air passages, and is gradually eliminated along with the mucous through the activity of the cilia attached to the lining epithelium. By the constant upward sweep of these, a 'current' is established towards the pharynx.

The very fine particles of dust which reach the deepest recesses of the lungs and then the interstitial tissue, are removed, although not wholly, by the lymph vessels to the bronchial lymph glands, which, in consequence, become black in colour. The colour of the lungs, therefore, depends almost completely on the purity of the atmosphere which is breathed and therefore, a coal miner's lungs are completely black.

Structure of the lung:- The lung is constructed for the purpose of bringing venous blood from the pulmonary artery into intimate contact with air brought to the lung by means of the trachea and bronchi. Thus an interchange

is effected by means of which venous blood takes up a quantity of oxygen from the air and gives up to the air small amounts of carbon dioxide. The dark venous blood is thus changed into bright red arterial blood.

The lung tissue is subdivided into many thousands of lobules. A thin layer of subpleural connective tissue lies immediately beneath the coating of pulmonary pleura. From the deep surface of this subpleural layer, fine septal processes penetrate into the substance of the lung and these with the connective tissue which enters at the hilum upon the vessels and bronchi form a supporting framework for the organ. Thus the lung is lobular and on the surface the small polygonal areas which represent the lobules are indicated by the pigment present in the connective tissue septa which intervene between them. The broad bases of these lobules abut against the subpleural layer whilst each of the narrow ends receives a minute division from the bronchial tree.

The Lung Unit: The unit of the lung is the lung lobule. This comprises a terminal bronchiole with its air spaces, blood vessels, lymph vessels and nerves. The larger branches of the bronchi traverse the substance of the lung giving off smaller and smaller branches which penetrate throughout the entire lung substance and forms a system of tubes through it. The finer branches of the bronchi are called bronchioles which, by further subdivision, give rise to the Respiratory Bronchiole of the Lung Unit. Within the lung unit, the respiratory bronchiole

gives off a number of terminal bronchioles or alveolar ducts each of which leads to a series of air spaces or atria. Each of the atria communicate with a further series of air spaces termed air sacs or alveolar saccules, the walls of which are pouched out to form the very numerous alveoli (or air cells) of the lung unit.

Structure of the Atria and Alveoli:- For the function which it has to perform, namely the interchange of gases between the blood and the air in the lungs, the alveoli are necessarily of very fine and delicate construction. The epithelium is reduced to a single layer of non-nucleated cells. It is no longer columnar and ciliated but has became flat pavement like epithelium. Outside the epithelium is a delicate layer of connective tissue which is strengthened by a network of elastic fibres which are especially marked around the mouths of the alveoli and is also carried over the walls of the air cells.

This last appears to me to be of some clinical significance as it is common knowledge that many (in fact most) coal miners of over fifty years are emphysematous and it appears likely that it is damage to the above mentioned elastic fibres by irritation of coal dust and stone dust that causes this condition. Irritation of the interstitial tissue, overloading of the lymphatics with foreign matter causes an irritation partly mechanical (anthracosis) and partly chemical (Silicosis) as Silica particles are soluble in tissue, fluids, which produces this emphysematous condition so frequently seen by the

practioner in coal mining areas.

This irritation of the interstitial tissue would produce a dilation of the alveoli with subsequent coalescence of adjacent alveoli and fibrosis of the fine blood vessels in the interstitial tissue with resulting obstruction to the pulmonary circulation and strain on the right side of the heart.

Once initiated this condition would tend to become progressive so that finally a gross Emphysema with right sided cardiac failure would result and this also is a condition one frequently sees in coal miners. Minor degrees of this condition are, however, more frequently seen than the gross-end result, and on X-Ray examination, a pulmonary fibrosis with Emphysema is apparent.

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PATHOLOGY OF PNEUMOCONIOSIS.

The lungs of all town dwellers are pigmented to a greater or lesser degree. In remote areas one may find a person who at autopsy has pink lungs, but such cases are few and I personally have only seen one such which was in a Shetlander.

A child has pink lungs but these quickly become slaty blue and mottled all over as his age increases.

The degree of this blackening varies from that of the greyish lungs of a country dweller and the various gradations of these to the condition of Anthracosis as seen in the underground worker in a modern coal mine.

Anthracosis as such was first described by a young Scottish undergraduate named Thomas Stratton just about a century ago. He described the black lungs of miners and coined the term Anthracosis.

Anthracosis can thus be applied equally to the lungs of a town dweller and those of a man who has worked for many years in a coal mine, but there are many differences and variations in this. In the coal miner's lung, one is dealing essentially with a mixed type of Pneumoconiosis due partly to the long continued inhalation of both coal and stone dust of mixed composition.

Thus the term Anthracosis is unsuitable for the reason that it is too wide in its applicatioh, and also because there are in the lungs of coal miners, a great deal of

Silica and Silicates present as well as coal dust. In addition, Anthracosis has come to mean a harmless condition, in fact a normal condition, whereas the Anthracosis that the coal miner suffers from has definite pathological features and can be a chronic incapacitating disease when seen clinically.

Thus the term dust reticulation has come to be used instead and this is used both from a pathological (or histological) standpoint and also from a radiological. In the pathological sense the reticulum to which the term applies is composed of dust laden cells and their connecting fibres. Therefore the term reticulation is used to describe a generalised increase in such tissue.

Anthracosis (or as we may now call it dust reticulation) is now considered as being more in the nature of a disease than was formerly thought.

The lungs have a uniform coal black colour and are usually voluminous due to the associated Emphysema. The texture of the lung is not so soft as in normal persons, it is indeed rather hard, granular and stringy to the touch. The bronchial glands are enlarged and of a jet black colour due to the accumulated carbon pigment. Small areas of an infiltrating fibrosis may be seen through the substance of the lung and these may have a gritty feel and be due to the action of Silica inhaled with the coal dust.

The excess of mixed dust inhaled by coal miners is in fact absorbed into the lymphatics of the lung by means of

phagocytes. The dust tends to accumulate in certain situations, especially along the peribronchial lymphatics and at the junction of the interlobular septa and the pleura. In the various positions where they are arrested they tend to give rise to reactive changes which vary according to the nature of the particles. It is my impression that these are partly mechanical and partly chemical. The mechanical effect is caused by coal dust causing a simple irritation, while the silica particles cause a chemical change, being slightly soluble in tissue fluids.

Thus is set up an overloading of the lymphatics in the interstitial tissue and by means of these irritative changes a fibrosis is produced which destroys the elastic tissue which supports and strengthens the alveoli of the lungs. This causes a dilatation of the alveoli and coalescence of neighbouring alveoli with the result that an Emphysema is produced.

The same irritation which causes an interstitial fibrosis, causes also an Endarteritis Obliterans, which, in turn, brings about an obstruction to the pulmonary circulation and a resulting strain on the right side of the heart.

Microscopically the dust deposits in a lacy pattern all over both lungs. Fine streaky processes are seen extending from the hilum of the lungs to the periphery. The change is diffused and extends all over both lungs, a point which is important in the diagnosis of the condition. It is diffused because it is simply an impregnation

of the lymphatic pathways with dust - the root glands are packed with it and in the lungs themselves the interstitial lymphatic pathways are full of it as are also the perivascular and peribronchial spaces. Other depots of dust are the subpleural tissue spaces. The result microscopically is a reticulum (or network) of fine cobweb appearance all over the lungs. Dust laden phagocytes have been arrested and transformed into fixed tissue phagocytes which are then knit together to form a network. Thus the condition is really a form of fine fibrosis and the tissue response is exactly proportional to the amount of foreign particles. No more cells take part in the reaction than are necessary to accomplish a simple segregation of the particles. There is no redundancy reaction (Belt 1943) and dust-reticulation differs sharply in this respect from silicotic fibrosis which is characterised by encapsulation and by a disproportion between the amount of fibrosis and the amount of dust. In other words Silicosis shows redundant fibrosis (Belt 1939) whereas dust reticulation does not.

In severe cases the dust becomes accumulated into small masses which are shown up in the X-Ray picture as fine rather diffuse nodulation, which is superimposed on the reticulation. In all my own series of cases, however, not one of nodulation or even nodular reticulation was found.

The dust itself is a mixed dust which is an important point as it explains to a limited extent, at least, why reticulation is essentially a simple foreign body reaction and not the complex chemical and reactive process one sees

in Silicosis.

On ordinary microscopic examination one only sees masses of carbon pigment, but by a special process the carbon can be incinerated leaving only the Silicates, which are found to be evenly distributed throughout the carbon deposits.

It is probable that dust reticulation represents the first of a series of pathological changes which may culminate in gross fibrosis and the severity of these pathological changes depends a good deal on the duration of exposure to the dust hazard. My own series of cases ranged, in age, from sixty-three to thirty-two, and the duration of exposure from fifty years to fourteen years. It is, however, noteworthy that the case with the longest exposure has well marked reticulation in his lungs and his sputum contains tubercle bacilli. He is in receipt of full disability compensation and is, one assumes, a case of Koniophthisis. In these series of cases, he is the first one described.

To return to the dust in the lymphatics after a fibrosis has been set up. The first thing that happens is that an interstitial fibrosis is provoked and this causes a peri-bronchial fibrosis or perivascular fibrosis which may be grouped together as Pulmonary Fibrosis. This has a very definitely incapacitating effect on a miner. He is not completely incapacitated for he can usually manage a light job of some nature on the surface but it very definitely limits his activities. Nine of my own cases, excluding those with reticulation, had Pulmonary Fibrosis as shown

by radiological examination and all of these cases had very definite limitation of their activities by reason of the cough and dyspnoea which seem to be the cardinal features of these cases. All had previously been hard working men and all of them chafed at the inactivity forced upon them by their physical condition. They had all worked as coal getters in distinction to surface workers and all wished to return to their own work.

I have no doubt that eventually these men will develope Bronchiectasis due to further fibrotic changes in the lung or they may develope right sided cardiac failure. The Bronchiectasis is produced by traction of contracting bands of fibrous tissue on the Bronchi. Secondary infection of the Bronchiectatic cavities then occurs producing the toxaemia purulent sputum and cachexia as seen for example in Case 2. Thickened pleurae are also very evident at autopsies on coal miners. one nearly always finds the pleura to be very adherent and thickened and attacks of dry pleurisy are extraordinarily common. One can only ascribe this to the deposition of dust in the subpleural lymphatics interfering with the lymphatic circulation and so causing a dry pleura and resulting pleural friction.

In addition to this, it is well known to practitioners in mining areas that besides the men who have ceased work owing to chest illness with or without compensation, there is a considerable amount of pulmonary disability of the reticulation type among coal miners. This latent pneumoconiosis is the cause of a serious amount of respiratory disability although the affected men are often able to

continue their accustomed work for some years within a limited range of effort. Frequently they are labelled Chronic Bronchitis or Bronchitis and Emphysema, but when they are examined under the X-Rays the condition of dust reticulation is seen.

Most coal miners seem to suffer from periodic attacks of Bronchitis and thus the condition steals on them very gradually.

The suggestion has been made that the 'trains' which take the miners to their working places underground, or in which they are conveyed back from the coal face, conduces to the developement of chest colds and Bronchitis. Jones (1936) has suggested that "the apparent harm attributed to the dust in coal mines is, in reality, dependent on Bronchitis resulting from exposure to cold on these 'trains' with secondary accumulation of coal dust in the lungs in consequence of the normal process of elimination of dust from the lungs being paralysed". This suggestion incidentally was accepted by the late Dr.J.S. Haldane.

To my mind this is not so, as I think the Bronchitis is induced by excessive inhalation of mixed dust, coal and stone, plus the inhalation of nitrous fumes from explosives and that the Bronchitic process is an attempt by nature to eliminate the excessive dust inhaled by entangling it in mucous and having it removed via the ciliary stream. It is only when the ciliated epithelium is itself paralysed by a chronic infection in the bronchi, that secondary infection takes place and a chronic bronchitis plus a pulmonary fibrosis paves the way for Bronchiectasis and

other serious chronic pulmonary diseases.

Reticulation as has already been mentioned, is produced by coal dust as well as by stone dust and this point is of importance as it indicates that coal dust is not the harmless dust it was formerly supposed to be - (Sutherland. Personal observation). Reticulation is in fact a Cardio-respiratory syndrome with dyspnoea as the important symptom and tachycardia as the important sign. The tachycardia has been proved by electrocardiographic methods to be a physiological condition.

Prior to July 1st 1943, reticulation of the lung field was not admitted by the Silicosis Board to be a cause for certification under the Workmens Compensation Acts. It was found, however, that coal miners were suffering from an incapacitating degree of dyspnoea with cough, tachycardia and other symptoms which came on suddenly and were permanent. Under these conditions on radiographic examination the lung was found to have a fine 'grained' appearance like a reticulum all over it with obliteration of the hilar shadow and the term 'dust reticulation' was then introduced radiologically as well as clinically.

From the above date reticulation was admitted as an incapacitating industrial disease. It should be noted, however, that a disease is not a disability and is not certifiable as such until it suspends a man from his employment.

On some of the films of coal miners chests seen when they are examined for Silicosis, one finds about four or

five opaque areas extending up the lung field. These are like the appearance one sees in malignant secondary deposits in the lungs. These are known as 'finger print markings' and are supposed to be due to localised areas of Emphysema which have become infiltrated with coal dust and then been compressed.

Another extraordinary condition. which one sees radiologically and also on post mortem examinations is the 'cricket ball mass' which may or may not be seen in association with dust reticulation. There is always, however, an associated basal Emphysema.

These ' cricket ball masses' are an extraordinary phenomenon and are found on post mortem as black masses which cut like a briquette. They have a rind like an orange and inside there is simply a black oily mass which on section, stains the fingers with a tarry substance. They are full of silicates (not silica) and are soft in the centre presumably due to deficient blood supply causing a necrosis.

Enid Williams, who has been doing some work in this connection, says that they are all tubercular but Kettle states that they are inflammatory in origin.

One such case which we had in this district but which was not one of my own cases, finally pointed as an abscess just below the clavicle and discharged pus which was found to contain tubercle bacilli. I should hasten to add that there was no question of a tubercular rib or vertebra and the case had been previously accepted as one of Silicosis.

I have reproduced the X-Ray positive of this case as such cases have to be accepted as Silicosis because no other diagnosis is possible (Dr.C.L.Sutherland). These are unusual and interesting conditions and so far as I know they have not been described in any text book. Coal dust and Silicates are evidently the cause of them as the dust can be recovered from these masses on the post mortem table and they also serve to again illustrate the fact that coal dust is very far from being the harmless type of dust that one imagined it was. These masses are probably Anthraco-silicotic nodules which have become confluent.

Pneumoconiosis of coal workers may take many years to develope, although not as long as one might imagine. It has been found, for instance, that a screen worker will develope pneumoconiosis after about fifteen years exposure and in this connection it should be noted further, that in this district at any rate, young boys of fourteen years commencing work at the mine, usually start on the screens. They are not exposed to stone dust as much as the underground worker, but the screens being enclosed, the concentration of coal dust is extreme. In one such case, a boy of sixteen developed a very troublesome cough with expectoration of sputum in the mornings after only fourteen months work on the screens. His sputum was free from tubercle.

Similarly, coal trimmers at the docks it is stated, are equally prone to develope pneumoconiosis after about fifteen years exposure. Bathgate (1936) examined forty-four coal trimmers and found that eleven of these men were

unfit for work on account of their pulmonary condition and he agreed as to the occurrence of gross X-Ray changes in the lungs of men working at this occupation. One notes that he states X-Ray changes because on clinical examination of some of my own cases, little evidence was found of disease, although on X-Ray examination gross changes could be seen.

Gough, in 1940, published the first description of the post-mortem appearances in steam coal trimmers. He found that of twelve coal trimmers on whom post mortem examinations were made, eight showed marked fibrosis and in five of these, pneumoconiosis was the main cause of death; in the remaining four lung fibrosis was slight although not entirely absent. It was stated by Gough that the fibrosis was practically identical in appearance with the so-called Anthraco-silicosis of coal hewers and to differ in appearance from Silicotic changes as found in the gold miner or quartz worker.

The relation of coal dust to Emphysema: - It is the exception rather than the rule when examining a coal miner to find a non-emphysematous chest. Emphysema seems to be almost an occupational disease of coal miners. In the younger miner it is basal Emphysema, but in the older miner it is generalised, Dust, therefore, must have some effect in this production of Emphysema and as Emphysema is a progressive and incapacitating disease its prevention is of considerable importance. It is usually assumed that any loss of elasticity which is present in Emphysema is secondary to over-distension. This view is generally held

but Christie (Brit.Med.J. 22.1.44 p.105) believes that in Emphysema the enlargement of the air sacs and the fact that this enlargement is most conspicuous at the periphery of the lung can be explained as being the direct result of the loss of elasticity. This causes a voluminous lung and expansion of the thoracic cage as seen in Emphysema.

The damage to the elastic tissue seems to be caused by the dust. The main systems of the elastic tissue of the lungs is situated along the perivascular and peribronchial channels as well as at the margins of the lobula and these are the usual depots for dust. The result is, I think, that a fibrous tissue reaction is set up which immobilises and interferes with the elastic tissue so much that it is finally destroyed altogether. Silicates or silica itself may also be harmful to the elastic tissue which is situated in such close proximity to the depots of dust.

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RELATION OF PNEUMOCONIOSIS TO PULMONARY TUBERCULOSIS.

This relationship has always been a vexed one and many eminent authorities have held precisely opposite views. Haldane for instance (1916) was of the opinion that coal mining in general carried no special liability to Tuberculosis in spite of a relative excess of non-tuberculous respiratory disease in coal miners. He even went so far as to say (1931,1935) that the lesions which were being so increasingly described in Welsh coal miners could not therefore, be Silicosis, and he and some other authors suggested that coal dust might even be protective against tuberculosis (Wainwright and Nicholls 1905; Haldane 1916-18). Kettle, however, (1930) did not agree with this view.

Other writers held that the occupational environment e.g. the coal dust protects from Tuberculosis unless Pneumoconiosis occurs in which case subsequent development of the infection is favoured.

It is, however, generally held that there is a low incidence of tuberculosis,among bituminous coal miners. This was confirmed by an investigation in the Donetz coal-field in the Soviet Union in 1930-35. Only 1.6% clinical tuberculosis was found among 1,315 underground workers and this incidence was lower than in the two control groups viz 123 surface workers - 3.5%, and 546 wives of miners - 2.5%.

Similarly among the 987 underground workers at the

Gorovka Colliery (which also had a low incidence as regards pneumoconiosis) clinical tuberculosis was found in 1.4% (MRC.Ch.Pulm.Dis. 1943).

It should be mentioned that the Soviet workers do not agree with the view often expressed that coal dust or the fibrosis set up by it, has a protective effect against pulmonary tuberculosis. They consider that the general social and hygienic conditions in the life of the miner and natural occupational selection are two factors of special importance. That is to say that it is only men who are above the average in physical fitness take up this kind of work and men who are not up to this physical standard are quick to leave. Support is given to the existence of these tendencies by the data collected.

In 1933, Enid Williams studied one hundred old retired miners and found tubercle bacilli in the sputum of six of them. Four of these were face workers. All the sputum positive cases had only slight constitutional symptoms and in four the disease had not previously been suspected.

In another investigation (Sen 1937) examined one Hundred definite cases of Pneumoconiosis, half being rock workers and half being hewers, and found twelve positive sputa, these being equally distributed among the two occupations.

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anthracite miner. Consequently the low standardised mortality ratio for these miners (79) can probably be correctly ascribed to the occupation per se(MRC Ch.Pulm. Dis. in Welsh Coalminers 1943).

In my own series of cases, although there were two cases of positive sputa, one of which was not suspected, clinically recognisable pulmonary tuberculosis was not seen. Although in this district there has been an upward trend of deaths from Pulmonary Tuberculosis in males during the last two years, this is not excessive taken in common with the rest of the country, and there is very little disparity between males and females as regards new notifications of Pulmonary Tuberculosis.

These figures are:-

Year.	Males.	Females.	Total.
1940	33	17	50
1941	24	22	46
1942	38	35	73
1943	29	30	59

Thus it will be seen that except for the year 1940, the figures for males and females very nearly approximate. I have taken the figures for females as controls in this connection as they are not exposed to the dust hazard and one can reasonably draw the conclusion that dust has little relation to the incidence of pulmonary tuberculosis. I cannot subscribe to the Soviet view that the men are above the average of physical fitness as a fair proportion are definitely not.

Similarly when taken in age groups there is a marked increase in the number of new cases of pulmonary tuberculosis

in the twenty to thirty-five years age group for females and this age-group also accounts for almost half the total number of deaths from pulmonary tuberculosis. There has not been a corresponding increase in the males (MOH's Report 1943).

This would seem to indicate that (as practically the whole of the male population of this district is occupied in coal mining) there is no more incidence of pulmonary tuberculosis among coal miners than among the rest of the general population and that, therefore, pneumoconiosis of coal workers does not predispose to Pulmonary Tuberculosis.

This was borne out in my series of cases where, although in each case the sputum was examined at least once, and frequently several times, only one case of unsuspected tuberculosis was found.

Thus the incidence of Pulmonary Tuberculosis is much less in cases of pneumoconiosis of coal workers than it is among cases of classical Silicosis where it is reported to be the cause of death in 74% cases.

In Pneumoconiosis of coal workers, the commonest cause of death is right heart failure with non-tuberculous respiratory disease second. It is believed that typical tuberculous lsions are seldom found lying side by side with dust lesions, but rather that the two combine to form a common lesion typical neither of Tuberculosis nor of Pneumoconiosis. Both of these diseases have a tendency to localise in the lymph channels and if these are largely crammed with dust, it is possible that there is no room or suitable environment for

for the tubercle bacillus.

Another point worthy of mention is that from coal is derived most of our antiseptics, notably the Phenol group and it is possible that the coal dust is inimical to the growth of the tubercle bacillus for those reasons, whereas Silica, which is very slightly soluble in tissue fluids, forms a very suitable nidus for the growth of the tubercle bacillus.

The modified form of Tuberculosis which is found in these cases of dust reticulation is so much modified that the tuberculous element may be scarcely recognisable except by finding the tubercle bacillus. The dust lesion predominates and the resulting lesion has the character of a dust reticulation plus a caseous fibrosis.

Thus Pulmonary Tuberculosis as found in the coal miner is a modified form of tuberculosis, being more fibrous, more widespread and more chronic. It is at the same time, less cellular and does not form giant cell systems. The most striking change is, however, the fact that the lesion is so heavily impregnated with dust. Even the caseous areas are saturated with dust. Thus it is that the tuberculous element in these cases is frequently missed and in one of my own cases, Case 1, the tuberculous character was completely missed despite a careful clinical and radiological examination until for the purpose of this thesis, his sputum was examined when tubercle bacilli were found.

It is likely that the tuberculous process is modified in two ways - firstly by the Silica (or silicates) exerting

an increased fibrous reaction response and probably favouring the growth and disseminination of the tubercle bacillus and secondly by the coal dust exerting an inhibiting influence by either adsorbing the tubercle or exerting some chemical process which is unfavourable to the tubercle.

In other cases such as Case 5, Pulmonary Tuberculosis is suspected both clinically and radiologically (see X-Ray plate) and yet the sputum remained persistently negative. This case seemed to be a combination of dust reticulation and pulmonary tuberculosis. A new term has been introduced for these cases namely Koniophthisis . Case 1. of my series is such a Koniophthisis and is now in receipt of a full scale disability pension. This raises the question of whether caseation may be seen only in tubercle or whether it may be produced by dust lesions alone. It should be remembered that the centre of a Silicotic nodule is necrotic probably due to the fact that there is a deficient blood supply in the centre, and similarly in these cases of Koniophthisis, one sometimes finds a cavity where none was suspected.

My own impression of this case (Case 5) is that this case had a latent Tuberculosis in his left upper zone (as seen in accompanying X-Ray) and that it was essentially a fibroid phthisis from the start and was slowly progressive over many years possibly in conflict with the inhibitory influence of the coal dust in his lungs. However, in the end the man died apparently from cardiac failure due to chronic bronchitis but most likely from Koniophthisis - to use the new term.

In conclusion one may therefore say that Pneumoconiosis does not predispose or protect from tuberculosis and that the figures in this district seem to indicate that coal mining is not an unhealthy occupation as regards pulmonary tuberculosis. When, however, pulmonary tuberculosis does develope in a case of Pneumoconiosis the two diseases are modified by each other to form a common lesion which has been named Koniophthisis.

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CLINICAL MANIFESTATIONS OF PNEUMOCONIOSIS.

The patient is usually elderly as the dust takes a considerable number of years to produce affects. Some 76.4% of my cases had been exposed to the dust hazard for thirty years or more. That is to say they were upwards of forty-four years of age.

They then find that they are becoming increasingly breathless at work. They cannot keep up with their younger or healthier, colleagues. They also find that in their spare time they are unable to go for long walks as many miners are accustomed to do.

Frequently at this stage, they have an attack of Bronchitis which leaves them with a persistent hard cough without the expectoration of any sputum. Clinically it appears to be a Chronic Bronchitis without any sputum.

Thus the patient now complains of two things - breathlessness and cough. One and all of my own series of cases complained of these two symptoms.

On examination there is little to find - some tachycardia with a pulse rate of about 100 per minute. Possibly a somewhat increased respiratory rate as well - 20 to 24 respirations per minute. In addition, the chest expansion is poor, averaging 1" to 1½". There may be some basal emphysema, but not to any marked extent. Other signs include - superficial venules around the line of attachment of the diaphragm as noted in Case 7. Then there may be some clubbing of the fingers Case 1, 6 and others, and

finally such conditions as ossified costal cartilages and suppression of thoracic breathing.

Later the cough may become somewhat looser with expectoration of a little greyish black tenaceous sputum. The patient at this stage is usually very distressed on account of his troublesome cough disturbing sleep. In the early morning he falls into a fitful doze only to be wakened by a further paroxysm of coughing. One notes that frequently they have to get up an hour before their usual time of rising in order to cough themselves clear. Others again have to get up and actually go out into the dawn as the fresh air helps them.

At this stage the patient is labelled Chronic Bronchitis unless one bears in mind that such cases may be Pneumoconiosis of coal workers. Even such a diagnosis can only be made after further investigation including radiological examination.

This latent Pneumoconiosis is the cause of a serious amount of respiratory disability among coal miners, although the affected men are often able to continue their accustomed work for some years within a limited range of effort. To take my own cases, some 60% of them are completely off work and the others have only light work without any responsibilities. Even so they rarely work for more than a few weeks without having to stop off work for a week or so. There is thus put upon themselves and their families a financial burden which, by inadequate or unsuitable foodstuffs and living accomodation causes further illness and so the vicious circle goes on.

On examination of the patient at this stage, little is to be found except some emphysema and associated dyspnoea. The condition going further a pulmonary fibrosis is set up causing this poor physical condition to become permanent. The man becomes a liability to himself, his family and the State.

This condition does not, however, remain quiescent - the ciliated epithelium has by now been destroyed and fibrosis and emphysema have further damaged the respiratory function of the lung. Thus one finds clinically that the patient is now grossly dyspnoec and is cyanosed, sibilant rhonchi are to be heard all over the chest and there is a general break-down in the cardio-respiratory system.

The contraction of the fibrous tissue in the lungs causes a dilatation of the bronchi and a final state of Bronchiectasis is produced. Finally, when secondary infection of the Bronchiectatic cavities having occurred, the patient's condition is pitiable in the extreme, Cyanosis, Dyspnoea, constant cough disturbing sleep, anorexia, right sided cardiac failure and loss of weight being his lot, with death not far off.

At any time during this decline from health, pulmonary tuberculosis may become a superadded infection and so further complicate the picture.

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TREATMENT AND REHABILITATION OF COAL MINERS SUFFERING FROM PNEUMOCONIOSIS AND ALLIED RESPIRATORY DISEASES.

At the outset I would state that the best form of treatment is prevention. Prevention that is, of the dusty atmosphere which causes these respiratory diseases and in an occupation like that of coal mining, this is extremely difficult to carry out.

It has been found that the chief source of the fine dust which is so inimical to the health of miners is that dust which is situated at the line of cleavage between the coal seam and the adjacent strata. This led to various forms of water treatment such as the Water Infusion method and the Mist Projector. In seams using coal cutters, the 'wet cutting' method was introduced to prevent the dust becoming air borne. The result of these measures ensures that a very substantial reduction is obtained in the concentration of air borne dust. Very little water is used so as not to interfere with the relative humidity of the atmosphere in the working place. The time of the miner is not wholly taken up with these preventive measures so that they will willimgly co-operate in these duties without supervision.

Other methods of allaying the dust have been tried by means of such 'wetting' agents as Perminal and DS103 - these act by decreasing the surface tension of the dust particles and so preventing the dust from rising.

These 'wetting' methods also help to diminish the dispersal of dust during shot-firing.(MRC Environment

I would like to mention, however, that it is difficult to get the wholesale co-operation of miners in these methods of suppressing dusts and when talking to them, they tend to depreciate these precautions and to blame them for causing Rheumatism, Lumbago etc. They have homely methods of their own for preventing dust inhalation - they chew tobacco or, in the case of younger miners, they tie a handkerchief over their mouths and noses. I do think, however, that they are becoming more dust conscious of late and as time goes on will co-operate more readily with this menace to their health.

Treatment of Pneumoconiosis: There is very little known about the treatment of this condition. In my experience, the treatment hitherto has been entirely symptomatic. Postural drainage appears to help some advanced cases as in Bronchiectasis, for it is my opinion that Bronchiectasis can be produced by the fibrosis of the lung induced by coal dust. Other writers advocate vaccines and general tonic measures, fresh air, adequate nourishment (which may be difficult to get if the patient is not working) and possibly actinic ray treatment.

Recently, however, Denny Robson and Irwin have published interesting accounts of the treatment of Silicosis by the inhalation of Aluminium powder. They state that if one assumes that silica exerts its injurious effects upon the lung by a slow transformation into silicic acid then if the solubility of siliceous material in the lungs could be sufficiently reduced the usual fibrotic response could be diminished or prevented (B.M.J.p 601. Nov.4th 1944). They

further found that aluminium powder rendered siliceous material insoluble in vitro by coating the silica particle with a thin film of gelatinous hydrated alumina which, on drying, formed crystalline aluminium monohydrate. By animal experiments it was found that metallic aluminium dust produced no fibrosis in the lungs. It was therefore argued that by mixing aluminium dust with quartz, Silicosis would be prevented. Animals exposed to quartz dust developed Silicosis in about five months, but the addition of 1% aluminium powder prevented this even after the period of exposure had been lengthened to twenty-two months.

This last year (1944) Crombie Blaisdell and MacPherson have reported the treatment of thirty-four cases of Silicosis in miners by the daily inhalation of metallic aluminium dust. This dust was freshly ground in a special grinder from small aluminium pellets.

The treatment was begun by giving five minute inhalations and increasing to thirty minutes. Some of the cases received up to three hundred treatments but the majority only had two hundred. Clinical improvement was noted in nineteen cases by a decrease in the breathlessness, improvement in the cough, tightness in the chest and fatigue. In the remaining fifteen cases there was no improvement, but the condition remained stationary in spite of daily employment in silica dust throughout the treatment.

Crombie and his colleagues believe that the inhalation of fine aluminium dust prevents the developement of Silicosis. Certainly these researches open up a new field in the

in the prevention of occupational dust disease.

Rehabilitation: To me it appears unlikely that rehabilitation will enable men to return to their former occupation, and in any case, it is definitely harmful for them to return to any dusty occupation and especially to coal mining.

The dust reticulation stage appears to be more or less static if the man is not again employed in any dusty occupation. If, however, he returns to a dusty occupation he is liable to develope further fibrotic lesions to the stage of massive shadows or pulmonary tuberculosis may develope as occurred in Case 1 of my series.

My impression is that the men should be treated on the lines of Tuberculosis patients, that is encouraged to lead an outdoor life, to have a nourishing diet, to avoid alcohol and tobacco and to have a sufficiency of rest. Also X-Ray and clinical examinations should be carried out at frequent intervals, not only to gauge the patient's progress (or otherwise) but also to provide a basis for the further study of the condition.

The above remarks refer chiefly to those who are totally disabled not only from Silicosis but also from what has been called Chronic Bronchitis, Pulmonary Fibrosis, Emphysema and Bronchiectasis. All these conditions if they entail disablement, can be grouped together under the term Pneumoconiosis of Coal Workers.

In the case of the dust reticulation case, however, light work away from dusty atmospheres can be undertaken.

This should not entail work in a confined atmosphere if possible, and the patient here too should have frequent clinical and X-Ray examinations.

Recently the Advisory Committee of the Ministry of Fuel and Power has published a report (Stationery Office) which states that there is insufficient knowledge available to recommend or advise large scale measures of treatment including rehabilitation. The Committee states that further research is necessary with a view to obtaining more accurate knowledge of the disease - its cause, progression, treatment and rehabilitation.

It further recommends the establishment of a Treatment and Rehabilitation Centre with accomodation for thirty in-patients, equipped with facilities for clinical study and research. Other recommendations are - frequent and periodic clinical and X-Ray examinations of miners and facilities for guidance in the matter of dust suppression. Also the provision of facilities for pathological research into the early dust changes in the lung, the progression of the lesion and the part played by tuberculosis and other infections.

Finally it is recommended that a Pneumoconiosis Bureau be established to co-ordinate all aspects of work in connection with Pneumoconiosis.

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MEDICO-LEGAL ASPECTS OE PNEUMOCONIOSIS.

Chronic Pulmonary disease of coal workers in relation to the Workmens Compensation Act. This has always been a complicated problem and as the knowledge of the condition has improved it has necessitated various amendments to the already existing Acts of Parliament and even the introduction of new Acts.

The original Workmens Compensation Act was introduced in 1897 but it was not until 1918 that Silicosis itself became a 'scheduled disease' for which compensation was payable. This Act made arrangements for compensation in the Refractories Industries a small group of industries making material refractory to heat for building furnaces. To delimit these industries from the fireclay industry the material worked with by those who became entitled to compensation had to contain 80% Silica (SiO_2). No extension of compensation to other occupations was made until 1928 when, under Section 47 of the Workmens' Compensation Act 1925, the Secretary of State introduced several schemes. The scheme in which coal miners were interested was the Various Industries (Silicosis) Scheme 1928 S.R. and O.1928 No.975. The Medical part of this scheme was put in the hands of the Medical Referees under the Workmens Compensation Act. These schemes extended compensation to workers in certain occupations and did not extend compensation merely by virtue of having the misfortune to have the disease. It was thought that the coal mining risk was adequately covered by the definition in the scheme which applied it to those employed 'drilling and blasting in Silica rock in or incidental

to the mining or quarrying of other minerals'. It should be noted here that Silica rock did not include any rock containing less than 50% free Silica - it included quartz ganister, sandstone, gritstone etc. The coal miner to get compensation under this Scheme had to be totally disabled by the disease and further, he had to have worked after the 1st January 1929 in drilling rock containing at least 50% Silica.

This of course fulfilled the expectation that these sufferers would come from hard heading workers only, since the available information was more or less that obtained by Dr.Tattersall. But it was soon found that hard headers did not only work in Pennant Stone - the only thick seam of rock in South Wales pits containing over 50% Silica. Hard headers drive through all sorts of seams of varying types of shales and rocks and it was very difficult to prove their chemical composition. This difficulty was thought to have been overcome by an amending scheme in 1930 (1930 S.R. and O. 1095) which dropped the percentage in the definition of Silica rock and added two clauses specifically mentioning coal miners '(a) drilling or blasting in stone in any sinking pit and in stone drifts or other straight or narrow working in coal mine or (b) operating any power driven machine used for drilling, cutting, ripping or breaking stone in any coal mine'.

On the 1st June 1930, a new Various Industries Scheme (1931 S.R. and O. 342) came into operation in which compensation was extended not only to those totally disabled but to cases of suspension (in other words partial disablement)

as well. The present Medical Board were put in charge of the Medical part of signing the necessary certificates.

From 1st June 1931, one can trace the number of claims from all over the country - the vast majority of the claims have been from South Wales - at least 97%.

Year.	Total Disablement.	Suspended.	Refused.	Total Claims.
1932	15	12	4	31
1933	88	34	52	174
1934	136	63	83	282
1935	156	55	155	366
1936	162	66	264	492
1937	243	76	355	674
1938	175	92	341	608
1939	271	164	389	824
1940	199	246	412	857
1941	185	284	488	957
1942	184	342	769	1,295
1943	286	514	1,144	1,944

In 1932 and 1933 the Medical Board issued certificates to applicants who found that they were unable to obtain compensation because the employers contended that they had not worked in a process covered by the two new clauses. The difficulty was in the fact that the employers took advantage of a proviso to these clauses to prove that the workmen had not 'been exposed to the dust of Silica rock'. They had only been engaged as colliers in the usual ripping of 'top' and 'bottom' but had not bored in Pennant rock - the local sandstone. A case was taken through the County Court, Appeal Court and House of Lords, and the final

decision was given not on the two clauses mentioned but on another clause which included processes involving 'breaking, crushing, mixing any dry deposit of silica or dry admixture'. This man (Morgan v Tirbach Amalgamated Anthracite Co.) had been employed at a colliery called Tirbach in which no silica rock that is gritstone or sandstone is found. He had been a collier in the 'pillar and stall' system and had done top and bottom rippings in shale stone. Very elaborate discussion as to what a 'deposit' or 'admixture' of silica was, were carried on by Lords Hanworth, Slessor and Romer. The House of Lords (Lords Atkin, Tomlin and Russell of Killowen) decided in favour of the workmen because this shale contains narrow veins of quartz called lenticles ranging up to ¾" in thickness. These lenticles are undoubtedly silica rock said their Lordships and that the breaking of the top and bottom caused the workman to be exposed to the dust of silica rock.

Before the House of Lords decision, so much pressure had been brought on the Home Office that the Scheme was amended (S.R. and O 1934No.1155) to apply to workmen employed underground on or after the date of the order (namely 22nd October 1934) so that the processes included 'any operation underground in any coal mine'.

Meanwhile the number of claims coming to the Medical Board steadily increased from 1933 to 1936, partly owing to the greater freedom of applications to the Board given by the amendment to the Scheme and partly by the publicity given to the Court case. The peak of applications for

certificates seemed to have been reached in 1936 since there was a drop in the claims in 1937. But although the applications were increasing, the proportion of refusals to certify was also going up and in 1936 it was recognised that these refusals were due to the fact that the applicants although suffering from a chest condition most probably caused by their work were not suffering from Silicosis in the accepted sense. It is around this fact that I have attempted to base this thesis, for one sees in industrial practice so many miners, who, although not suffering from Silicosis, yet suffer from a chronic chest condition such as Chronic Bronchitis or Emphysema that suspends them from work very frequently or completely disables them.

The attention of the Pulmonary Diseases Committee of the Medical Research Council was drawn to this fact in 1936, but it was not until 1938 that the special investigations by Drs.Hart and Aslett was arranged for. In 1938 owing to the collapse of the Franc and the loss of the French market, many pits in South Wales were closed, and this led to an increase in the claims and also to a diminution of the proportion of refusals. Thereafter, however, the refusals increased in proportion and the total disablement certificates relatively decreased. This relative decrease in total disablement certificates could be due to a number of causes:-

(1) The natural elimination of the gross cases.

(2) The greater knowledge of the widespread incidence of the condition in miners.

(3) The desire on the part of the younger miners to get out of the mines especially as the avenues of alternative employment were increased by the setting up of ordnance factories.

The report by Drs. Hart and Aslett was published in 1942 (Special Report Series No.243: Chronic Pulmonary Disease in South Wales Coal Miners (1) Medical Studies). As a result of its publication Parliament amended the Workmens Compensation Act Section 47 to allow the issue of a fresh scheme (The Coal Mining Industry, Pneumoconiosis, Compensation Scheme 1943. S.R. and O. 1943 No.885). This provides for compensation to those miners suffering from 'dust reticulation' and replaced the Various Industries Scheme for Coal Miners who were working in the pits after July 1st 1943.

A further Scheme the Pneumoconiosis Benefit Scheme (S.R. and O 1943. No.886) was introduced at the same time to provide a benefit (not compensation) for miners suffering from Pneumoconiosis who had been employed in the coal mines from October 22nd 1934 to June 30th 1943 who were not entitled to compensation under the Schemes. The introduction of this Scheme greatly increased the applications from South Wales and also in other districts including Durham and Northumberland, but it is too early yet to say whether conditions comparable with South Wales will be exposed in other coal-fields.

This short history of the Compensation side of the question illustrates the difficulties in trying to meet a situation which is being influenced by factors quite apart from the workman. One of these factors is the diagnosis of

chest conditions by means of X-Rays. This requires a good radiographic film which in turn requires a good X-Ray apparatus. For the diagnosis of Silicosis and dust-reticulation a technically good radiographic film forms 'the most reliable single factor', an expression which occurs in most of the reports of the South African Miners Phthisis Medical Bureau and is repeated in the reports for the eight years ending 1938. A good X-Ray film of the chest was not very easily obtained and up to 1928 most X-Ray plants were not capable of showing the conditions we now call dust-reticulation. The radiological investigation carried out by the Welsh National Memorial Association led to the conclusion that the mottling was similar to that caused by the inhalation of Silica dust in cases from the eastern part of the South Wales coalfield, but in cases from the western side, that is the Anthracite area, the mottling is definitely finer than that observed in typical films from silicotic gold mines in South Africa.

The cases coming to the Silicosis Board in the early days were in the vast majority advanced cases which complied with the radiographic definition of Silicosis laid down by the International Conference at Johannesburg in 1930. The fact that amongst those certified was a considerable proportion of men who had never worked in stone, led to criticism of the Medical Board chiefly by Prof. J.B.Haldane. The difficulty about the lack of a silica exposure had been met with by the Medical Referees in South Wales before the Medical Board took over and had led in death cases to chemical analysis of the lungs. Up till then it had been

thought of in terms of Silicon Dioxide (SiO_2) or crude silica and it had been thought that this was easily estimated. In South Wales, however, the chemical analysis was for total silica since Dr.Sladden (Silica Content of Lungs 15th July 1933) and others found serious difficulties in the satisfactory determination of free silica in lung tissue. Even so, however, the determination of the silica content of the lungs after death, even if one accepted the chemical analysis as diagnostic, gives no help with living cases.

The writer is greatly indebted in this chapter to the help, advice and notes of Dr.C.L.Sutherland, the Chief Medical Officer to the Silicosis Board.

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CLINICAL SECTION.

CLINICAL CASES.

CASE 1. Koniophthisis.

Anthony H. Age 63. Coal Hewer.

Complains of Cough and breathlessness.

Duration - 4 years.

History:- This man has been employed as a Coal Hewer for 41 years. He then worked in the "Waste" for a further 9 years. He states that he developed a cough 4 years ago following an attack of Influenza. The cough is worst in the mornings, usually about 6a.m. when he has to get up and go into the fresh air, when he coughs for an hour or more and expectorates much blackish green sputum. He is very breathless on exertion and it is on this account that he has had to give up his work altogether.

Family History:- Nil of note. Both parents died at an advanced age.

Habits. Non- smoker and very moderate drinker.

Examination: This man is obviously very dyspnoec as when taking his clothes off for examination. He is tall and very thin but not emaciated.

There is definite clubbing of his fingers.

Resting pulse rate 94 per minute.

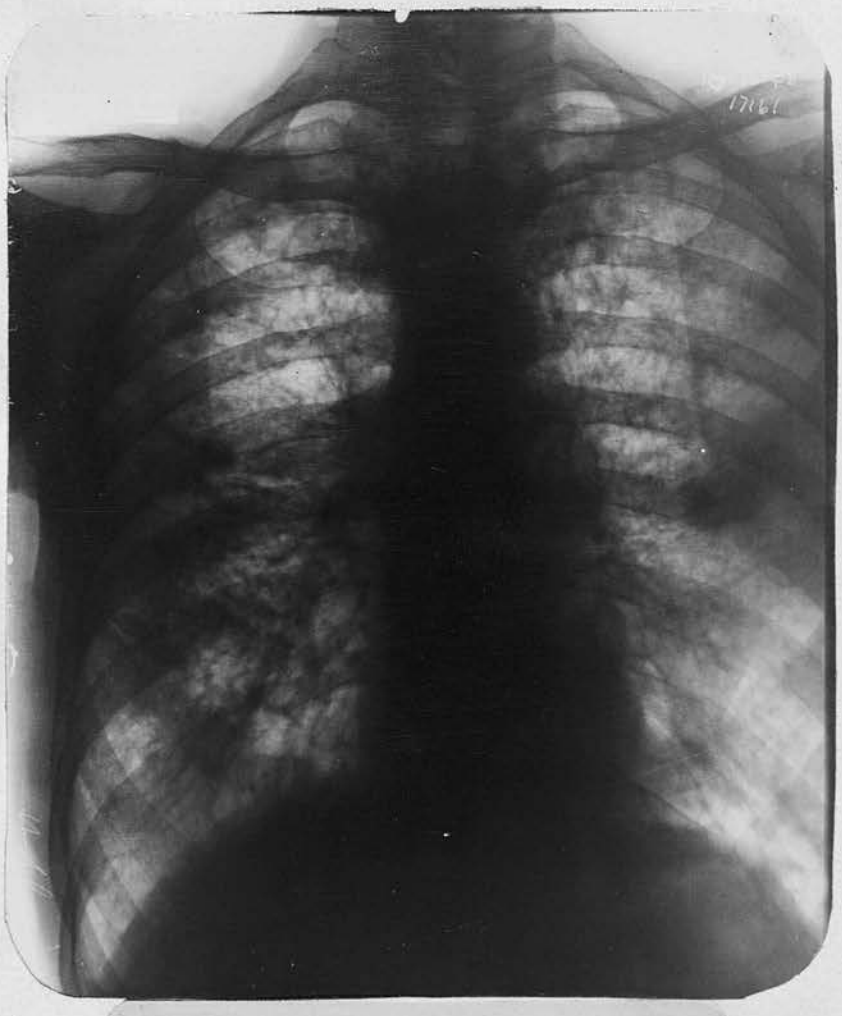
Resting respirations 20 per minute.

No cyanosis and no oedema of the feet.

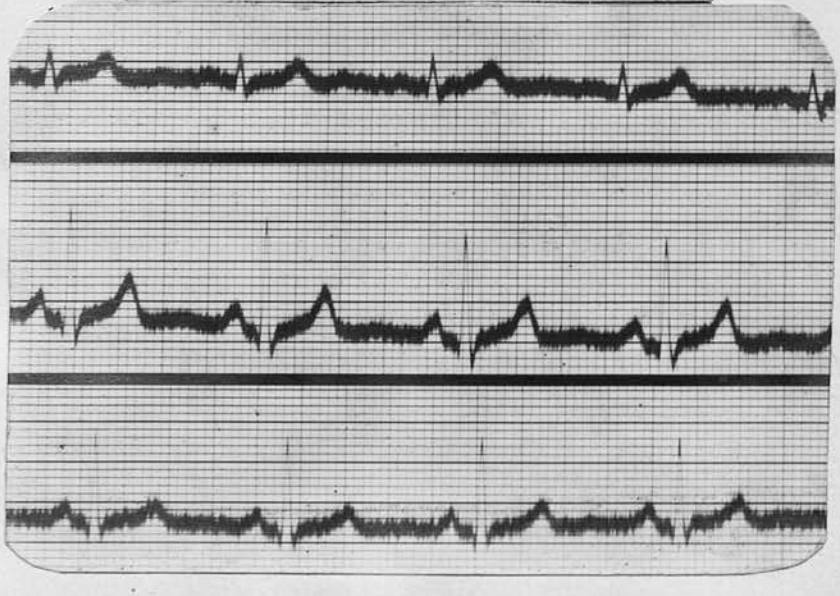
Chest moves very little on respiration which is of the abdominal type.

Apex beat in 5th. interspace 2½" from the mid line.

Chest expansion 38" - 39" ie 1" expansion.



CASE 1.



Vocal fremitus diminished.

Tympanitic percussion note all over the chest with diminution of superficial cardiac dullness two fingers-breadths to right of nipple line. Myotatic irritability apparent on percussion. On auscultation the vocal resonance is diminished. Vesicular breath sounds heard all over the chest with an occasional rhonchus.

Heart sounds normal and healthy.

Blood pressure 140/90 mms Hg.

X-Ray Examination. Skiagram shows extensive reticulation and mottling over all lung zones. He has extensive pulmonary fibrosis.

Sputum Examination. Found - a few Tubercle bacilli. Later examinations showed numerous tubercle bacilli.

Electrocardiogram shows a high peak in the T wave in Lead II. The Q wave is apparent in Lead III only. Otherwise no cardiac abnormality is present.

This man has now been certified by the Silicosis Board to be suffering from Koniophthisis and has been awarded compensation.

Koniophthisis is a condition in which Tuberculosis and the dust condition combine to form a common lesion characteristic of neither. Thus a modified form of pulmonary Tuberculosis is produced which may be scarcely recognisable as such unless the sputum is examined. Usually the dust lesion predominates and the resulting condition has the character of a dust reticulation plus a caseous tuberculosis.

In this case the Tubercle bacillus was finally isolated in the sputum but in the next case, no Tubercle bacilli have so far been found, although radiographically the case is also one of Koniophthisis. Thus the diagnosis may easily be missed if no radiological examination is carried out.

-ooOoo-

CASE 2. Koniophthisis.

Peter C. Age 67 Scalloper (Coal Cutter)

Complains of cough which has been worse during the last five months but has been present for years.

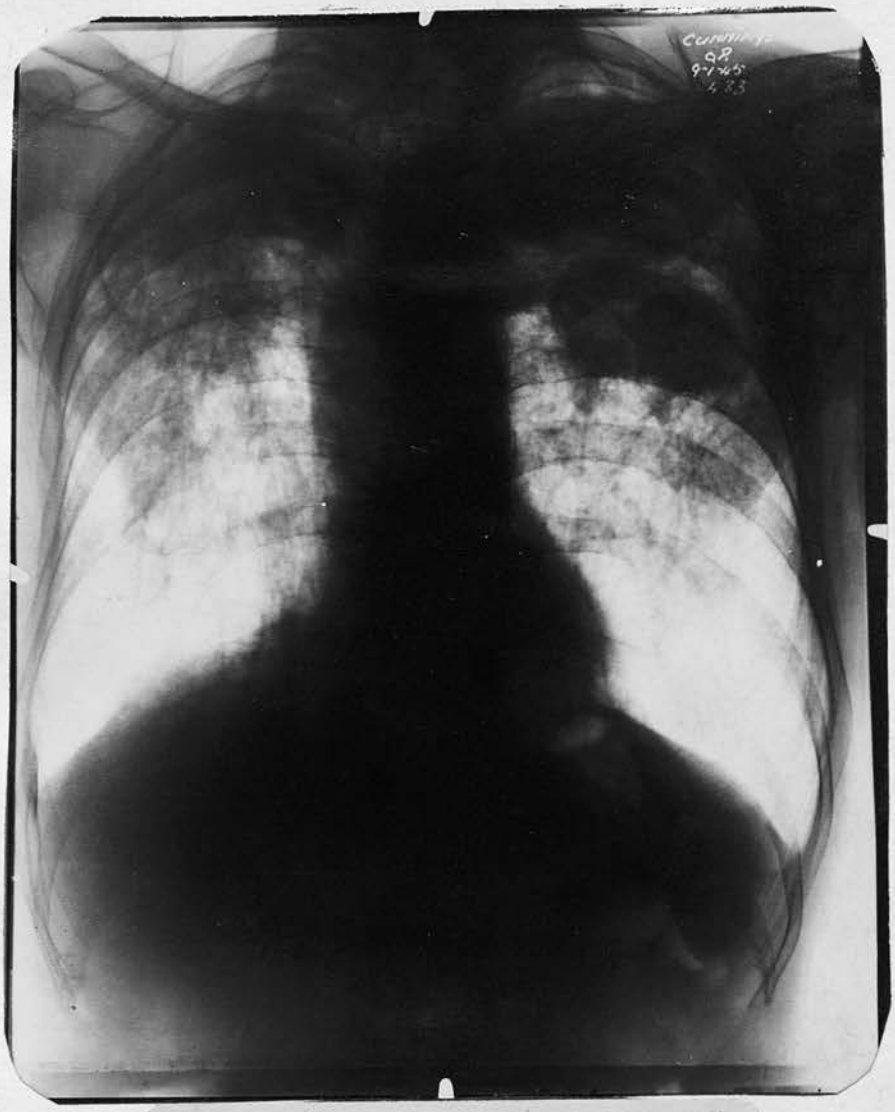
Duration - 10 years but much worse during the last five months.

History: This man started work at the age of 13½ as a pump boy but later began to 'hand put'. He served in South Africa in the Boer War from 1899 until 1904, when he was demobilised. He recommenced work as a coal hewer and scalloper. This last is a coal cutter operator and he remained at this work for 31 years, some 21 years as a coal hewer and 10 years as a scalloper. The scalloping machines were operated by compressed air and they raised a tremendous amount of dust much of which was stone dust from the dust which was scattered around to prevent explosions. For the last 10 years he states he had a slight cough but nothing much to complain about and as he states "everybody on the coal cutters had a cough". He is breathless on exertion and cannot walk (even on the flat) very far before he has to stop for breath. The cough is very bad in the mornings - he has to cough for a long time every morning before he gets relief. Blackish green sputum is expectorated. He had a slight haemoptysis about 18 months ago.

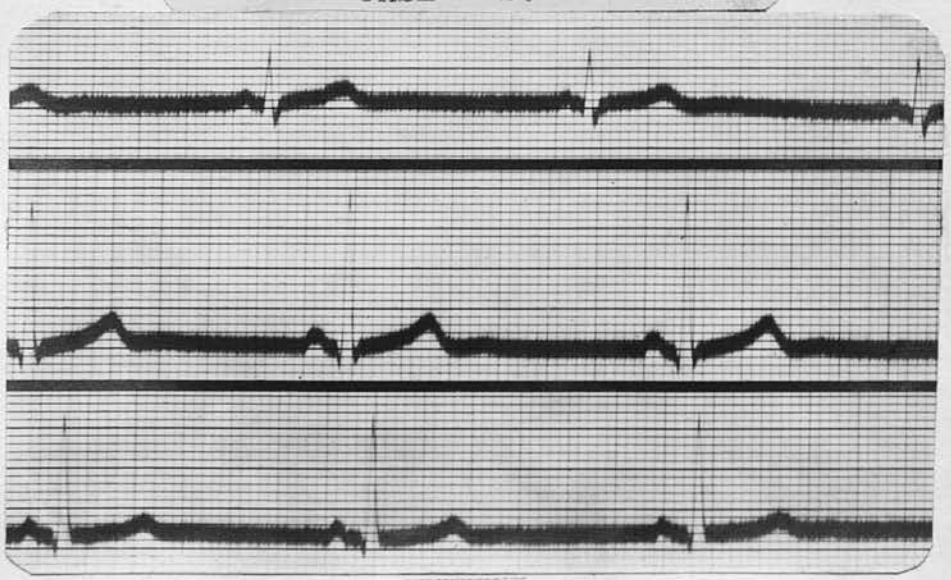
Family History: Both parents are dead but they were both over 80 when they died.

Habits: Smokes 2 ozs. of tobacco a week. Only drinks an odd glass of beer.

Examination: Very frail, almost emaciated man. He looks



CASE 2.



much older than his years. Muscles thin and wasted and exhibiting myotatic irritability and myoedema.

Breathing is entirely abdominal in type. Costal cartilages ossified. Slight scoliosis of the spine. No clubbing of the fingers.

Resting pulse rate 80 per minute.

Vessels arterio-sclerotic.

Chest measurements 33" - 34" ie 1" expansion.

Apex beat in 5th interspace $3\frac{1}{2}$" from the mid line.

Superficial cardiac dullness 1" within the nipple line.

Impaired percussion note at right apex - elsewhere it is definitely hyper resonant.

Vocal fremitus diminished except possibly at the right apex.

Cavernous breathing at right apex. Elsewhere it is bronchovesicular. No accompaniments.

Vocal resonance diminished except possibly at the right apex. Heart sounds normal.

Blood pressure 160/100 mms Hg.

Weight 7st. 11lbs. Height 5' 7".

Sputum examination negative.

X-Ray Examination: Old standing reticulation of pneumoconiosis with superadded Tuberculous areas in both upper lobes. Evidence of Koniophthisis.

In his history card the first mention of Bronchitis is in January 1942 although as long ago as 1921 he had an attack of Pleurisy. In.Jun 1942 this man sustained a bruised chest. An X-Ray examination shows no sign of fracture. There is no mention made in the X-Ray report of any pulmonary disease.

In 1940 he was discharged from hospital with the following report - "Spondylitis of the cervical vertebrae". Again there is no mention of any pulmonary disease although he had evidently had yet another radiological examination.

Electrocardiogram: Shows normal tracing with no cardiac abnormality.

-ooOoo-

CASE 3. Silicosis and Tuberculosis.

Joseph E.R. Age 43. Master Shifter.

Complains of cough and breathlessness. Also loss of weight and lassitude.

Duration - 2 years.

History: This man is a master-shifter, that is an underground official. Previous to this he worked as a stoneman for 6 years. He states that he was quite well until he had an attack of left sided Pleurisy in 1941. He made a poor recovery from this and never felt completely well. He had another attack of Pleurisy in 1942, this time on the right side. After this second attack he had to give up work altogether on account of increasing breathlessness. He also had a persistent cough with much sputum and felt generally weak and easily tired. His appetite was poor and he was confined to bed.

Family History: Nil of note.

Examination: The patient is confined to bed and is in very poor general health. Skin dry and without elasticity. He has a frequent short cough and is very breathless.

No clubbing of fingers. Myotatic irritability present.

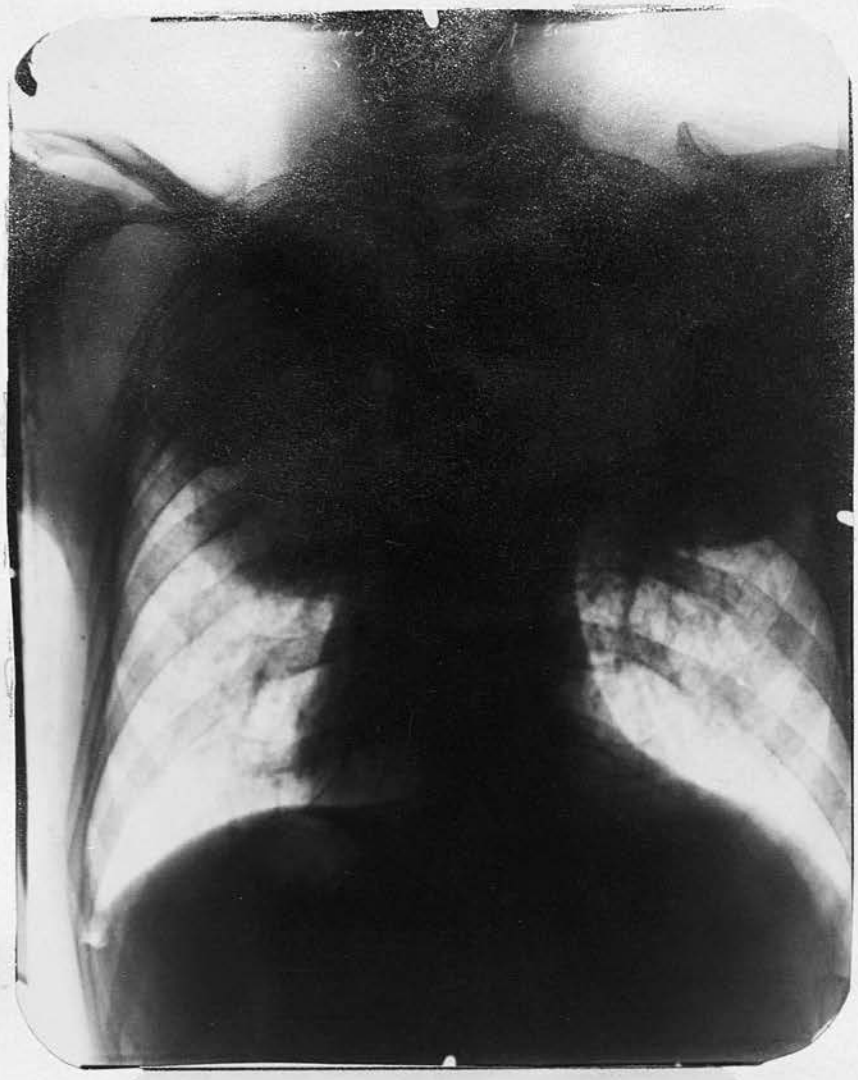
Resting pulse rate 100 per minute.

Chest moves very little on respiration which is mainly of the abdominal type.

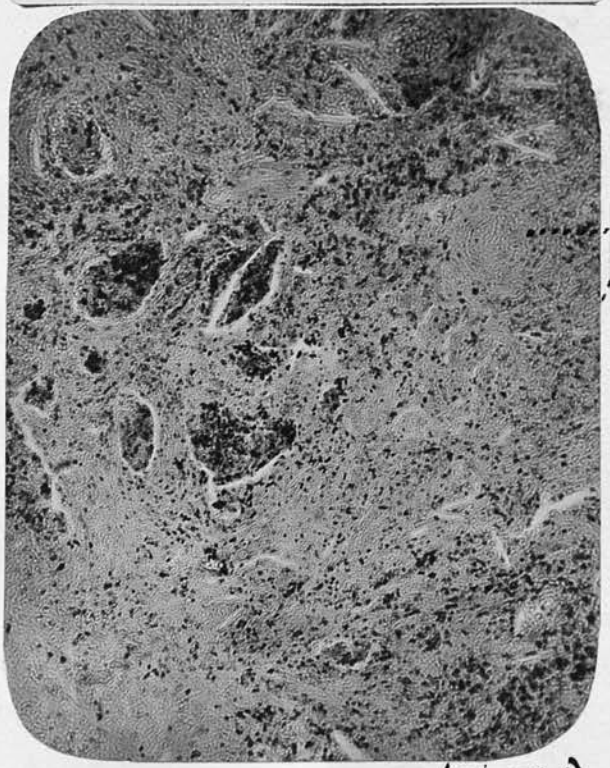
Apex beat in the 5th. interspace $3\frac{1}{2}$" from the mid.line.

Vocal fremitus increased at both apices.

The percussion note is impaired at both apices and also in both mid and upper zones.



CASE 3.



SILICOTIC NODULES

SECTION FROM CASE 3. (BIOPSY)

On auscultation there is prolonged expiration and rhonchi can be heard all over the chest with the exception of the apices.

Vocal resonance increased at both apices.

Heart sounds normal. The pulse is regular but rapid.

Temperature normal.

Sputum negative to Tubercle bacillus.

X-Ray Examination: At this stage he was X-Rayed and the radiographs show massive shadows in both lungs which may be due to Silicosis or neoplasm.

This left the diagnosis in doubt and as the patient refused to have a biopsy done things were at a standstill until a new developement arose. In February 1943, a diffuse swelling appeared in the left upper thorax and left axilla. At the same time the patient became much worse - very anaemic and cachectic. This appeared to confirm the diagnosis of pulmonary neoplasm and he was recommended to see a Thoracic surgeon. He was accordingly admitted to hospital for investigation and the swelling in the left infraclavicular region was explored and found to be an abscess which seemed to be coming from the chest like a fistula between the ribs. The pus was traced to an area of necrotic lung a piece of which was sent for section and found to be silicotic. In addition tubercle bacilli were isolated from the pus and the diagnosis of Silicosis complicated by Tuberculosis was confirmed. Later his sputum, hitherto persistently negative, was found to be positive to Tubercle.

This man died shortly after in June 1943, being admitted

as a case of Silicosis by the Silicosis Board.

This remarkable case was, therefore, a Silicosis complicated by Tuberculosis in a bitumenous coal miner. He worked in a coal mine in this neighbourhood in a mine in which there have occurred many cases of Silicosis. The coal miners around this district have very few such cases and the only difference is that the mine where this man worked is some 2,000 feet deep, while the other mines are only around the 1,500 feet mark.

The writer has no doubt that had a post mortem been available a 'cricket ball' mass would have been found in the left infraclavicular region of the lung - a black tarry mass of necrotic lung tissue and coal and stone dust.

-ooOoo-

CASE 4. Dust Reticulation.

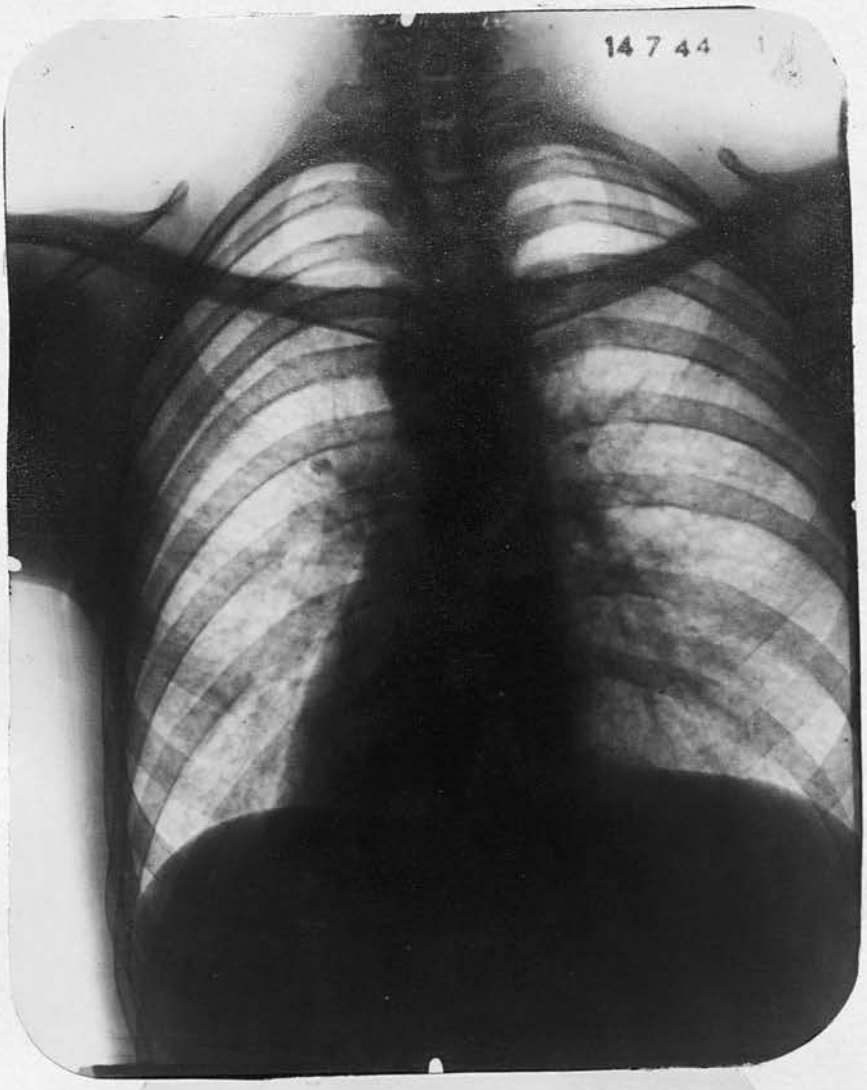
Ralph K. age 31. Coal Cutter.

Complains of cough and slight dyspnoea on exertion.

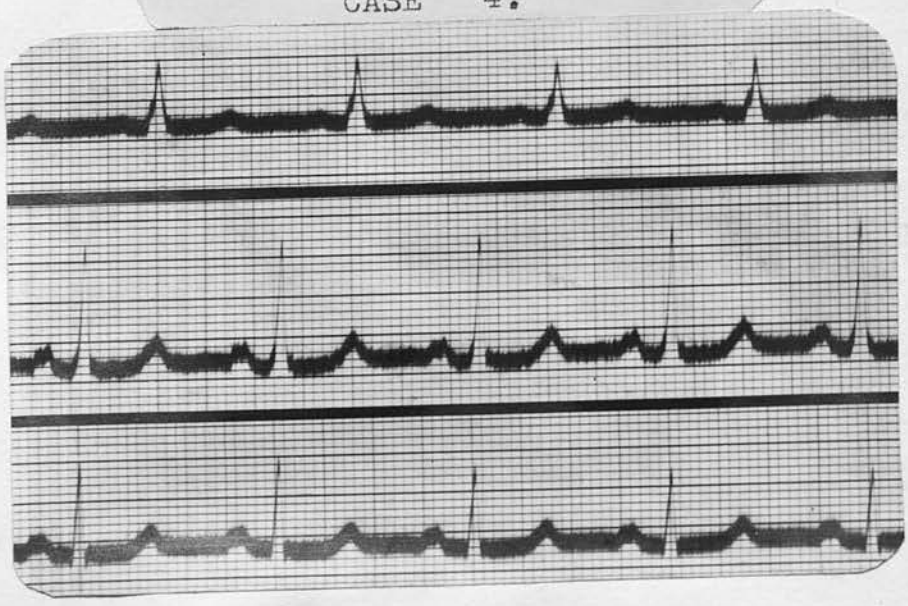
Duration - 5 years.

History. Some five years ago he had a slight cough with black sputum which never seemed to improve much. He thought he had 'catarrh' but did not take much notice of it. This cough persisted for some time and then he had a serious accident - he sustained a 'broken back' (fractured transverse processes 7th - 8th Thoracic Vertebrae) He was in a plaster jacket for 18 weeks and then went back on to light work at the surface where he remained for some 8 months, but for the last 18 months he has been a labourer underground. About 2 months ago his cough got worse and finally he had to stop work and stay in bed. He had a severe attack of acute Bronchitis with high temperature, drenching sweats and considerable prostration. He improved somewhat but a slight pyrexia remained for a fortnight and that, together with the fact that he had some moist crepitations at the left apex led me to call the Tuberculosis Medical Officer in consultation. He was X-Rayed and a final diagnosis of non-tubercular pulmonary fibrosis was made.

He was employed as coal cutter until the accident occurred. His work entailed cutting stone also and he describes the stone dust as being like flour and standing on his skin like white flour at the end of his shift. He states that the stone dust was always being blown in his face owing to the direction of the air current. All this time



CASE 4.



he had this persistent cough with black sputum and further volunteers the statement that even after he had been off work for 8 months he was still coughing black sputum.

He states he has felt better lately and does not have any sweats, although he still has the black sputum.

Family History: His father is still alive age 57 and does not suffer from any Bronchial trouble. His mother died of Sarcoma at the age of 33. No other member of the family suffers from chest trouble.

Habits: Smokes ten cigarettes a day but at the week-end he smokes as many as fifty. He drinks about a pint of beer a day.

Examination: Tall thin but intelligent patient. Cadaverous looking appearance in the face. Colour poor. No cyanosis. No engorgement of cervical veins. Chest moves easily on respiration. Definite clubbing of fingers. Somewhat dyspnoec on exertion. The supraclavicular fossae are fuller than normal and the chest is of the emphysematous type although not typically barrel shaped and there seems to be a good respiratory excursion on inspection. Slight winging of Scapula and some scoliosis.

Respiratory excursion is good 35" - 37½" - 2½" expansion.

The apex beat is in the 5th interspace 3½" from the mid. line.

At the level of the nipples the chest is 10" across and 8" deep. No myotatic irritability or myoedema.

Vocal fremitus normal.

Liver dullness increased one fingersbreadth below the right subcostal margin.

Chest is hyper resonant to percussion all over.

Breath sounds normal vesicular - occasional rhonchus at left base.

Sputum negative to tubercle bacillus on repeated examinations.

Blood pressure 110/60 mms Hg.

Weight 9st. 10½lbs and has gained 3lbs in the past 2 weeks.

This patient collapsed after I had finished taking his blood pressure - his pulse rose to 110 and he felt faint, his skin became cold and clammy with a profuse cold perspiration on brow and face. He recovered after a few minutes.

X-Ray Examination: He was found to have a Pulmonary Fibrosis due to dust reticulation. Further report - definite reticulation stage of Silicosis.

Skiagram: Showed a definite scoliosis of the spine with equally definite dust reticulation of both lung fields.

Electrocardiogram: Pointed T waves in Lead 2, otherwise normal.

This man then is a case of dust reticulation of the lung; he has worked under very adverse circumstances being exposed to both stone and coal dust. He developed a clinical Bronchial catarrh due to irritation of the Bronchi with carbon particles and this catarrhal process cleared the carbon particles from the Bronchi in the mucous exudate. Finally he had a serious accident and his vitality would be thereby lowered and when he resumed work the cough persisted and finally he developed an acute Bronchitis from which he only made a slow recovery.

Referred to Silicosis Board for examination. Confirmed to be suffering from dust reticulation and recommended for light work.

-ooOoo-

CASE 5. Early Dust Reticulation.

Edmund J.W. Age 47. Stoneman.

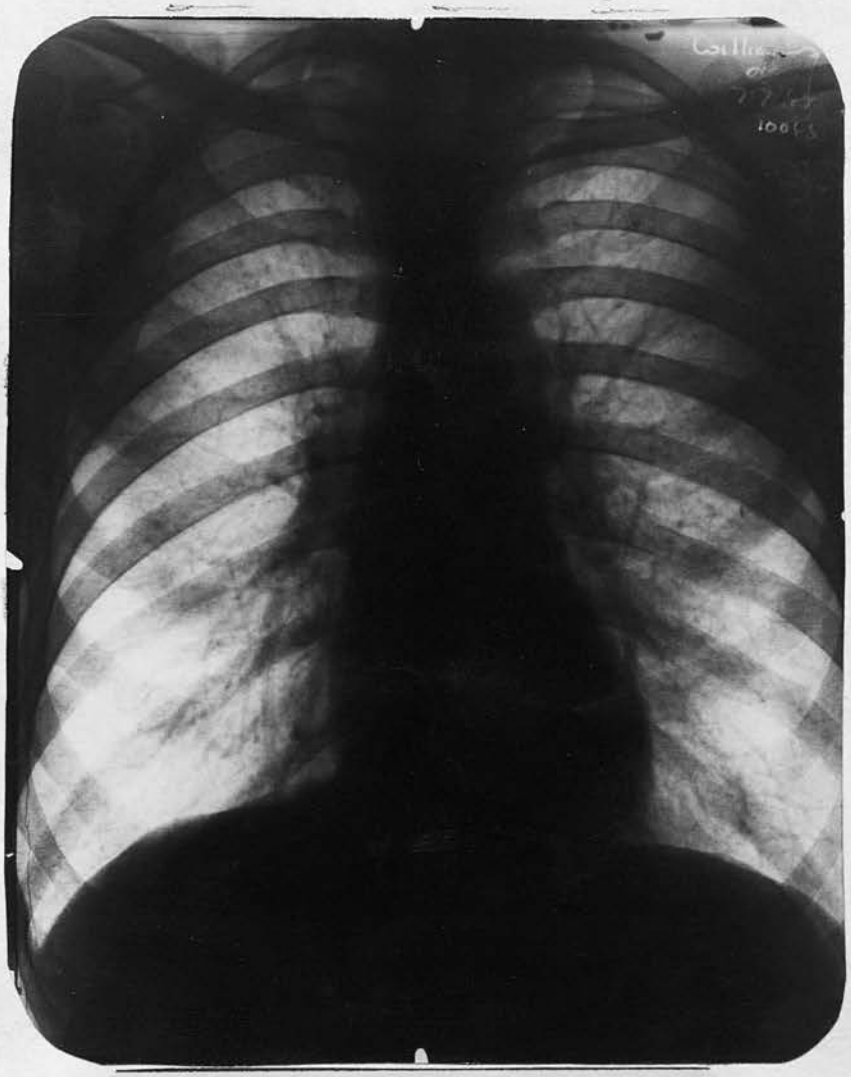
Complains of cough, breathlessness and pain in left side.

Duration - 12 years.

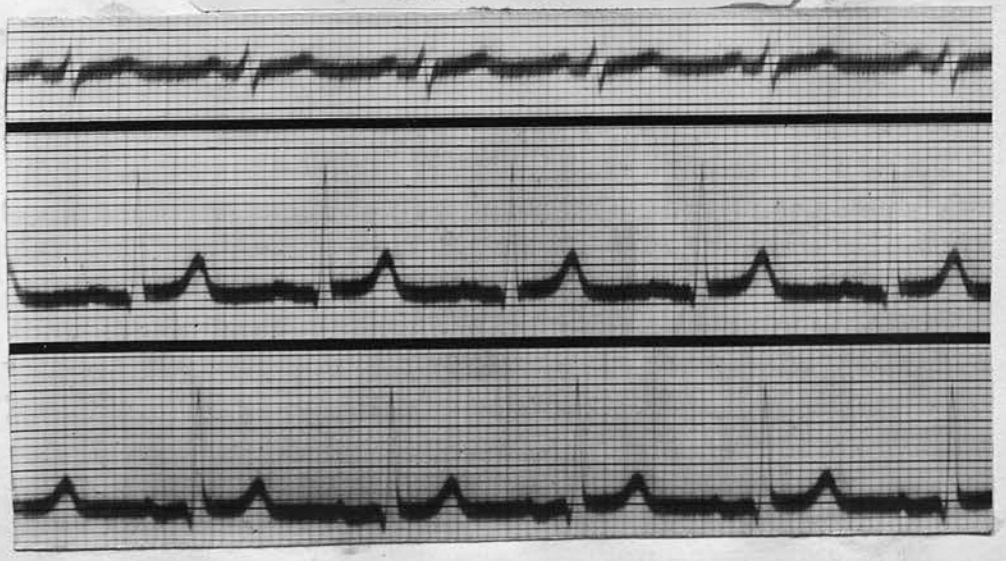
History: This man was quite well until 12 years ago when he developed a pain in the left side which lasted for some months. After this he had a cough and seemed to be breathless. These attacks of Bronchitis he states, have recurred at irregular intervals ever since. The cough, he states, comes with this left sided pain, and is of a 'racking' character. There is no sputum. When he is off work the cough ceases but when he returns to work the cough comes back.

He is a 'stone-worker' - that is he is employed taking stone down and making places for the tubs. He gets the stone down with a pick and does not do any shot-firing, although the stone dust is lying thickly all over the place and the passage of a'set' of tubs raises this fine floury dust so that the air is full of it. He has always been employed as a 'stoneman'. The stone dust he states, makes him cough although he does his best to avoid it. The dust is always there. He states that if he gets about 4 to 5 hours rest he feels well but if he lies longer than this the pain on the left side of the chest recurs and he has to get up (cf Case 8). When he stoops down the pain is considerably lessened. Probably muscular spasm. The frequent cough is his chief complaint.

Family History: His father died age 40 of Rheumatic Fever. Mother is still alive age 78.



CASE 5.



Habits: Smokes about 8 cigarettes a day and is a teetotaller. At one time he was a singer of some local repute, chiefly amateur operatic societies, but now he has not the breath, he states, and this seems to be getting worse.

Examination: Rather thin, introspective type of man with sallow complexion and frequent cough. He worries because he is breathless at work and tries to keep up with the others but is unable to do so. No definite cyanosis.

Chest wall thin but dilated. Some distension of the apices.

Expansion is $1\frac{1}{8}$" from $32\frac{1}{8}$" to 34".

Thoraco abdominal type of breathing. Respirations 28 per min

Resting pulse rate 96 per minute.

Apex beat in 5th interspace $3\frac{1}{8}$" from the middle line.

Superficial cardiac dullness normal, being about one fingersbreadth medial to the nipple line.

Vocal fremitus - no appreciable alteration.

Hyper resonant percussion note especially at the apices.

Vesicular breath sounds with, however, some prolongation of expiration. No accompaniments. Vocal resonance normal.

Heart sounds normal.

Blood pressure 140/100 mms. Hg.

No sputum for examination.

X-Ray Examination: Possible early reticulation of right and left lungs.

He further states that every time I have examined him I have set up a cough by tapping him on the left side just below the clavicle.

The chief features of this case are the frequent

irritable cough and dyspnoea and the fact that the cough ceases if he is off work only to recur when he goes back to work. Evidently the stone dust is an irritant to this man's bronchial mucosa and he is in process of developing a Pulmonary Fibrosis. He has a slight but definite emphysema as is evident by his prominent lung apices and this will tend to get worse as time goes on. He is now at the stage when if removed from his dusty environment the condition would probably improve or at least not progress to the next stage of cardiac impairment - Bronchiectasis or dust reticulation.

This man has always been a stone worker in the mine and is exposed to an atmosphere that is thick with stone dust so that pulmonary symptoms almost inevitably develope. The history here goes back a long time, some 12 years, but he is vague about the onset of his symptoms which seem to have been insidious, and also it has to be remembered that stone dusting before shot-firing has only been indulged in for some eight years or so.

He also complains of left sided pain which is easier when he stoops down and comes on after some four hours sleep. This may be due to a muscular spasm or it may be due to an early Bronchiectatic condition due to traction on a bronchus. It has been said that Silicosis is not uncommonly combined with Anthracosis and that then it leads to Bronchitis rather than to Tuberculosis. The reason suggested for this is that finely divided charcoal has the power of adsorbing Tuberculin.

This case is probably of such origin. His skiagram suggests an early dust reticulation.

-ooOoo-

CASE 6. Dust Reticulation.

Robert S.P. Age 47. Filler on Longwall-later Stonework.

Complains of heavy weight on chest and some breathlessness. Slight Cough.

Duration - 3 weeks.

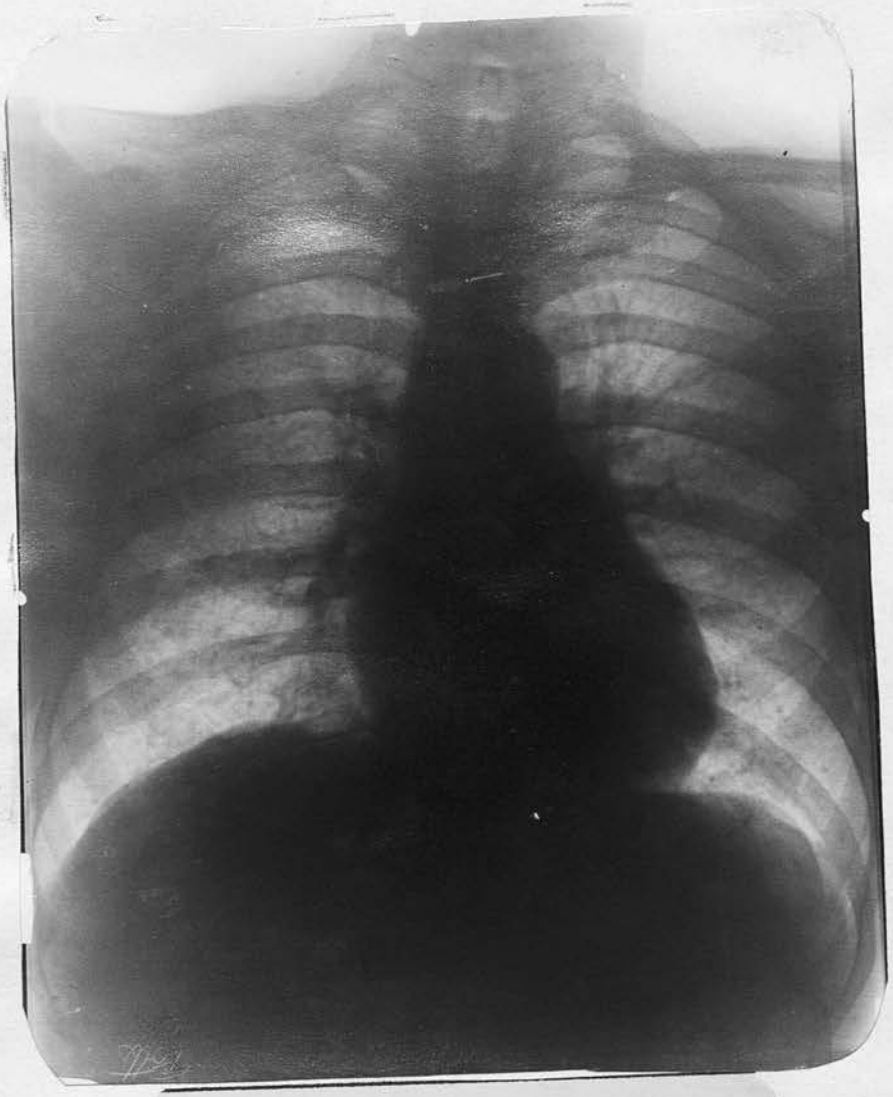
History: Patient has been well until some three weeks ago when he felt a sensation of weight over the front of the chest. This was worse when he got out of bed and better when he was in the fresh air. In addition he had a slight cough. He noticed that when he took a deep breath he had a pain in the front of the chest - substernal in position. Occasionly when hurrying he gets the pain also, but this is not consistent.

He worked as a longwall filler during which he was exposed to much 'powder reek' and stone dust. He is now on light work underground and is therefore still exposed to stone and coal dust.

Family History: Father died of Cerebral Haemorrhage at 65. His mother died of heart disease aged 63.

Habits: Smokes very little. Teetotaller.

Examination: Pale complexion. Seems to sweat easily when being examined. Chest moves poorly on respiration which is mainly of the abdominal type. Wide subcostal angle and widely spaced ribs. Deep antero posterior diameter of chest approximately $9\frac{1}{2}$" x $10\frac{1}{2}$". No oedema of feet and no clubbing of fingers. Some distension of supraclavicular fossae. Chest excursion on respiration very poor $34\frac{1}{2}$" to 35" - $\frac{1}{2}$" expansion.



CASE 6.

Apex beat in 6th interspace $3\frac{1}{8}$" from the middle line that is one fingersbreadth inside the nipple line.

Diminished vocal fremitus.

Hyper resonant percussion note all over chest especially at the apices.

Vesicular breath sounds with no accompaniments.

Vocal resonance diminished.

Blood pressure 135/80 mms Hg.

No sputum available for examination.

X-Ray Report: Pulmonary Fibrosis due to Pneumoconiosis.

This man is suffering from Pulmonary Fibrosis due to Pneumoconiosis. He is completely incapacitated from work.

Not accepted by Silicosis Board.

-ooOoo-

CASE 7. Dust Reticulation.

William J. Age 35. Stone Worker.

Complains of cough and breathlessness.

Duration - 3 years.

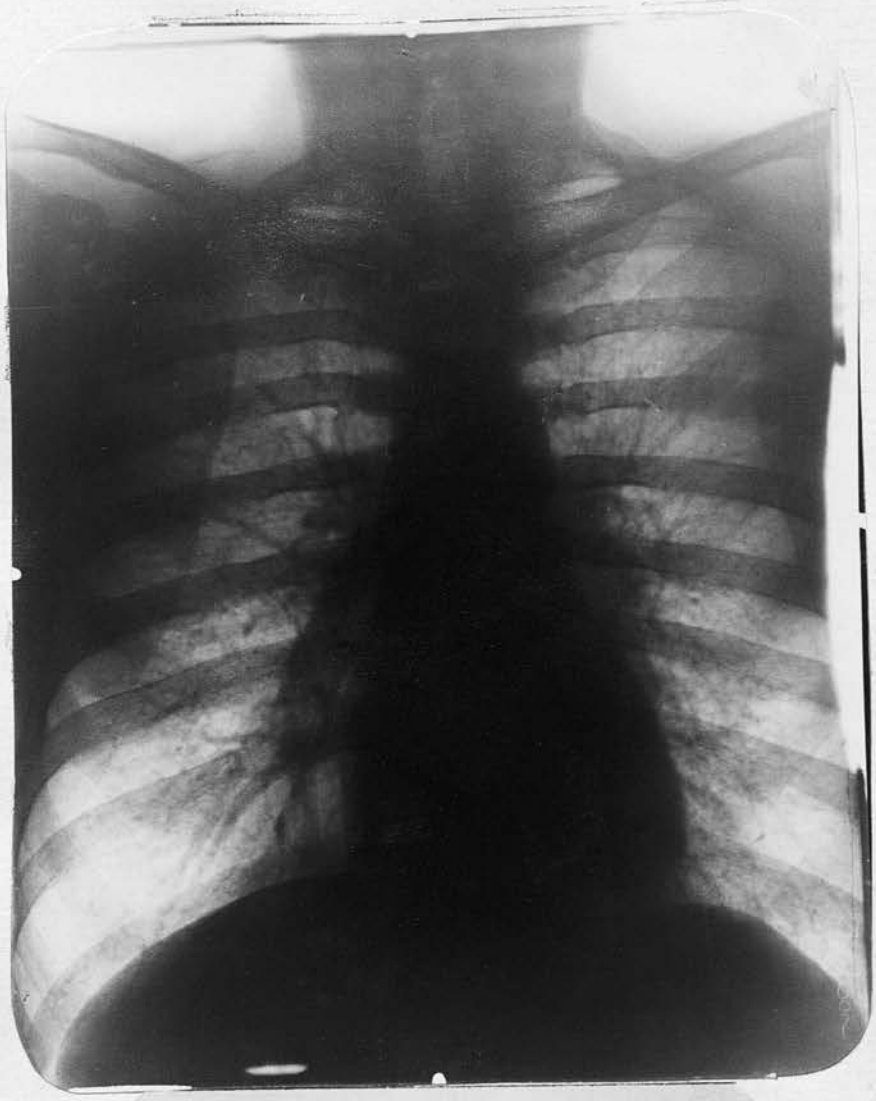
History: The above man was a healthy well built man of 35 years who was a good workman until he began to notice that he was suffering from frequent attacks of 'coughs and colds'. These occurred several times a year and now he finds that he cannot carry on his work like he used to do. He dislikes being idle but this frequent cough and breathlessness is compelling him to limit his exertion. Even when going to work he finds that if he hurries it brings this persistent cough on and he has to stop and rest. The sputum is black at first when he comes home from work - later, however, it clears and becomes white in colour. The elimination of dust, therefore, by his bronchial mucosa must be efficient.

He is a 'stoneman' and has been working as such for 11 years. He drills the stone, fires shots and in short is exposed to both coal and stone dust.

Family History: Nil of note.

Habits: Smokes about 10 cigarettes a day - never more. He drinks very little.

Examination: Cough very evident and obviously very irritable. He is a well built man but has a pale complexion. No cyanosis and no clubbing of fingers. Chest moves freely on respiration



CASE 7.

- thoraco abdominal type of respiration. The supraclavicular fossae are well filled indicating a certain degree of emphysema.

He has a definite scoliosis of the spine.

Myotatic irritability on lightly tapping the pectoral muscles.

Apex beat in 5th interspace 4" from the middle line, that is one fingersbreadth within the nipple line. No gross diminution of superficial cardiac dullness.

Chest excursion 35½" - 37½" = 2" expansion.

Resting pulse rate 108.

Resting respirations 24 per minute.

Resonant percussion note all over the chest - somewhat hyper-resonant at apices.

Vocal fremitus normal.

Vesicular breath sounds all over the chest. No accompaniments.

Vocal resonance normal.

Heart sounds closed.

Blood pressure 135/95 mms Hg.

Sputum Examination: Negative.

X-Ray Report: Some reticulation of the lung field.

Not accepted by the Silicosis Board.

Two days after the above report this man developed a pain in the left chest with friction rub, elevation of temperature and pulse. He exhibited all the physical signs of a pleurisy.

The exposure to coal and stone dust that he has experienced for 11 years has produced a degree of reticulation but not sufficient to warrant compensation.

-ooOOoo-

CASE 8. Dust Reticulation.

John D. Age 48. Coal Cutter.

Complains of cough and breathlessness also pain in chest.

Duration: 4 - 5 months.

History: This man has always been healthy until about 4 - 5 months ago when he began to suffer from a cough which was worst in the mornings as soon as he got up. He would cough up a lot of mucous and then would be fairly free from coughing for the rest of the day. During the night he sometimes had to get up for the cough but not very often. He also found that he was very breathless and was unable to walk very far without stopping. If he does any work he becomes very breathless. At work he cannot carry any of his equipment very far without stopping for a rest. He has worked as a coal cutter for 20 years and a year ago he was (for 6 months) cutting through stone alone. He was operating the coal cutter for 17½ hours one day and a number of men were affected by the stone dust that day and had to go home. The coal cutter he operated is worked by electricity.

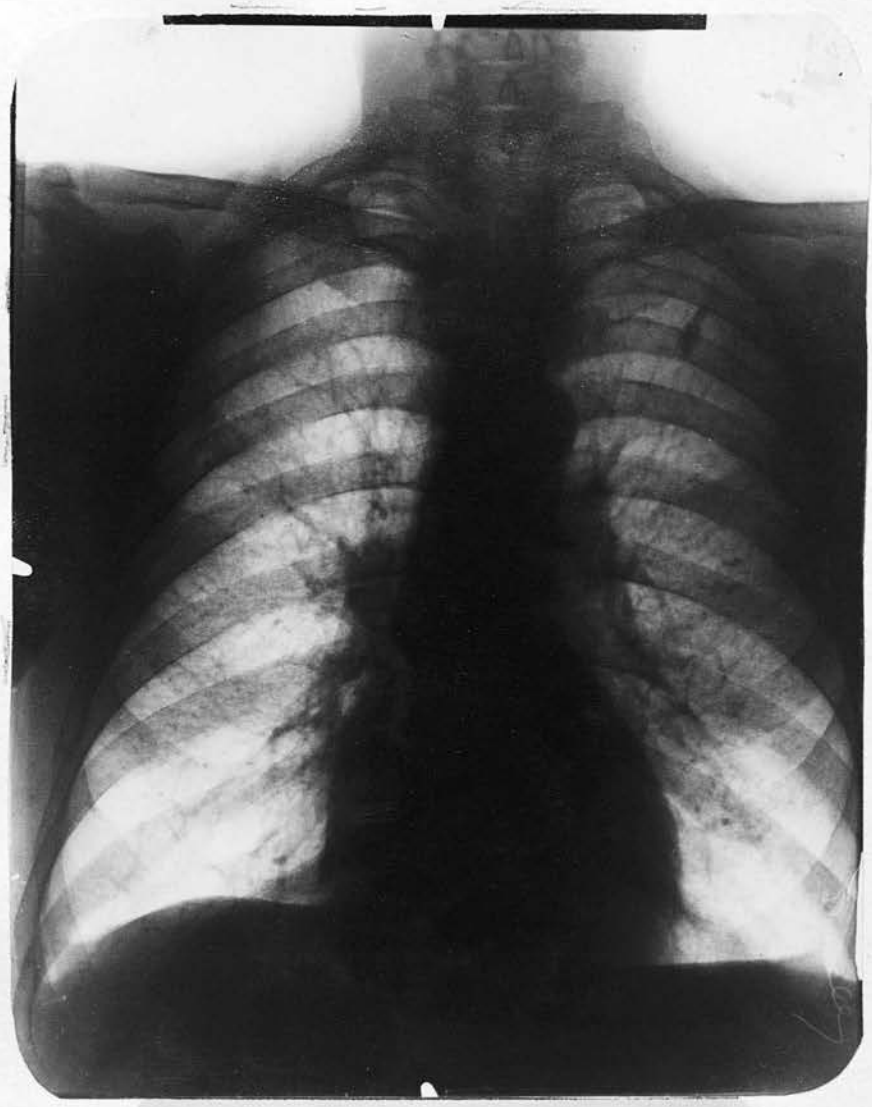
He also at this time began to complain of a tightness in the chest.

During the past 6 months he has lost 19 lbs in weight and finds that he sweats very easily.

Family History: Nil of importance.

Habits: Smokes a pipe. He hardly drinks atall.

Examination: Resting pulse rate 88, per minute.



CASE 8.

Respirations 18 per minute.

He is a well nourished, intelligent type of man. Weight 9st. 12 lbs. Chest moves easily on respiration, which is of the thoraco-abdominal type. No clubbing of fingers.

Myotatic irritability of chest muscles present.

Apex beat in 5th interspace 3" from the middle line.

No obvious emphysema.

Vocal fremitus normal.

Resonant percussion note all over chest - possibly hyper-resonant at apices.

Vesicular breath sounds no accompaniments.

Heart sounds normal. Vocal resonance normal.

Blood pressure 135/90 mms.Hg.

Sputum Examination: Negative.

X-Ray Examination: Pulmonary fibrosis with non-specific reticulation.

The history of this case is very suggestive of Pneumoconiosis but on physical examination there is, as usual, very little to find. Only on radiographic examination is a condition of non-specific reticulation shown - a coal miners lung and it illustrates the fact that men can contract pneumoconiosis and die from it without any very gross physical signs being apparent. The case illustrates the necessity of a good radiographic examination in all these cases of latent pneumoconiosis.

-ooOoo-

CASE 9. Dust Reticulation.

John O.K. Age 52. Boiler Cleaner.

Complains of cough and breathlessness.

Duration: 3 - 4 years.

History: This man has worked in a colliery Boiler Plant for 20 years. For the last 7 - 8 years he has been a boiler cleaner. This work consists of chipping the deposit off the inside of the boilers and also of cleaning out the flues. This is very dusty work although some water is usually thrown down to keep down the dust. The flues contain not soot he states, but a substance like burnt soot. About 3 - 4 years ago he developed a cough and found that he was very breathless on exertion. This cough is worst during the night and especially in the early morning when he gets up. The sputum is thick and a dirty yellow colour. He finds that the cough is much worse if he lies on his left side.

Family History: Nil of note. Both parents died at advanced ages.

Habits: Teetotaller and non-smoker.

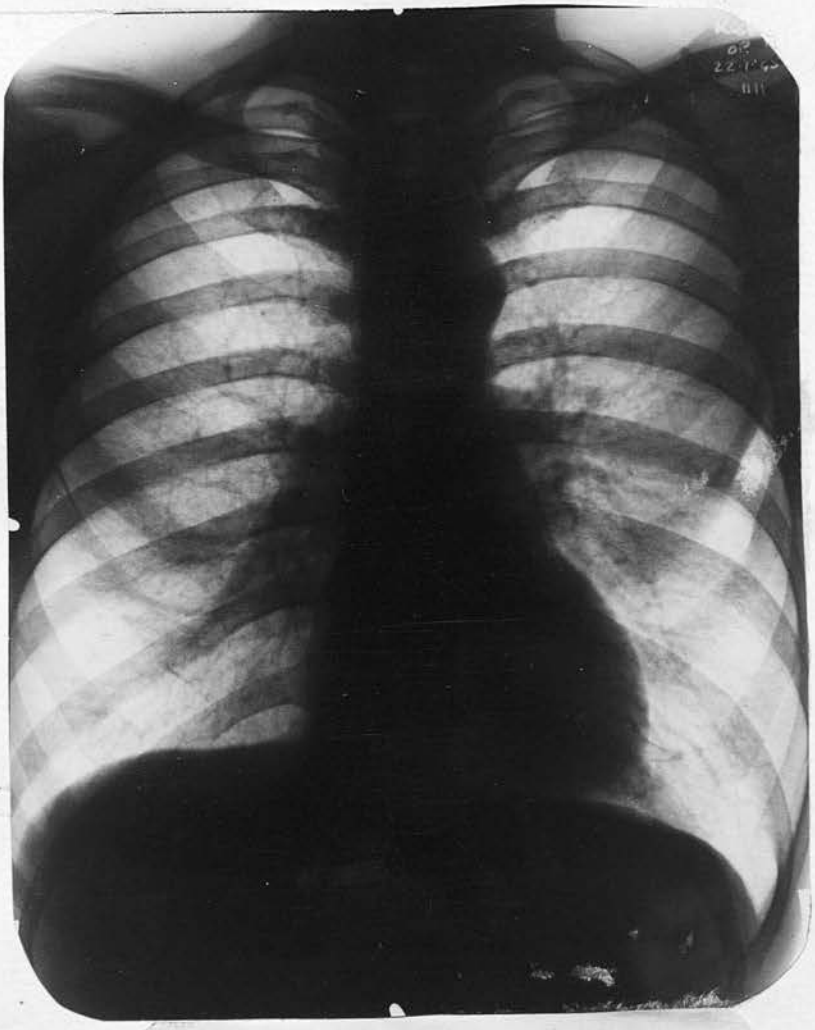
Examination: Small quiet man obviously dyspnoec as when taking his clothes off for examination.

Resting Pulse 100 per minute. Resting respirations 20 per min

Emphysematous type of chest but not markedly so - more obvious at apices.

Myotatic irritability of chest muscles. Some slight scoliosis. No finger clubbing.

Chest measurements $35\frac{1}{2}$" - 37" ie $1\frac{1}{2}$" expansion.



CASE 9.

Apex beat difficult to determine but apparently in 5th interspace. Area of superficial cardiac dullness diminished to about 1½" from the middle line.

Chest resonant to percussion but hyper-resonant at apices and to a lesser extent at the base.

Vocal fremitus within normal limits.

Vesicular breathing with however prolongation of expiration and rhonchi during expiration. No moist accompaniments.

Heart sounds normal.

Blood pressure 130/95 mms. Hg.

Sputum Examination: Negative to tubercle bacilli.

X-Ray Examination: Radiologist reports a non-specific reticulation but does not consider a Silicosis/Board would pass for disability.

This man has worked in and about coal mines all his life. For the last 20 years, however, he has been employed as a boiler cleaner. This exposes him to a considerable amount of dust which has affected his general health so that he now suffers from non- specific reticulation of the lung field. He is never free from cough and is very breathless on exertion.

-ooOoo-

CASE 10. Pulmonary Fibrosis.

William L. Age 41. Stoneman.

Complains of recurrent attacks of cough and breathlessness.

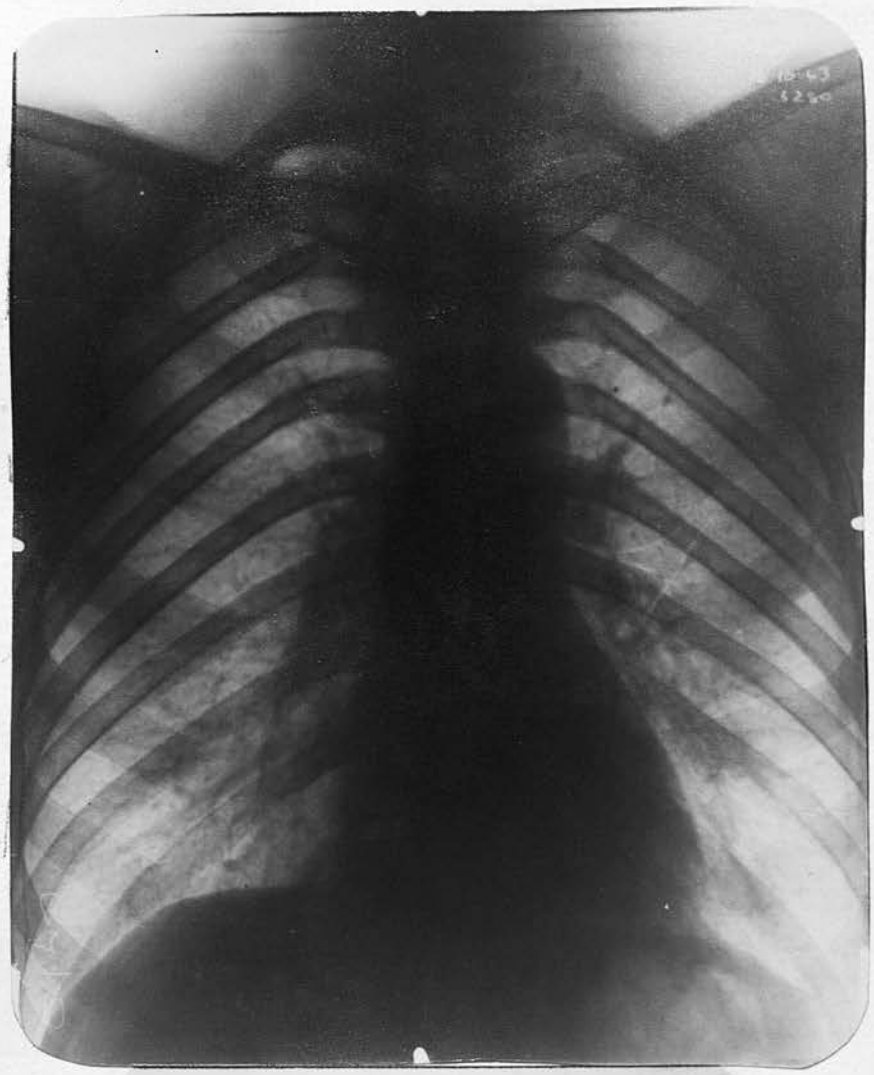
Duration: 2 years.

History: Some 2 years ago this man complained of cough and pain in the chest. He had some pyrexia with a friction rub over the right base and was treated as a Pleurisy. He made a poor recovery the cough persisting and there was also some dyspnoea apparent. His brother had just died from a Bronchogenic Carcinoma and he seemed to worry about it a good deal and rather imagined he might have it himself. When he did finally recover he resumed light work and was unable to follow his own work as a 'stoneman'. This work (as a stoneman) is dusty and involves drilling stone and blasting it and is in fact an occupation in which one is exposed to the inhalation of a considerable amount of stone dust. This man then, since his initial attack, has only been able to do light work and he has now fallen off work again, although his condition is by no means serious. He always has a slight cough but this does not inconvenience him. There is no sputum.

Family History: Mother alive age 78. Father dropped dead in the mine - of apoplexy age 55. One brother died of a Bronchogenic Carcinoma.

Habits: Smokes about 15 cigarettes a day. Does not drink.

Examination: Big, fairly well developed man. Chest moves freely on respiration with a respiratory rate of 18 per minute



CASE 10.

while in bed. Thoraco-abdominal type of respiration.

Resting pulse rate 80 per minute.

No clubbing of fingers. No cyanosis.

Wide subcostal angle and widely spaced ribs. Sallow complexion. The apices of the lungs are well filled up but one could not call them distended.

Chest expansion 2¼" - from 35" - 37¼".

Apex beat in 5th interspace 3" from the middle line. The chest is deep and the ribs are widely spaced so that one gets the impression of an Emphysema without having very much to go upon except that there is gross diminution of the superficial cardiac dullness. This is unsuspected until percussion is carried out when there is at least three fingers breadth diminution in the superficial cardiac dullness. Vocal fremitus diminished.

Hyper resonant note on percussion all over chest except on the right side in mid. zone where there is a duller note on percussion.

Vesicular breath sounds with some rhonchi on expiration.

Vocal resonance slightly diminished.

Sputum Examination: Negative to both Tuberculosis and Carcinoma.

X-Ray Examination: Negative to Tuberculosis but on examination there is some pulmonary fibrosis and.possibly early reticulation in both mid zones.

This man then had a Pleurisy followed by cough and dyspnoea which never really cleared up altogether. He had been working as a stoneman for years and had been inhaling

stone dust during that time. The result would be a slowly increasing pulmonary fibrosis which culminated in an attack of Pleurisy two years ago. The Pleurisy would be associated with the fibrosis and it is a fact that at autopsy on miners I have noticed on numerous occasions that the pleura is unusually thickened and adherent and that the lungs were infiltrated with fibrous tissue - the so called peribronchial infiltration This patient would be left with an area of thickened pleura on the right side which gives a flatter note on percussion.

This patient with thickened pleura and lung fibrosis will probably go on to develope a Bronchiectasis in later life due to the contraction of the fibrous tissue.

This is another case where if the patient is kept out of a dusty atmosphere he will maintain good general health although the risk of a Bronchiectasis is always present.

-ooOoo-

CASE 11. Pulmonary Fibrosis - Fibroid Phthisis.

Thomas E. Age 57. Coal Hewer.

Complains of cough and breathlessness.

Duration: 32 years.

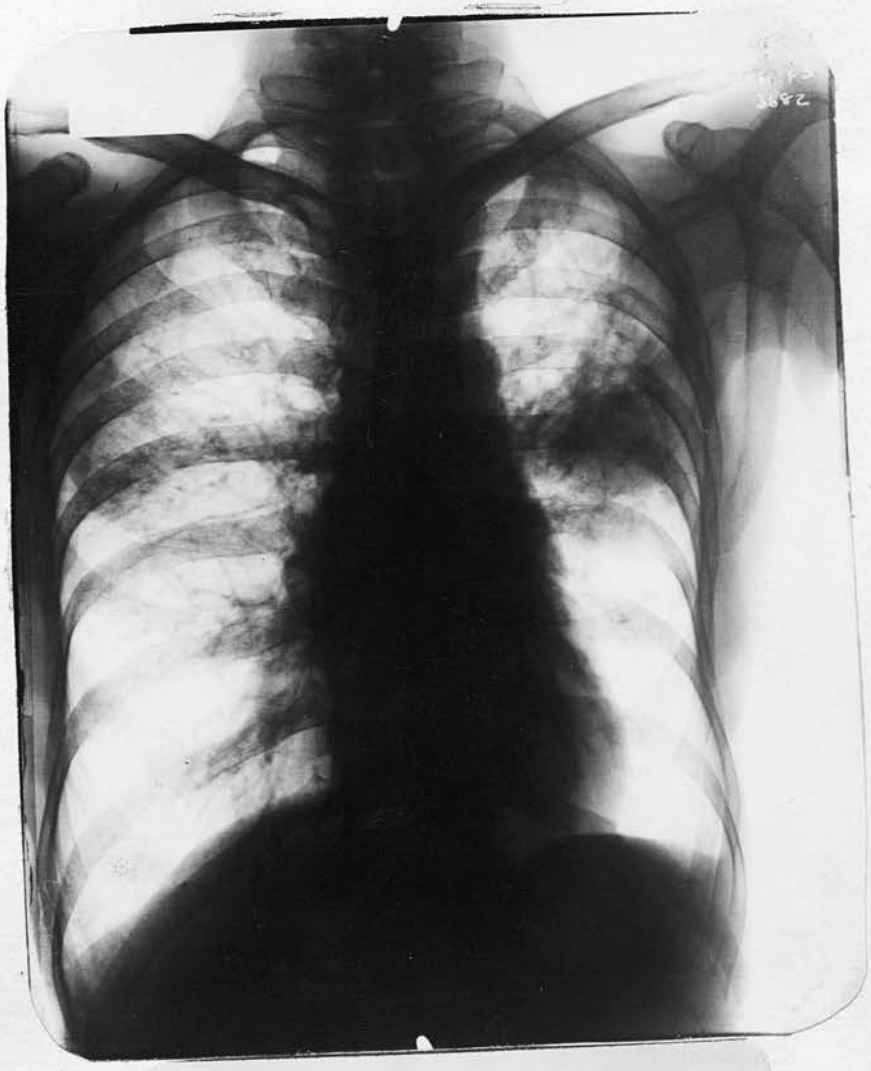
History: This man had an accident in 1912 being crushed between a 'coal tub' and a prop supporting the roof. After this accident he asserts he was subject to attacks of Bronchitis - these being of only slight intensity he did not loose any work on account of them. They occurred during the winter. From this I would conclude that he suffered from the familiar 'winter cough' of coal miners. In 1916 he came to work on the surface as his chest was worse in a dusty atmosphere. Since then he has been subject to frequent attacks of Bronchitis until finally he had to give up work altogether. He is now a chronic invalid and has not worked for nearly a year, being very breathless and suffering from a chronic cough with expectoration.

Family History: Father alive aged 85. Mother died aged 79.

Examination: This man is extremely thin. His facial bones stand out and all his bony prominences are easily discernible. He is very breathless. Even lying in bed his respirations are 36 per minute. No cyanosis. His skin is sallow and has lost its elasticity. His mucous membranes are of good colour but his respirations are chiefly abdominal.

The chest moves poorly on respiration having only 1" of expansion - 29½" to 30½".

Apex beat in 5th interspace.



CASE 11.

No bulging of intercostal muscles and no particular bulging of supraclavicular fossae.

Apex beat one fingersbreadth medial to nipple line in 5th interspace. Blood pressure 80/55 mms.Hg.

Vocal fremitus increased on right side. Normal on left side. On percussion he is stony dull on the left side - painful to the percussing finger. Resonant - almost hyper-resonant on the right side. Râles heard with the naked ear on the right side especially at the base.

On auscultation he has bronchial breathing at the left side with aegophony.

On the right side breath sounds are vesicular with some prolongation of expiration and crepitations at the right base. From the result of this examination one concludes that this patient has a collapsed lung on the left side.

X-Ray Examination: 1936 - Extensive pulmonary fibrosis with some dilatation of the bronchi and possibly early bronchiectasis.
1943 - in this skiagram there is some probable fibroid Tuberculosis of left upper lobe.
1944 - marked involvement of left upper lobe. Area of fibrosis left mid zone.

Sputum Examination: His sputum examinations have throughout all been negative. His sputum is nummular now and profuse.

This man died of cardiac failure due to Chronic Fibroid Phthisis.

-ooOoo-

CASE 12. Chronic Bronchitis with Emphysema.

Thomas H. Age 45. Coal Cutter.

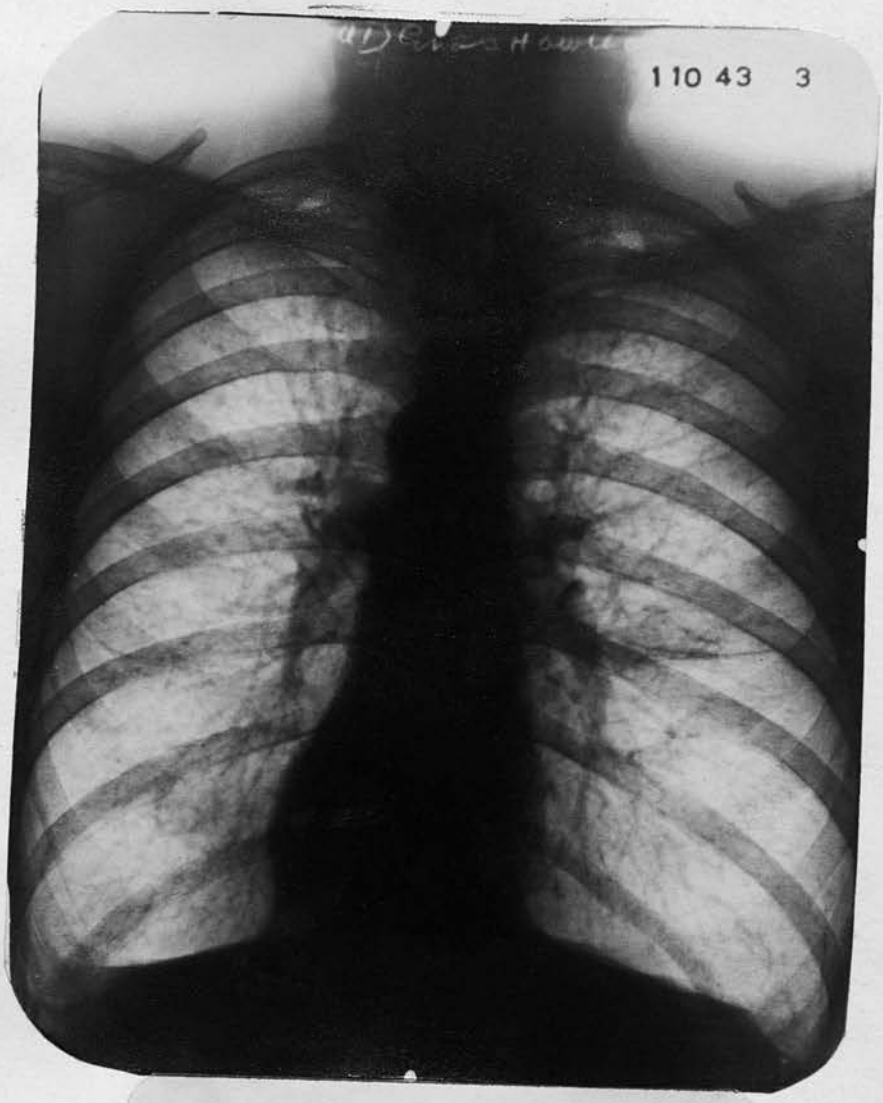
Complains of shortness of breath and cough.

Duration: 3 years.

History: This man has been very dyspnoec for 3 years. Prior to this he had had some five attacks of acute Bronchitis and one haemoptysis. The attack of haemoptysis was in 1936 and at that time it was noted that there were some râles at the left apex although investigation proved negative and he subsequently recovered full functional efficiency. In 1941 he noticed that he was not so strong as before and that he was getting very short of breath. He could not carry on his work as before and he felt "that something was coming on". His work involved lifting the belts of the coal cutters and he found he was incapable of this. Finally he became so breathless that he had to give it up and finish with the mines altogether.

He has now been off work over a year and during that time his condition has deteriorated. He has worked underground for 30 years and during most of that time he was a coal cutter. The seam is only about 2 feet high and therefore he works in a very confined space in which shot firing was going on so that the air was thick with mixed dust and nitrous fumes. In addition he was at one time a driller in coal or stone. His sputum is blackish.

Family History: Nothing of note.



CASE 12.

Examination: Small well built man. His face congested with small superficial venules in the nose and cheeks. Slight tinge of cyanosis. He is very breathless on exertion and this is the characteristic feature of this case. He has a frequent short cough. His dyspnoea has increased to such an extent that he cannot even go out for a walk in fine weather but sits outside when it is sunny.

There is some clubbing of the fingers. There is a 'fullness' of his supraclavicular fossae and he had a wide subcostal angle with widely spaced ribs.

Respirations 24 per minute. Resting pulse rate 76 per min.

Chest expansion is $2\frac{1}{2}$" from 35" - $37\frac{1}{2}$".

The apex beat is not apparent and the superficial cardiac dullness cannot be ascertained due to the Emphysematous nature of the lungs.

Vocal fremitus is diminished.

The chest is hyper-resonant to percussion all over especially at the apices and bases.

Vesicular breath sounds without accompaniments.

Heart sounds normal. Blood pressure 105/80 mms.Hg.

X-Ray Examination: Radiologist reports some peribronchial thickening and basal emphysema but no evidence of Tuberculosis.

Sputum Examination: Negative.

Diagnosis - Chromic Bronchitis with Emphysema.

This man has been a coal cutter and driller for 30 years. He has reached the age of 45 and is a chronic invalid due to dyspnoea being unable even to walk. He has suffered from several attacks of Pleurisy and Acute Bronchitis. The writer

has noticed thah coal miners suffer repeatedly from attacks of Pleurisy and also from atypical Pneumonia. This I have found in 2 or 3 cases which have come to autopsy, was due to a thickened Pleura in the deep layers of which was much coal dust which was probably interfering with the lymphatic circulation and so causing a dry pleura and hence friction. From his skiagram one would conclude that as there is much peribronchial fibrosis he is probably an early Pneumoconiosis.

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CASE 13. Pulmonary Fibrosis with Emphysema.

Henry H. Age 32. Filler.

Complains of cough and shortness of breath.

Duration: 2 years.

History: This man states that he has had occasional Bronchial colds for the past 9 years but lost no work until 2 years ago when he became worse and has not worked since. He was in bed for a week with cough and sputum. There was, however, very little sputum relative to the cough. After this his sputum was examined and his chest was X-Rayed for tubercle both with negative results. He now has a persistent cough with expectoration of sputum and some breathlessness on exertion. The sputum is thick yellow and rather sticky. He states that the cough is worse during the night and early morning. He has, however, occasional bouts of coughing and dyspnoea which come on during the night. He wakes up coughing and remains so for an hour ot two after which he is very breathless.

Family History: Nil of note.

Examination: Thin delicate looking man. Weight 8st. 9lbs. height 5' 11½". Obviously underweight and somewhat dyspnoec.

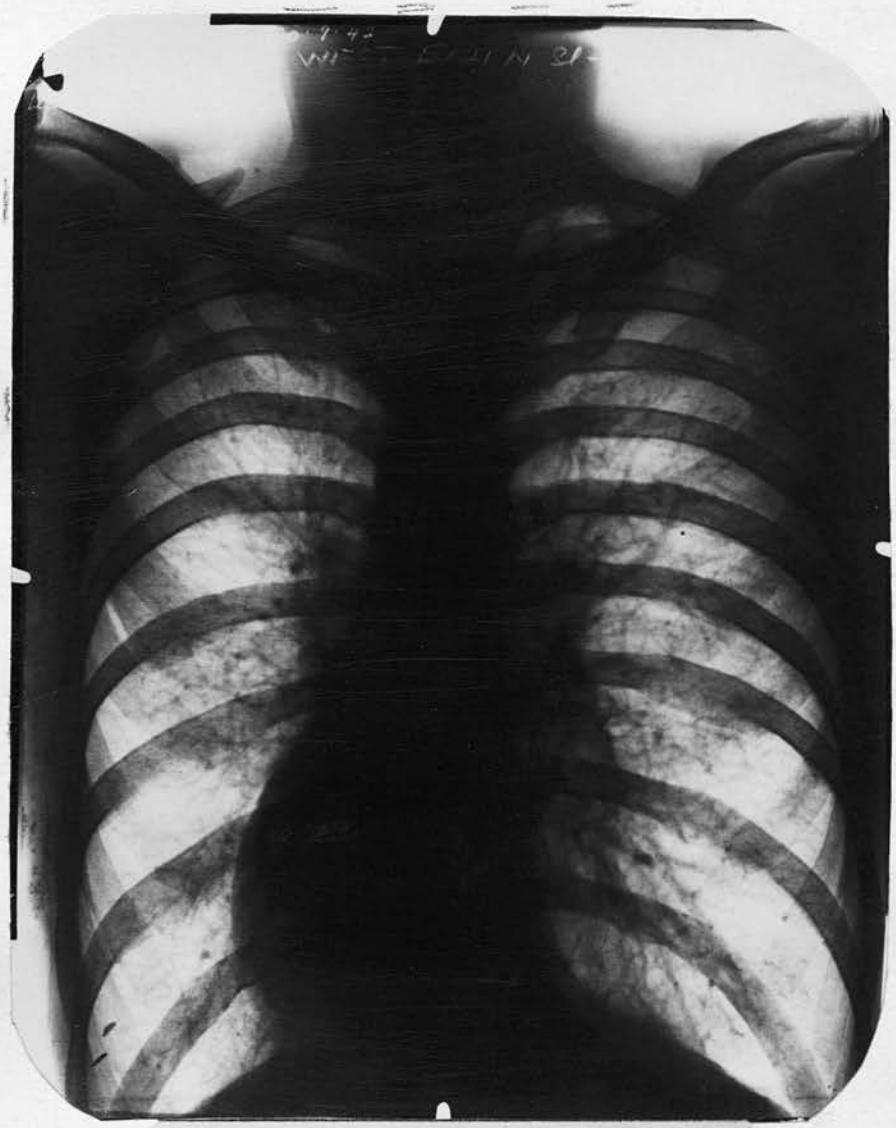
Respirations 24 per minute. Resting pulse rate 80 per min.

Thin poorly formed chest with myotatic irritability.

Fingers slightly clubbed - drumstick appearance.

Chest expansion 1½" from 32½" - 34".

Apex beat behind 6th rib 3" from the middle line. Some diminution of superficial cardiac dullness 2¾" from the middle



CASE 13.

line about three fingersbreadth medial to the nipple line.

Percussion note resonant all over the lung. On the right side the breath sounds are harsh vesicular with some crepitations during expiration. On the left side vesicular breathing with no accompaniments.

Rhonchi at both bases.

Blood pressure 115/80 mms.Hg.

X-Ray Examination: 1942 - No evidence of any Tuberculosis. Radiologically he has generalised peribronchial thickening with Emphysema.

He was labelled Chronic Bronchitis and Emphysema.

This man was employed as a 'filler' when he finished work. A 'filler' is a man who works on his knees filling the hewed coal into tubs. He works therefore with coal cutters, usually in low cramped places.

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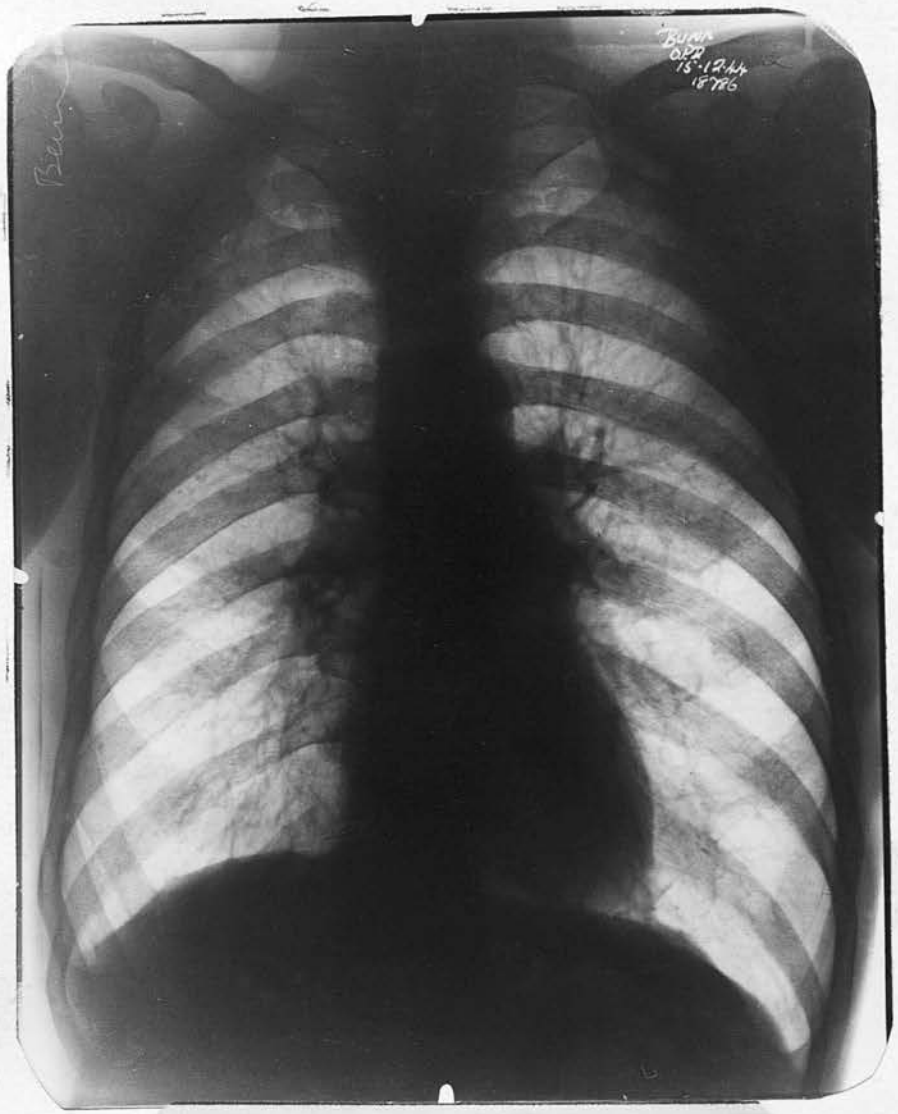
CASE 14. Miners Asthma - Emphysema.

J.G. B. Age 36. (Deputy Overman 6 years.
(Coal Cutter 5 -do-
(Putter 9 -do-

Complains of breathlessness and cough.

Duration: 3 years.

History: This man has worked for over 20 years in the mine and always enjoyed good health until three years ago when he felt himself getting very breathless so much so that on two occasions he collapsed at work with acute dyspnoec attacks and had to be carried out of the mine. Previous to this he had had no trouble with his chest although as a deputy overman he had been doing a lot of shot firing. Finally he became so breathless that he had to give up mining altogether and worked in a factory. Although he is breathless all the time, and especially if he has to climb a hill or undertake any exertion, he suffers from an acute exacerbation about twice a year when he is confined to bed. During these attacks his respiratory rate increases to about 28 per minute and there are numerous rhonchi all over his chest and his accessory muscles of respiration are brought into play, yet it is not a true spasmodic asthma. The attack lasts for about two days but he is not as distressed as a true asthmatic - and then gradually subsides. He states he has been much better since he gave up coal mining. To get some relief from his dyspnoea he has to use an Adrenaline spray (1 in 100) which he carries about with him in a special case and has to use three times daily. He has to take it to work with him or whereever he goes.



CASE 14.

Family History: His father (also a miner) died of chronic bronchitis at the age of 63.

Examination: Intelligent patient. Small and rather underweight 8st 12 lbs. Definitely dyspnoec. Clubbing of fingers.

Scoliosis of vertebral column.

Definitely very emphysematous chest - distension of supraclavicular fossae and widely separated ribs. Chest appeared to be in a state of full inspiration all the time. Typical appearance of an emphysematous chest.

Fibrillary twitching of the pectoral muscles and myotatic irritability.

Resting pulse rate 100 per minute.

Resting respirations 20 per minute - very full inspirations.

It was impossible to determine the apex beat or the area of superficial cardiac dullness on account of the emphysematous lung intervening between the heart and chest wall.

Very poor respiratory excursion ie 1" from 33" - 34".

Hyper resonant almost tympanitic percussion note all over the chest.

Vocal fremitus diminished.

Vesicular breath sounds all over chest with high pitched rhonchi on expiration. Vocal resonance diminished.

Heart sounds muffled but closed.

Blood pressure 125/95 mms.H.g.

Sputum Examination: Negative.

X-Ray Examination: Emphysema with some evidence of chronic Bronchitis.

This case could be described as a true Miners Asthma. He has chronic pulmonary disease which on examination is suggestive of Chronic Bronchitis and Emphysema with super added Asthmatic condition which is, however, not a true spasmodic Asthma. The acute asthmatic attack lasts two days and although he is confined to bed, yet he is not so distressed as a spasmodic Asthma. He obtains relief with an Adrenalin Spray.

-ooOoo-

CASE 15. Miners Asthma.

William H.B. Age 33 years. Coal Cutter.

Complains of breathlessness and cough.

Duration: 6 years.

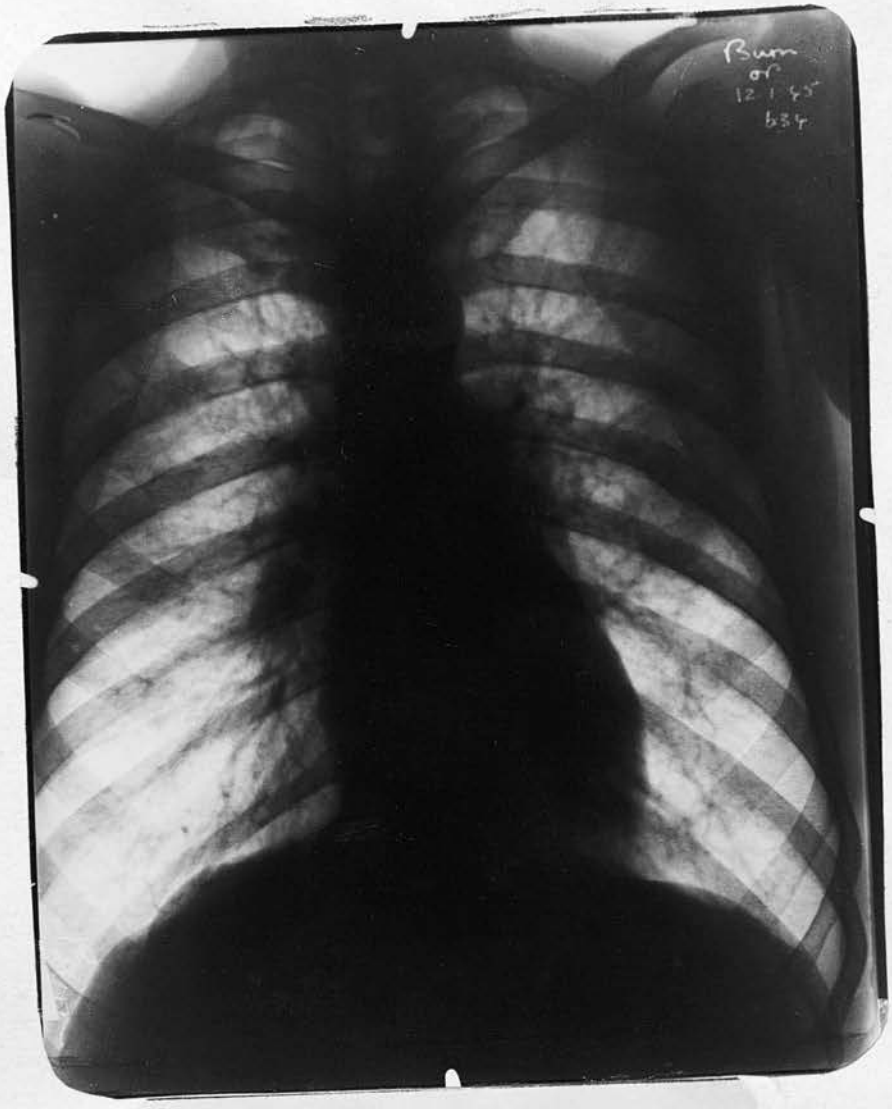
History: Patient was a coal miner and worked underground in the mine until he was about 27 ie 6 years ago, when he began to complain of frequent head colds which affected his chest causing a cough. These attacks increased in frequency and he began tocomplain of "tightness in his chest". These attacks used to come on suddenly during the night - he had to sit up in bed and then open the window. This became more and more frequent until he had to leave off work for weeks at a time. After the breathing paroxysm subsided he would cough up a little 'phlegm' and feel better. This man was skin tested and found to be sensitive to mixed inhalants. He was therefore desensitized with mixed inhalants (Bencards) intravenously. This treatment seemed to improve him but only to a limited extent.

Examination: Average height, lightly built, serious and rather introspective disposition. Pale complexion but mucous membranes of a healthy colour.

His chest moves freely on respiration which is of the Thoraco-abdominal type. No barrel chest, no bulging of intercostal spaces and no prominence of the supraclavicular fossae.

Chest measurements at nipple line $33\frac{1}{2}$"-$35\frac{1}{2}$"= 2"expansion.

Apex beat in 5th interspace about one fingersbreadth medial to nipple. No diminution of superficial cardiac dullness.



CASE 15.

Vocal fremitus normal.

Percussion note resonant all over.

Breath sounds harsh vesicular with prolongation of expiration and numerous high pitched rhonchi all over the chest.

No crepitations on auscultation.

Heart sounds closed in all areas.

Blood pressure 118/80 mms.Hg.

X-Ray Examination: Probably dynamic dilatation of Pulmonary Arteries and pulmonary fibrosis.

Sputum Examination: No Tubercle bacilli found.

This man represents a case of Miners Asthma as it used to be called. A condition of Chronic Bronchitis with super-added attacks of spasmodic Asthma. He has been employed as a coal cutter for some 13 years until he had to give up underground work altogether and come to bank on account of his chest condition. He is never without an Adrenalin Spray and is sensitive to mixed inhalants on skin testing.

His skiagram shows basal emphysema and peribronchial infiltration - a commencing pulmonary fibrosis.

-ooOoo-

CASE 16. Pulmonary Fibrosis.

John W. Age 39. Filler.

Complains of cough and breathlessness.

Duration: 1 year.

History: This man started work in a neighbouring coal mine at the age of 14. He worked on the 'screens' and later as 'putter', coal hewer and 'filler'. He came to this coal mine five years ago and worked uninterruptedly for four years as a filler. One day, however, he went to work as usual but had to stop on account of a pain in his abdomen. The pain subsided and he returned to work but had to stop again and finally he developed Lobar Pneumonia. He remained in bed with pneumonia for six weeks and was then allowed up. The cough, however, persisted and he found he had to be propped up on account of his breathlessness at night time. He had much sputum which was examined and found to be negative to tuberculosis. He has been off work ever since with cough and breathlessness, he cannot walk uphill or do any work on this account.

Family History: Nil of note.

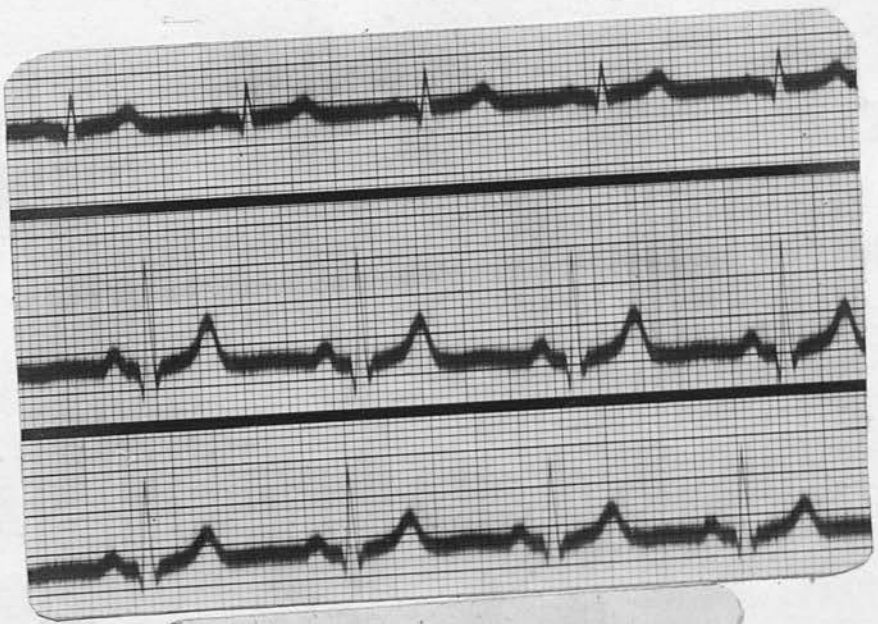
Examination: Thin worried looking man. Tall but spare. Obviously dyspnoec - in fact this is the most striking feature about him.

Respirations 32 per minute. Resting pulse 108 per minute.

Ribs horizontal. No obvious emphysema. Thoraco-abdominal breathing with emphasis on the abdominal.

Myotatic irritability of chest muscles.

Apex beat in 5th space $2\frac{1}{2}$" from the mid. line.



CASE 16.

No diminution of superficial cardiac dullness.

Resonant percussion note all over chest.

Vesicular breath sounds with moist accompaniments at both bases and diminished air entry.

Heart sounds normal.

Blood pressure 125/85 mms.Hg.

Sputum Examination: Negative.

X-Ray Examination: Peribronchial infiltration with Pulmonary Fibrosis. Electrocardiogram: High peak in T wave in Lead II. He also shows marked Q waves in all leads. His E.C.G. shows a marked similarity to that of Case 2. who is suffering from Koniophthisis.

This man is a case of Pulmonary Fibrosis who has had an attack of Acute Lobar Pneumonia which has increased the fibrosis thus making him what might be called a Pulmonary cripple.

-ooOoo-

CASE 17. Chronic Bronchitis and Emphysema.

Ambrose M. Age 57. Coal Hewer.

Complains of breathlessness and cough.

Duration: 5 years.

History: This patient has worked as a coal hewer in the mine for 40 years. He followed his occupation as a coal hewer until 5 years ago when he had to give up work on account of increasing breathlessness and cough.

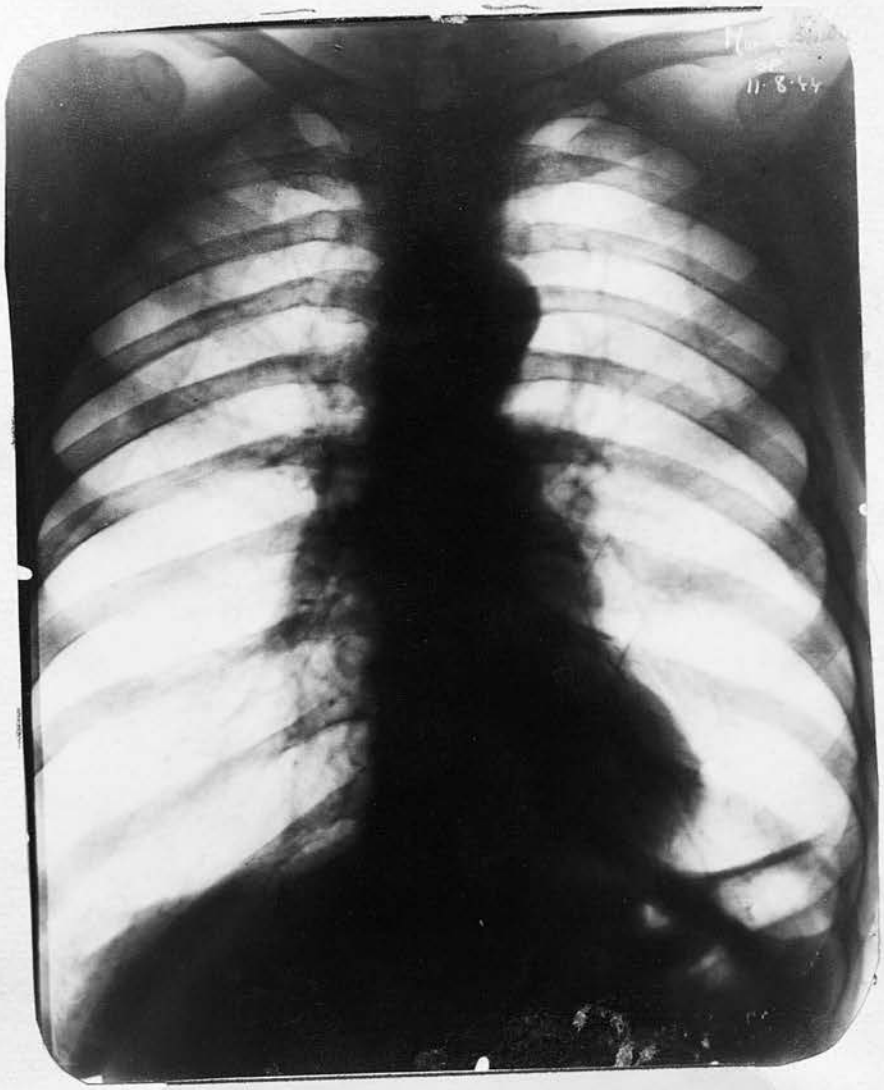
Examination: Congested venules across face involving malar regions and nose. He is very thin and has lost more weight recently. Cyanotic appearance. The most striking feature about him is his dyspnoea. He is breathing at the rate of 24 respirations per minute and the breathing is frequently interrupted by a single dry sounding cough. At intervals he has a paroxysm of coughing which results in clear mucous. No offensive smell. The accessory muscles of respiration are all in action but the breathing is almost entirely abdominal in type and the chest has a very poor excursion on respiration $34\frac{1}{2}$" - 35" - thus only $\frac{1}{2}$" expansion. This patient has a very distressed appearance generally.

There is some bulging of the apices and of the intercostal muscles.

Vocal fremitus diminished.

Apex beat in 5th space about two fingers within the nipple line.

On percussion the chest seems hyper-resonant in all areas except over the praecordia where there is a diminution of the



CASE 17.

superficial cardiac dullness. Myotatic irritability present.

On auscultation the breath sounds were of a harsh vesicular type all over the chest with an occasional rhonchus.

Vocal resonance diminished.

Heart sounds closed.

Sputum Examination: No tubercle bacilli found.

X-Ray Examination: No evidence of Bronchiectasis nor definite pneumoconiosis. Pulmonary fibrosis and probable pleurisy left lower zone.

-ooOoo-

CASE 18. Pulmonary Fibrosis with Basal Emphysema.

James D. Age 58. Overman.

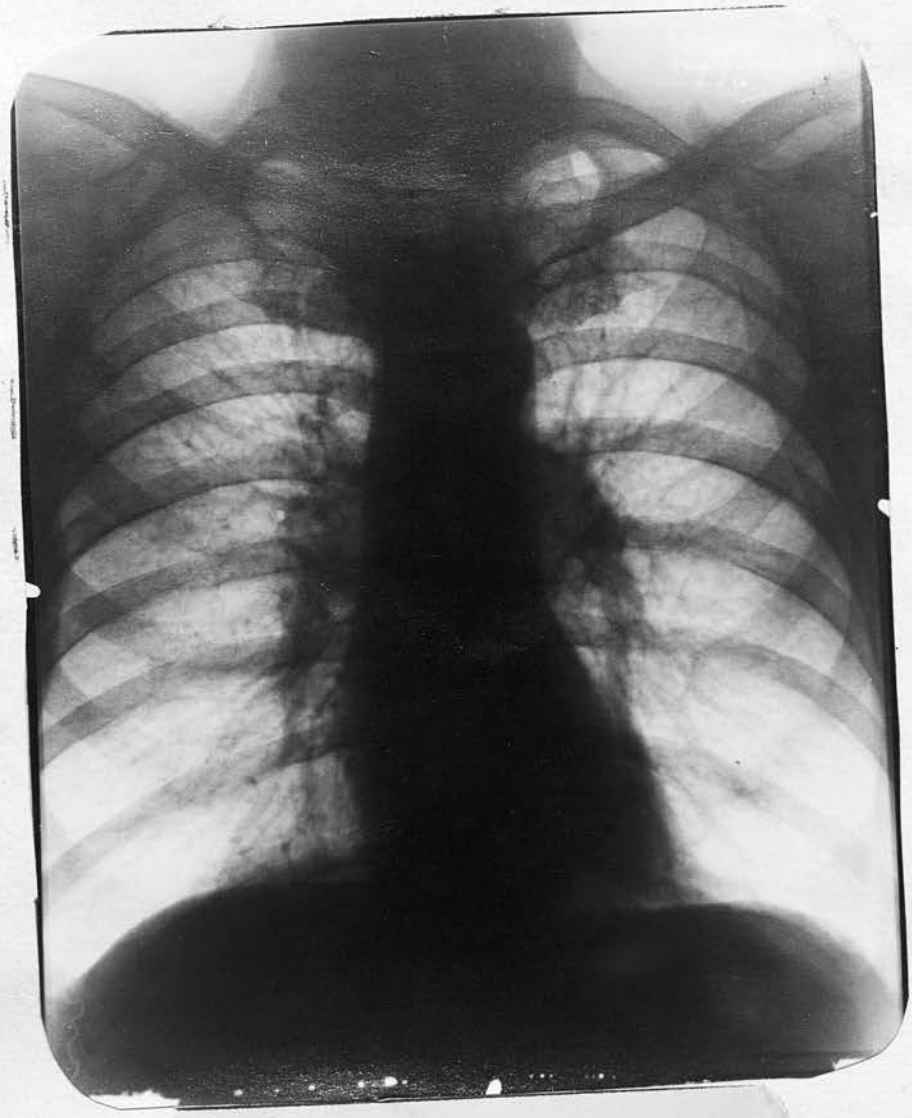
Complains of cough and breathlessness. Expectoration of Sputum.

Duration: 18 months.

History: This man has always been healthy and robust until about 18 months ago when he developed a cough and sputum of a blackish yellow colour. This persisted some months until he suddenly had an attack of acute dyspnoea with numerous rhonchi all over the chest, and tachycardia. He almost collapsed in the street at the start of this attack and was very cyanosed throughout. With rest and cardiac tonics he gradually recovered although it was some five months before he recovered sufficiently to resume work. The tachycardia was the last sign to clear up. After he resumed work he was not unduly breathless although he could not carry out sustained exertion without some distress and he suffered from attacks of nocturnal dyspnoea. He used to wake up at night gasping and would cough incessantly for one to one and a half hours until he coughed up some sputum after which he got relief. While at work, however, he was not unduly distressed He still has periodic attacks of nocturnal dyspnoea and has to use a spray of Adrenalin and Pituitary which gives him some relief. The cough remains but it is only of infrequent occurrence. He is a Fore-overman that is an underground official, which, whilst not necessitating severe manual labour/underground, does entail a good deal of travelling underground.

Family History: Nil of note.

Examination: Big man with remarkably fresh complexion for a



CASE 18.

miner. Some slight cyanotic tinge to be seen in the lips. Thick chest wall with no bulging of the apices although he has a wide subcostal angle and wide intercostal spaces. Chest moves easily on respiration which is of the thoraco-abdominal type.

Resting pulse rate 88 per minute. Respirations 24 per min.

Skin is rather moist and sweaty - in fact he sweats easily.

Chest expansion $1\frac{1}{2}$" from $37\frac{1}{2}$" to 39".

Apex beat in 5th interspace 4" from the middle line.

The superficial cardiac dullness seems to be diminished being about three fingers medial to the nipple line and difficult to elicit.

Chest wall resonant to percussion - somewhat higher note than normal. Vesicular breath sounds all over with a few rhonchi at the bases. Vocal fremitus normal.

Blood pressure 115/90 mms.Hg. Heart sounds normal.

Blood Examination: - on account of his florid complexion.

Haemoglobin 115%
Red blood cells 5·370000 per cu.mm.
White blood cells 6000 per cu.mm.
Colour Index 1·0

) Insufficient evidence to warrant a diagnosis of Polycythaemia. Evidently a compensating polycythaemia to increase the oxygen carrying capacity of the blood

The differential white count showed no abnormality.

X-Ray Examination: Right and left lungs showed pulmonary fibrosis with basal emphysema. No evidence of silicosis, Tuberculosis or pulmonary neoplasm. Radiologist states that the film 'shows a coarse mottling' and the ribs are horizontal indicating an Emphysema.

This man then has a Pulmonary Fibrosis with some basal emphysema. The emphysema is not marked - there is no bulging

of the apices and no characteristic barrel chest. He has, however, a wide costal margin and widely spaced ribs and the percussion note, while not hyper resonant, is of a higher note than normal.

This man having been so exposed has gradually developed a pulmonary fibrosis which being of insidious onset has not caused any symptoms until the day he collapsed when, after some extra exertion, he has put too much strain on his heart which has dilated and caused symptoms of cardiac embarrassment with the usual result of dyspnoea, cyanosis etc.

-ooOoo-

CASE 19. Emphysema and Pulmonary Fibrosis.

Edward A. Age 48. Coal Cutter.

Complains of cough and breathlessness.

Duration: 4 years.

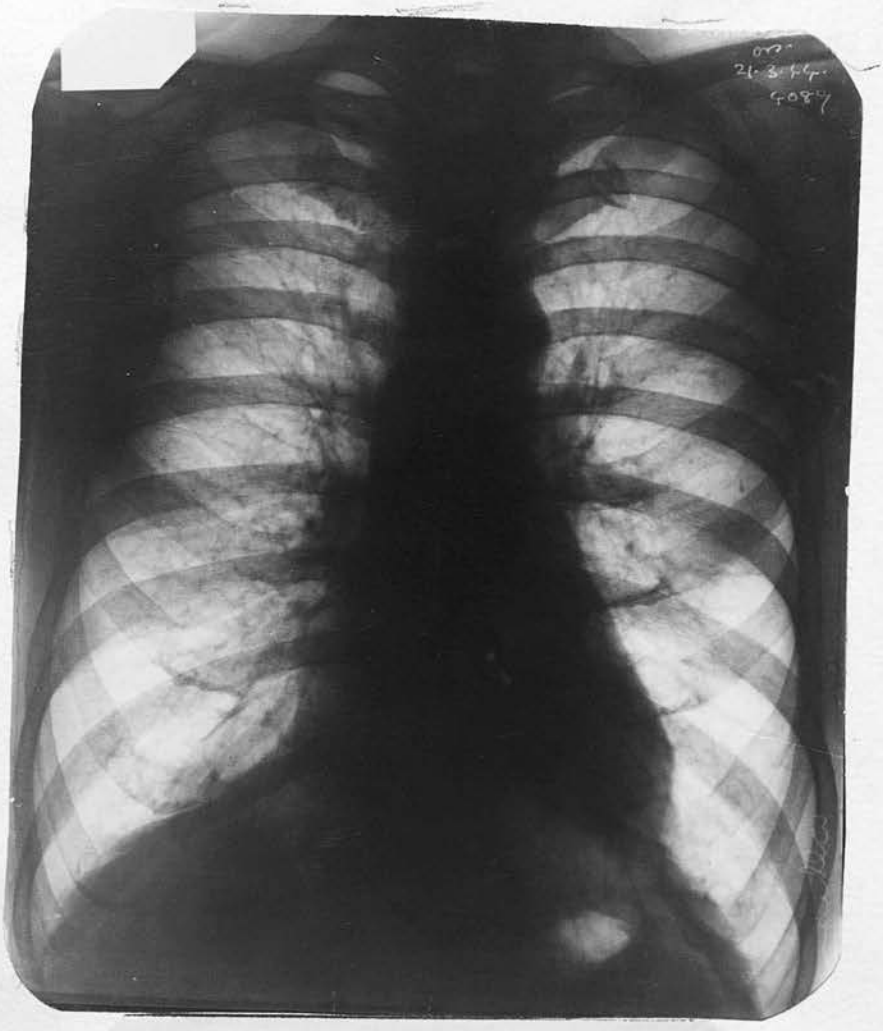
History: This man started work down the mine at the age of 14. and has worked there ever since, although he is now employed on light work at the bottom of the pit shaft. Most of his time below ground has been on coal cutting especially the drilling operation which is a preliminary to coal cutting. In this connection he also used to throw stone dust about which is distributed around any place where there is any possibility of an explosion. He then developed attacks of Bronchitis and later when I saw him he had frequent attacks of Influenzal Bronchitis with high temperature. These acute exacerbations havebeen more frequent in the last four years. During this time he has also become more dyspnoec and his chest has felt 'tight'.

Family History: Nothing of significance except that one brother is 'bronchitic'.

Examination: This man has the usual rather muddy complexion of a miner though his mucous membranes are of a good colour. He has a persistent frequent cough. He is very barrel chested, the chest seemingly being held in full inspiration. The apices are very prominent. There are superficial venules to be seen extending over the course of the diaphragm across the body.

He has a wide costal angle. His arms are thin and the general nutrition of the body is poor.

Thoraco abdominal type of breathing with however very



CASE 19.

little movement of the chest - there being more an up and down movement of the chest and abdomen.

Chest measurement $36\frac{1}{2}$" - $37\frac{1}{2}$" = 1" expansion.

Apex beat is in the 6th interspace $4\frac{1}{2}$" from the mid line.

Vocal fremitus diminished.

The percussion note is hyper resonant all over. It is not possible to ascertain the outline of the heart by percussion as the percussion note is hyper resonant over the heart.

Apices are especially hyper resonant.

Breath sounds are faint and vesicular all over. There are a few rhonchi at the bases.

Vocal resonance diminished.

Blood pressure 120/90 mms.Hg.

X-Ray Examination: No evidence of Tuberculous infection but Emphysema and Pulmonary Fibrosis evident.

Sputum Examination: Repeatedly negative to Tubercle.

This patient then is suffering from Chronic Bronchitis and Emphysema. He has in addition an asthmatic element. This is probably caused by some spasm of the Bronchioles due to irritation by particles of dust. He presents a typical picture of a developing Emphysema - the chest appearance is one that might be seen in a text book illustrating Emphysema. There are superficial venules along the line of attachment of the diaphragm - this is frequently seen in cases of Emphysema and is most likely due to the obstruction in the Pulmonary circulation causing venous congestion throughout the body.

This man has been employed a s a coal miner for 35 years and was originally a coal cutter. In this occupation he would drill holes in the coal face being thereby exposed to much coal

dust and when the machine was working he would be breathing air thick with coal or stone dust. He now has a Pulmonary Fibrosis as his X-Ray shows. He has Chronic Bronchitis and Emphysema. The next phase in the process is Bronchiectasis.

It is this 'syndrome' of Chronic Bronchitis, Emphysema, Bronchiectasis or Pulmonary Tuberculosis which I wish to emphasise in these cases. This man represents the Chronic Bronchitis and Emphysema stage. He has already Pulmonary Fibrosis.

-ooOoo-

CASE 20. Chronic Bronchitis and Emphysema.

Thomas C. Age 62. Stoneman.

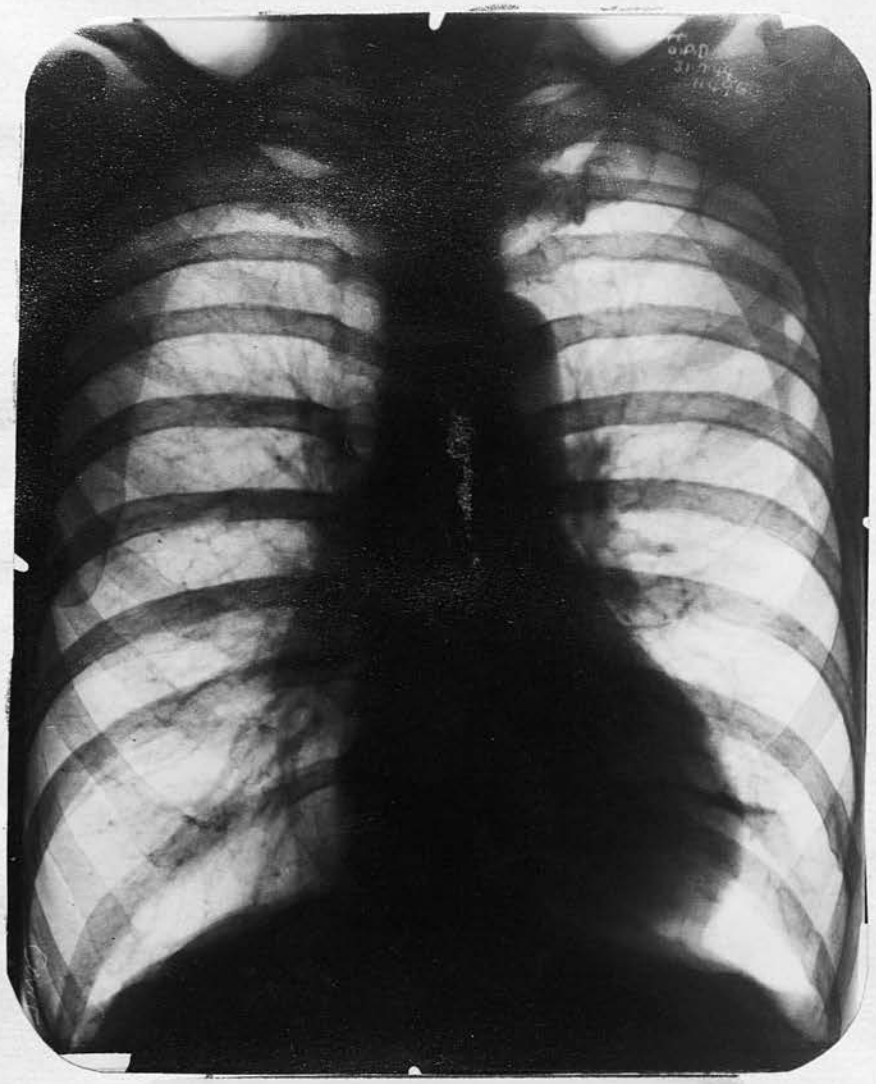
Complains of cough and breathlessness.

Duration: 30 years.

History: This man started work as a farm labourer and worked as such until the age of 23 when he commenced work in a coal mine. In 1913 at the age of 33, he commenced to have 'Bronchial trouble' - a troublesome cough worse in the winter. He was working at the time in a dusty seam and as a 'stoneman'. This work consists of clearing the roof of the tunnels and making them higher so that the tubs can come through. To do this they drill holes in the stone and insert a charge of explosive which is then detonated. After this they clear away the resultant stone and proceed on like that. This patient was the driller. The stone is not limestone but blue stone - a shale which splits into sections like slate. Eventually the cough and dyspnoea became worse and he had to give up work underground and started work in a brickyard where the same stone was ground up and made into bricks. He was for a time even employed in the grinding mill and finally he was employed cleaning out the fires and general labouring.

Family History: Nil of importance.

Examination: This man is tall but of poor physique. Muscles thin and wasted, some cyanosis. Obviously very dyspnoec and has a frequent loose cough with expectoration of muco-pus. No offensive odour. The cough is much worse in the mornings coming on about five minutes after he gets up and after about half and hour's coughing he feels better. During this time he



CASE 20.

expectorates a large quantity of yellow muco-purulent sputum.

The breathing is thoraco abdominal and is very frequent - 28 respirations per minute. More an up and down movement than true expansion. Definite clubbing of fingers.

Respiratory excursion 34¼" - 35¼" = 1" expansion.

Myotatic irritability on tapping the pectoral muscles.

Some bulging of the supraclavicular fossae.

Emphysematous type of chest. Ribs running almost horizontally. Vocal fremitus diminished.

Hyper resonant note to percussion all over the chest with slight impairment at the right apex.

Apex beat in 6th interspace one finger medial to nipple line

Superficial cardiac dullness ½" to right of nipple line.

Some dilatation of left ventricle beyond the nipple line.

On auscultation harsh vesicular breath sounds with prolongation of expiration and numerous crepitations both inspiratory and expiratory on the right side but only expiratory on the left. Vocal resonance normal.

Blood pressure 200/140 mms.Hg.

X-Ray Examination: Chronic Bronchitis and Emphysema. No evidence of Pneumoconiosis.

Sputum Examination: Negative to Tuberculosis.

This man is then suffering from Chronic Bronchitis and Emphysema. He is dyspnoec(resting respirations 28 per minute) due to deficient oxygenation. He has worked under the worst possible conditions for most of his working life - drilling stone for explosive charges and then clearing up the stone amid the reek and dust after the explosion. Thus he has been exposed to both constant stone dust and coal dust and it is

little wonder that now at the age of 63 he is extremely breathless and is troubled with an intractible cough. The toxaemia from the Bronchitis is causing the emaciation and wasting of his muscles and the rather muddy colour of the skin.. Frequently he has slight rises of temperature due to this pus in the bronchioles, and always he is dyspnoec.

On looking at his skiagram, the wide spaced ribs which are almost horizontal are very apparent. It suggests Emphysema. In addition there is an in-crease in the hilar shadow and infiltration is apparent along the peribronchial lymphatics especially in the right lung. The condition is suggestive of a slight degree of fibrosis. The heart, too, is hypertrophied to a certain extent both right and left ventricles. The left sided hypertrophy is borne out by the fact that he has a definite increase in blood pressure 200/140 mms.Hg. and by the manner of his death as he died from a cerebral Haemorrhage.

-ooOoo-

CASE 21. Chronic Bronchitis and Emphysema.

James C. Age 52. Coal Hewer.

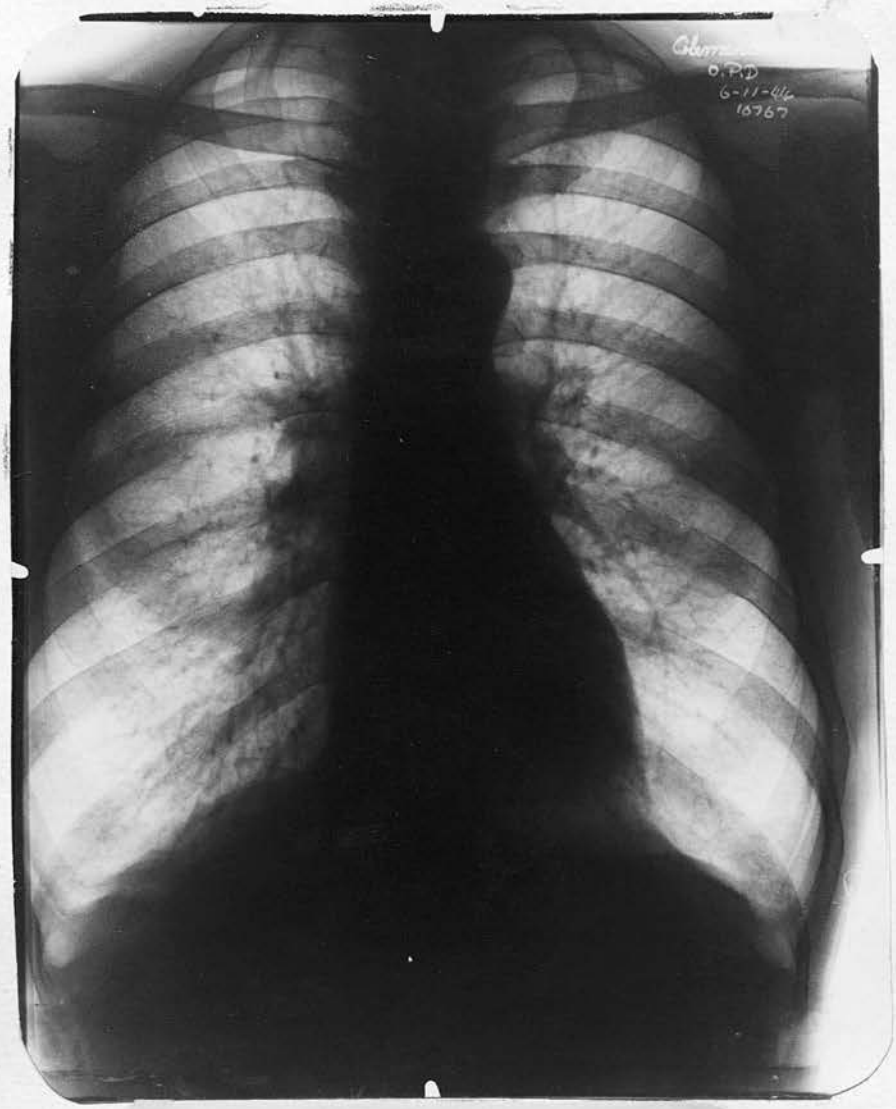
Complains of cough, breathlessness and tightness of the chest.

Duration: 7 years.

History: This patient was quite healthy until 7 years ago when he had an attack of Acute Bronchitis. Since then every winter he has been off work with chronic bronchitis and breathlessness. He finds that exposure to dust and cold makes him worse. Lately,however, he has found that even in the spring and summer he is very dyspnoec and the cough is not now absent for any length of time. The sputum he coughs up is thick and yellow and he always has a cough in the mornings. This is the time that he feels worst- his chest feels 'tight' and he feels 'gaspy' and he has to go outside to get fresh air. He also finds that his cough is made worse by any exertion such as pushing or lifting or hurrying. The attacks of Bronchitis are worst in the winter and occur repeatedly throughout this season. He is not confined to bed with these attacks but feels breathless and poorly generally and finds that he sweats easily even in bed at night. He has not worked for over nine months. He was always a coal hewer until seven years ago, when the dust became too much for him and he had to get light work.

Family History: Nil of note.

Examination: Thin worried apprehensive type of man. Dyspnoec on exertion as when removing his clothes. Frequent cough with expectoration of muco purulent sputum. Chest moves fairly freely on respiration,although the respiratory excursion is poor.



CASE 21.

Thoraco abdominal type of respiration. Poorly formed chest with some myotatic irritability. No supraclavicular fossae - as they are filled up. Wide costal angle

Chest measures 32"- 33½" - expansion 1½".

Apex beat in the 5th interspace 3½" from the mid line.

Superficial cardiac dullness 3" from the mid line about one fingersbreadth inside the nipple line.

Respirations 20 per minute. Resting pulse rate 76.

Heart sounds closed.

Percussion note definitely hyper resonant all over the chest especially in the apices.

Breath sounds are vesicular but on deep expiration there are numerous rhonchi all over the chest and a bout of coughing is initiated.

Blood pressure 140/90 mms.Hg.

X-Ray Examination: Chronic Bronchitis and Emphysema. No evidence of dust reticulation.

Sputum Examination: No tubercle bacilli found.

This man is suffering from Chronic Bronchitis and early but definite Emphysema. He has been a coal hewer until 7 years ago and in such employment would be exposed to much coal dust and a certain amount of stone dust. The constant irritation with the dust in time causes a degree of pulmonary fibrosis and starts the cough - this latter being a protective mechanism to get rid of the inhaled dust. Hence the 'black spit' of coal miners is really a protective mechanism and indicates that the ciliated epithelium is functioning normally. But this man has muco purulent sputum which indicates infection and as a consequence his ciliated epithelium will be destroyed and

and replaced by columnar epithelium. This in turn will favour the stagnation of muco purulent secretion and of any further dust so that Bronchiectasis will then develope.

-ooOoo-

CASE 22. Pulmonary Fibrosis and Emphysema.

William T.J. Age 58. Coal Cutter.

Compains of cough and breathlessness.

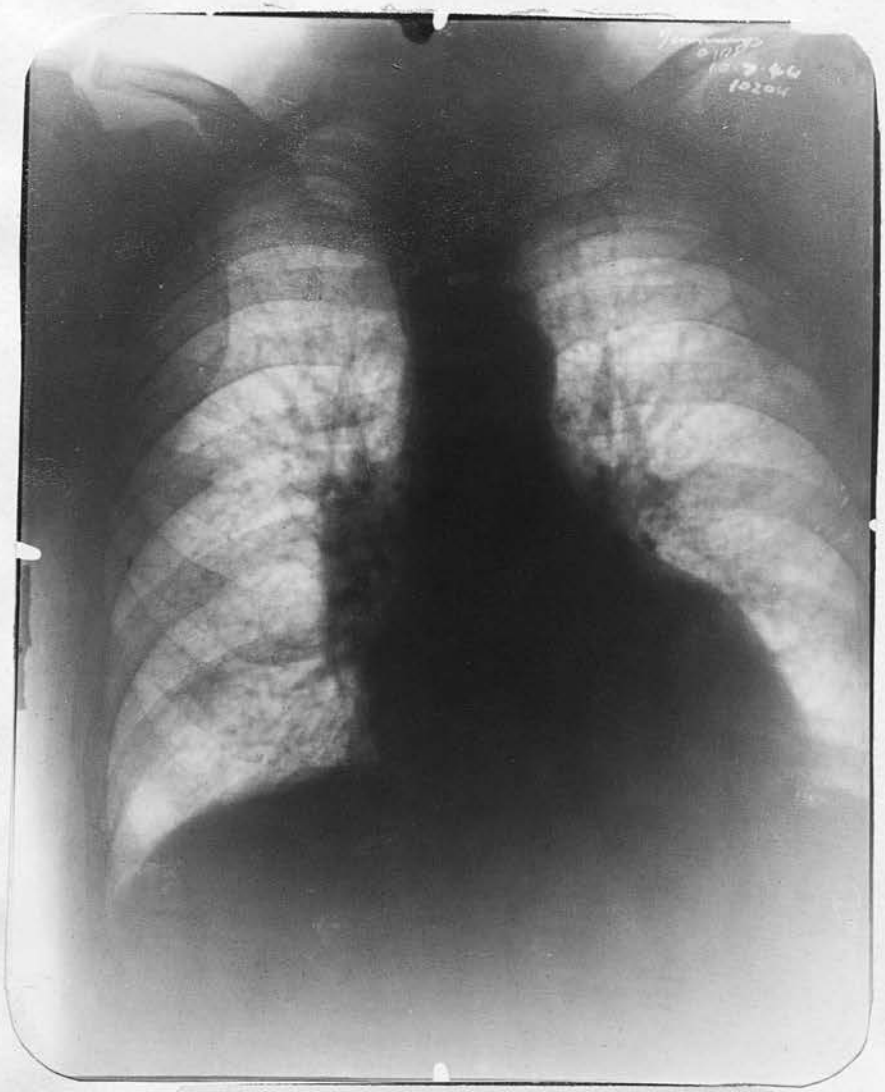
Duration: 3 years.

History: This man is constantly complaining of the above symptoms and gives the following history - at the age of 14 he came to work in the mines as a 'scraper-out' that is scaping out the 'cuttings' made by the coal cutting machine. At the age of 20 he was a coal cutter - this is a very dusty occupation. In 1914 having always been healthy he joined the Army and was discharged in December the same year. He informs me that he got wet through in the first month of his army life and developed a troublesome cough for which he reported sick. He was discharged from the Army as medically unfit. He has remained at the mines as a coal cutter ever since until some five years ago when he had to give it up on account of his breathlessness. His chest condition is getting worse and he is very breathless now. No oedema of the feet.

Examination: This man is very dyspnoec. It is the most striking feature about his condition. His respirations are 24 per minute and rather noisy. He has a slightly cyanotic tinge in his cheeks. Definitely barrel chested with ballooned up apices and prominent sternum. Wide subcostal angle. No congestion of great veins.

Respiratory excursion $37\frac{1}{2}$" - 39" expansion $1\frac{1}{2}$"

Apex beat in 6th interspace 5" from the mid line, that is to say the heart is enlarged one fingersbreadth beyond the nipple line.



CASE 22.

Vocal fremitus diminished.

On percussion the chest is hyper resonant all over especially in the apices. The liver is somewhat enlarged one fingersbreadth below the right costal margin.

Heart sounds are closed.

Resting pulse rate 100 per min. Occasional extra systole.

Vesicular breath sounds all over chest. No accompaniments.

Blood pressure 110/90 mms.Hg.

X-Ray Examination: Reveals Pulmonary Fibrosis and Emphysema. Radiologist reports no evidence of Pneumoconiosis.

Sputum Examination: No tubercle bacilli found.

This man is suffering from Pulmonary Fibrosis and marked Emphysema with commencing heart failure. How did he become like this? He was apparently healthy in August 1914 when he enlisted but was discharged some three months later as medically unfit. The truth probably was that he was unfit from the start but it took the way of life in the Army to demonstrate it. He was probably suffering from a mild degree of Emphysema in 1914 and was a coal cutter. Here the machine actually cuts the coal and the coal cutter attends the machine. In this capacity this man could manage his work but when placed in fresh air in the Army among many other fit young men his physical disability became apparent. He would be breathless and have a troublesome cough. He would be incapable of prolonged sever physical exertion and so would be discharged from the Army as physically unfit.

He went back to his coal cutting and here he would manage to keep going looking after the machine. Gradually, however,

the Emphysema would cause a Pulmonary Fibrosis and thus further incapacitate him.

On looking at his skiagram the first thing that strikes one is the hypertrophy of the left ventricle and also to a lesser extent of the right ventricle also. Pulmonary Fibrosis is also evident.

This man is quite incapacitated and can only walk a few yards before he has to stop and get his breath. Possibly his cardiac condition contributes to this but he has much lung fibrosis as can be seen from the skiagram and this would materially limit his activities and cause most of his symptoms by embarrassing the Pulmonary circulation.

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CASE 23. Pulmonary Fibrosis.

Andrew A. Age 49. Stoneworker.

Complains of shortness of breath and cough with expectoration of sputum. /

Duration: 2 years.

History: This man was quite healthy and joined up for military service in 1939 and served in the Army for two years in Category A 1. before being discharged. Since he was discharged he has been working as a 'stoneman'. Previous to that, in fact ever since the last war he was a 'timber-drawer' that is a man who removes the timber, props etc after the coal has been removed from the working place. This is an arduous and dangerous occupation but not unduly dusty. A 'stoneman's ' work is, however, very dusty, being exposed to coal dust, stone dust and the reek of explosives. He states that he has been troubled with his chest for a number of years, but he further states that his chest has got worse lately. He has now found that he is getting increasingly breathless - so much so that he cannot keep at work and is extremely breathless if he attempts the slightest exertion. He also has a frequent and persistent cough with expectoration of greenish yellow sputum. The cough is worse when he lies down and he has very disturbed nights on this account. As soon as he wakes in the morning he coughs up a lot of sputum.

Examination: Big, powerful looking man with a frequent short cough. Resting pulse rate 92 per minute.

Resting respiratory rate 24 per minute.

Weight 10½ stones. He used to weigh 12st. 7 lbs.

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Examination: Big, powerful looking man with a frequent short cough. Resting pulse rate 92 per minute.

Resting respiratory rate 24 per minute.

Weight 10½ stones. He used to weigh 12st. 7 lbs.

Breathing abdominal in type and muscles of chest somewhat atrophic. Myotatic irritability present.

Supraclavicular fossae filled up with distended voluminous lung apices.

Chest expansion 36" - 37½" = 1½".

Apex beat in 5th space ½" medial to the nipple line.

Superficial cardiac dullness diminished to 2½" from the middle line.

Vocal fremitus diminished.

Resonant percussion note - hyper resonant at apices.

Vesicular breath sounds without accompaniments all over chest

Heart sounds normal.

Vocal resonance diminished.

Blood pressure 185/130 mms.Hg.

X-Ray Examination: Pulmonary Fibrosis with Chronic Bronchitis.

Sputum Examination: Negative to Tubercle bacilli.

This man has been exposed to the dust hazard for some 25 years. He evidently had a Chronic Bronchitis for some years which caused a deterioration of his condition and he now has a Pulmonary Fibrosis which will probably progress. At intervals his general condition deteriorates - he loses weight, suffers from night sweats and has a slight temperature, but on investigation no evidence of Tuberculosis is found.

-ooOoo-

CASE 24. Pulmonary Fibrosis.

John T. Age 45. Scalloper-later Deputy.

Complains of tightness of chest and shortness of breath. Also cough.

Duration: 2 years.

History: Patient was quite a healthy man until two years ago when he developed a cough and breathlessness. He recovered from this and then had a second attack two months later. This condition of cough and breathlessness recurs if he gets the least chill. This breathlessness is worst in the mornings and so is the cough- he feels as if he has to clear his chest every morning. This man has done a lot of shot-firing in his time (5 years) although he is not doing it now, but he has noticed that his chest is bad if he is exposed to dust.

Examination: Intelligent man. Dyspnoec when taking his clothes off for examination. He states, incidently, that he always seems to be very breathless on Sunday evenings when he is in the habit of taking a walk. He has a hard cough with expectoration of thick black sputum.

Resting pulse rate 108 per minute.

Thoraco abdominal type of breathing.

His chest is well formed and moves easily but he says that he has to take a deep breath every few minutes "to satisfy his breathing as it were".

Apex beat in 6th interspace from the mid line. Left border of heart 1" medial to nipple line.

Chest excursion 36½" - 38" expansion 1½".

Heart sounds normal.

On percussion the chest is hyper resonant only in the apices

Vesicular breath sounds with no accompaniments.

Blood pressure 120/90 mms.Hg.

Sputum Examination: Negative to Tubercle bacilli.

There is very little to find on examination of this patient's chest but it is a fact that many of these men become grossly incapacitated due to Pulmonary Fibrosis without there being anything very gross to find on clinical examination.

-ooOoo-

CASE 25. Emphysema.

Clement F. Age 56. Stoneman.

Complains of cough and breathlessness.

Duration: 10 years.

History: Some 10 years ago this man had an attack of Bronchitis and in 1933 he had a crushed chest and had his ribs strapped. Following these happenings he noticed that "he was getting thick in the chest and short of wind". He also had a slight cough - more a "tickle in the throat" he said. Later, his cough became worse until he states he nearly "used to cough himself unconscious". His chief trouble now is that he cannot take any sustained strain as he gets breathless. If he has to haul on a rope or carry a weight he gets very breathless. He also sometimes wakes in the night with nocturnal dyspnoea. He works as a 'stone worker'. This is very dusty as one might expect and to make things worse the place is very heavily dusted with stone dust before the explosive charge is detonated.

EXAMINATION: This man has a typical emphysematous type of chest with a tinge of cyanosis in the lips.

Chest expansion 1¼" - 38" to 39½".

Diminished vocal fremitus.

The area of superficial cardiac dullness is very much diminished as it is hardly possible to elicit any dullness to percussion over the cardiac notch.

Hyper resonant percussion note all over the chest.

Vesicular breath sounds all over chest with some rhonchi at the bases. Vocal resonance diminished. Heart sounds healthy.

Resting pulse rate 90 per minute. Respirations 22 per minute

Blodd pressure 140/90 mms.Hg.

Sputum Examination: Negative.

This man then is suffering from Emphysema with commencing right sided Cardiac failure. It would seem that the initial illness was an acute bronchitis and that the cough persisted and in the course of 10 years he has now developed a definite Emphysema.

-ooOoo-

CASE 26. Bronchiectasis.

Ralph E. Age 55. Coal Hewer.

Complains of cough and breathlessness, sweating on exertion.

Duration: 26 years (since 1918)

History: Patient was quite healthy and worked as a miner until 1914 when he enlisted in the Army. He was graded A 1. until 1918 when he was gassed he states with Chlorine. He worked for four months as a coal hewer after demobilization but found the work to be too much for him and applied for a pension and was successful. In 1935 he had to give up work underground altogether. Every morning at 6.30 he gets up and coughs for an hour expectorating a large amount of sputum which is first thick and yellow - later grey. This morning cough is so severe that he nearly goes into a syncopal attack by his description "everything goes black and I feel dizzy".

Family History: Father alive age 80. Mother died age 65 - heart disease.

Examination: Short thick set man. Very dyspnoec. Numerous audible accompaniments to his breathing. Emphysematous type of chest, $\frac{1}{2}$" expansion 38" to 38$\frac{1}{2}$". Very full in apices wide subcostal angle. No clubbing of fingers.

Apex beat in 6th interspace 4" from middle line.

Vocal fremitus diminished.

On percussion the chest was hyper resonant all over. Superficial cardiac dullness diminished one fingersbreadth to left of nipple line.

Vesicular breath sounds all over with numerous rhonchi and

crepitations at both bases. Heart sounds normal.

Vocal resonance diminished.

Blood pressure 140/90 mms.Hg.

Sputum Examination: Negative to Tuberculosis.

This man has essentially a Bronchiectasis. He had a Chronic Bronchitis and Emphysema to start with and this has gone on to Pulmonary Fibrosis and hence Bronchiectasis. The morning cough, the typical sputum which separates into three layers on standing and the history clearly indicate a Bronchiectasis condition.

-ooOoo-

CASE 27. Pulmonary Fibrosis.

John W. T. Age 46. Coal Cutter & Driller 24 years, Stone worker 1½.

Complains of cough and breathlessness.

Duration: 7 - 8 years.

History: This man has been a coal cutter and driller since the last war until 18 months ago when he became increasingly breathless and had to have a lighter job - i.e. as a stoneworker. He found that about 18 months ago when he got among the stone dust and coal dust he could not get his breath and could not keep at work for long. This condition became worse and so did the cough which had now developed until about six weeks ago he had to give up work altogether.

Examination: Powerful, healthy looking man. Has a frequent short cough and is definitely dyspnoec.

Resting pulse rate 84 per minute. Respirations 24 per min.

Thoraco abdominal type of breathing.

Apex beat in 5th interspace 4" from the middle line.

Left border cardiac dullness also 4" from middle line.

Area of superficial cardiac dullness not diminished.

Hyper resonant percussion note in apices. Resonant note at other areas of the chest. Heart sounds normal.

Vesicular breath sounds except at bases where numerous rhonchi can be heard.

Blood pressure 160/120 mms.Hg.

Sputum Examination: Negative to Tubercle bacilli.

X-Ray Examination: Radiologist reports "No evidence of abnormal thoracic changes. Some evidence of Chronic Bronchitis"

There is little to be found on clinical examination of this man's chest and yet he is very definitely incapacitated. One might venture to say that this is an early Pulmonary Fibrosis.

-ooOoo-

CASE 28. Chronic Bronchitis and Emphysema.

John H. Age 51. Stone worker.

Complains of cough and breathless.

Duration: 3 - 4 years.

History: About three to four years ago this man began to notice that he was becoming very breathless. He was quite unable to run or to even walk up inclines without difficulty and he was unable to keep up his standard of work. The cough likewise began to trouble him a good deal and he found that it was worst when he lay down at night. He gets attacks of coughing by day as well as by night. When he coughs there is little or no mucous produced. He has been employed as a coal cutter for 12 years and after that was a coal hewer for about 4 years. For the last 7 or 8 years he has been employed as a stone worker.

Examination: Rather stout and sallow but healthy man. Rarely ill.

Pulse 84 per minute. Respirations 18 per minute.

Large emphysematous type of chest. Supraclavicular fossae prominent. Chest moves very little on respiration which is chiefly of the abdominal type. Wide subcostal angle.

Left border of cardiac dullness one fingersbreadth within nipple line in the 5th interspace.

Chest expansion 1" = 41" to 42".

Resonant percussion note all over chest.

Vesicular breath sounds all over chest with however diminished air entry. No accompaniments. Heart sounds normal.

Blood pressure 160/120 mms.Hg.

Sputum Examination: Negative.

-ooOoo-

CASE 29. Emphysema.

F. G. G. Age 45. Stoneworker.

Complains of cough, breathlessness and tightness of chest.

Duration: 7 years.

History: This man has always been healthy until about 7 years ago when he began to notice that he was becoming increasingly breathless on exertion. At the same time he felt a tight feeling in his chest. Finally in 1940 he was admitted to a sanatorium for investigation but was discharged after three weeks as non-tuberculous. He had been x-rayed and was told he had a 'dusty lung'. He started work at the age of 14 on the screens where he remained for six months. He then started to work underground first as a hewer and later with the scalloping machines (coal cutters). For the last 12 years he has worked as a stoneman.

Examination: Chest well formed and well muscled. Thoraco-abdominal type of breathing. Chest moves easily within a limited respiratory excursion. Rib cartilages ossified and ribs widely spaced indicating a degree of emphysema, which is borne out by the fact that the supraclavicular fossae are well filled.

No clubbing of fingers. Definite scoliosis of spine.

Pulse resting 72 per min. Respirations 22 per min.

Frequent short cough. Apex beat in 5th interspace 1½" from the mid line.

Area of superficial cardiac dullness somewhat diminished, three fingersbreadths within the nipple line.

Resonant percussion note all over chest - hyper resonant in the apices.

Vesicular breathing with moist accompaniments on inspiration

and rhonchi on expiration all over the cheat. Heart sounds normal.

Sputum Examination: Negative to Tuberculosis.

X-Ray Examination: Evidence of Emphysema.

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CASE 30. Dust Bronchitis.

James S. Age 16. Coal Separator Boy.

Complains of coughing and spitting.

Duration: 14 months.

History: This boy has been working at a 'separator' for 14 months. This work consists of separating the coal into various sizes varying from large coal to coal dust. This work is carried on within a building. He states that on the bottom of the tubs as they come in to be tipped there is always a layer of stone dust an inch or more deep. The result is that the air is thickly impregnated with coal dust and to a lesser extent with stone dust. His chief complaint is that persistent cough with expectoration of sputum - evidently muco-purulent sputum. He states that he sleeps quite well but that when he gets up in the morning he has a severe attack of coughing and brings up a lot of greenish black sputum. There is no complaint of breathlessness.

Examination: Small pale youth. Chest moves easily on respiration which is of the Thoraco-abdominal type. He has an occasional loose cough..

Chest expansion 28" - 30½" = 2½"

Apex beat in 5th interspace 2½" from the middle line.

Resonant percussion note all over the chest.

Vesicular breath sounds all over chest with numerous rhonchi on expiration especially at left apex and right base.

Sputum Examination: Negative.

A case of Chronic Bronchitis in a youth caused by work in an excessively dusty atmosphere.

-ooOoo-

CASE 31. Bronchiectasis.

Robert McR. Age 55. Coal Hewer.

Complains of cough and breathlessness, also pains in the chest.

Duration: 25 years. (since 1919)

History: This man was quite healthy and was employed as a coal hewer in the mine until 1914 when he enlisted. He then came home in 1918 and the following year while awaiting to be discharged he contracted "Double Pneumonia". After recovery from this he was discharged from the Army and resumed work as a stoneman in the mine. He was not using explosives then but has done so both before this and after. He worked thus for a year and then resumed work as a coal hewer and has done so ever since until 1938 when he had to give up mining altogether and since then he has worked in various factories. He worked thus at three places Leeds, Birmingham and Coventry. In each of these places he suffered from Pneumonia he states. Ihave records of two of these attacks - one from Leeds and the other from Selly Oak Hospital Birmingham. The first attack is described as an acute Bronchitis and Asthma and the second is described in the report as being 'treated as a Lobar Pneumonia'. He appears to have had Pneumonia four times in the past 5 years.

Examination: Big powerful looking man though somewhat wasted. He is very definitely dyspnoec and has a frequent troublesome cough. He has a big well formed chest with Thoraco-abdominal type of breathing. Some myotatic irritability and myoedema present indicating poor general condition.

Chest expansion $38\frac{1}{2}$" - 40" - $1\frac{1}{2}$".

Apex beat is in 5th interspace $4\frac{1}{2}$" from the middle line.

Vocal fremitus decreased.

On percussion the chest is somewhat hyper resonant although not to a marked degree - more apparent at the apices.

Faint vesicular breath sounds with numerous rhonchi especially on expiration. Heart sounds normal.

Blood pressure 120/80 mms.Hg.

Sputum Examination: Negative to Tubercle.

This man is in my opinion suffering from Bronchiectasis. The physical signs in this condition are frequently only those of the Bronchitis with the possible addition of right sided cardiac failure and its attendant cyanosis. These signs are wanting in this case but the history, the morning cough, the sputum and the cachectic condition of his body suggest it very strongly. In my opinion the pneumonic attacks are not pneumonia at all but simply due to an exacerbation of the Bronchiectasis with some associated pleurisy. His response to Sulphapyridine is explained by the fact that this would have a lethal effect on the Streptococci and Pneumococci and other organisms in the dilated bronchi and would thus improve his condition by lessening the toxaemia.

-ooOoo-

CASE 32. Emphysema with Right Sided Cardiac Failure.

Wilfred H.E. Age 45 Stoneworker & Coal Cutter.

Complains of cough and increasing breathlessness.

Duration: 2 years.

History: The above patient complains of extreme breathlessness and a frequent troublesome cough with expectoration of sputum. The breathlessness is extreme - even filling his pipe causes him to become breathless. The cough is persistent and he cannot sleep for it and to use his own expression "you could light the fire with the sputum" - meaning that it is coal black in colour. About two months ago he complained of a slight pain on the right side of the chest and appeared to have a clinical pleurisy. While examining him for this complaint his liver was found to be enlarged about one fingersbreadth below the right costal margin. He worked as a stone worker for about nine years and was then a coal cutter on the arc wall machines for twelve years. He was then a coal hewer with pneumatic picks for a further seven years.

Examination: Slight build, obviously dyspnoec. Chest moves easily on respiration which is of the thoraco-abdominal type. Horizontal ribs widely spaced.

Respirations 24 per minute.

Chest expansion $33\frac{1}{2}$" - 35" = $1\frac{1}{2}$".

Apex beat in 6th interspace $4\frac{1}{2}$" from the middle line.

Hyper resonant percussion note all over the chest. Some increased Liver dullness below the right costal margin.

Vesicular breath sounds with an occasional rhonchus.

Heart sounds healthy.

Sputum Examination: Negative to Tuberculosis.

X-Ray Examination: Evidence of Emphysema.

This patient is evidently developing a right sided cardiac failure.

-ooOoo-

CASE 33. Chronic Bronchitis = Emphysema.

William R. Y. Age 50. Stone Mill Worker.

Complains of cough with sputum. Paroxysmal breathlessness.

Duration: 28 days.

History: He has worked for eight years as a 'putter' followed by ten years as a coal hewer with hand pick. He then began to work at 'bank' in a brickyard. He has remained there for some eight years. He states that this work is very dusty. He states he has always been healthy but about a month ago he began to be troubled with a cough with expectoration of sputum. He also states that he wakes up every morning about 4 a.m. in a breathless state. He then starts to cough and cannot sleep after that. He has to get up and open the doors and stand there in the open until the fresh air eases his breathless state.

Examination: Well built man of good physique. Thick powerful chest. Moves easily on respiration. Supraclavicular fossae well filled but not an emphysematous chest.

Resting pulse rate 64 per minute.

Chest measurements $37\frac{1}{2}$" - $38\frac{1}{2}$" expansion 1".

Superficial cardiac dullness 4" from the mid line that is 1" within the nipple line.

Resonant percussion note all over chest.

Vesicular breath sounds all over the chest. No accompaniments.

Blood Pressure 140/95 mms.Hg. Heart sounds normal.

This appears to be a case of dust Bronchitis with commencing Emphysema. The paroxysmal dyspnoea is very suggestive of this especially occurring as it does in the early hours of the

morning. This has been a feature in several of my cases and they all say the same thing - they have to get up and open the door and the fresh air helps them.

The bronchitis itself is, of course, the result of a physiological condition causing a hyperaemia of the bronchioles and so hypersecretion of mucous to get rid of the inhaled dust.

-ooOOOoo-

CASE 34. Miners Asthma.

Percy N. Age 41. Scraper-out 1½ years
Stone-duster 3 years.
Putter 12 years.

Complains of breathlessness and cough.

Duration: 10 years.

History: This man was healthy until 1934 when he found himself developing a cough and breathlessness on exertion. At that time he was working as a 'putter' but he became so bad that he finally had to get light work on the 'engine planes'. He worked there for three years in a very dusty atmosphere being employed as a greaser and also as a stone-duster until 1937 when he developed Lobar Pneumonia. After he recovered he worked for two years on light work in this dusty occupation and finally had to give up work altogether in 1939. He is now very breathless on exertion and has a chronic cough with expectoration of greenish black sputum. The cough is worst in the early morning when he has to get up at about 4 a.m. and open the door and sit there until he has coughed up all the sputum and thereafter he can sit by the fire again. He uses an Adrenaline spray frequently especially in the early morning.

Examination: Resting pulse rate 80 perminute.

Resting respiratory rate 28 per minute.

Chest well formed but ribs horizontal and the chest appears to be held in the position of full expansion - emphysematous type of chest.

Chest measurements 35½" - 36" expansion ½"

Chiefly abdominal type of breathing.

Area of superficial cardiac dullness diminished being about 1" within the nipple line.

Resonant percussion note all over the chest except the apices where it is hyper resonant.

Vesicular breath sounds with however prolongation of expiration and loud rhonchi during expiration. These rhonchi are much louder on the left side than the right side and are more apparent at the apices especially the left apex.

Blood pressure 125/85 mms.Hg.

Sputum Examination: Negative to Tuberculosis.

X-Ray Examination: Emphysema and probable Asthmatic changes.

This man is subject to acute exacerbations of his condition during which he has much recourse to an Adrenalin spray (1 in 100). This acute condition goes on for one or two days and then subsides into his usual chronic state. He has not worked for six years and is permanently incapacitated.

-oooOOOooo-

CASE 35. Pulmonary Fibrosis.

Peter D. Age 41. Coal Cutter.

Complains of cough with expectoration of sputum.

Duration: 2 years.

History: This man has been employed as a coal cutter on the compressed air machines for 12 years. The coal cutting machines are operated by compressed air and as there is shot-firing going on throughout the shift the work is very dusty. For the past two to three years this man has found that he has to wake up early and as he states "cough himself clear". He goes on coughing for about an hour and then has a period of relief. He states he also gets periods of breathlessness and is very breathless if he hurries.

Examination: Thin man but of good physique. Athletic build. He has a short frequent cough which is the most noticeable feature about him. His chest moves easily on respiration which is of the thoraco-abdominal type.

Resting pulse 80 per minute. Respirations 20 per minute.

Chest measurements 34½" - 36" expansion 1½".

Apex beat in 5th space 4" from the mid line and ½" within the nipple line.

Resonant percussion note all over chest.

Vesicular breath sounds all over chest with no accompaniments.

Heart sounds normal.

Sputum Examination: Negative.

This is a case of a healthy man of good physique who has been exposed to extreme concentrations of dust for 12 years.

His cough is a protective mechanism to clear his bronchioles of inspired dust. Only lately has this become troublesome and only lately has he begun to be breathless so that his lymphatics are evidently becoming choked with dust and a fibrotic response is being set up which if he goes on working as a coal cutter will culminate in a chronic bronchitis with emphysema.

-ooOOo-

CASE 36. Emphysema.

W. S. Aged 59. Scalloper.

Complains of cough with sputum and breathlessness.

Duration: 6½ years.

History: This man worked as a 'scalloper' for 20 years - this is a coal cutting machine and is operated by compressed air. At the age of 52 he developed a cough with expectoration of sputum and breathlessness. The cough was worse during the night and the early morning and he states he had to go outside to get fresh air and to cough his chest clear. He has been off work nearly seven years and states that he has improved somewhat but he is still very breathless especially if he goes up a bank. The cough is still frequent and he is very distressed on exertion.

Examination: Short thick set man with slightly cyanotic tinge in the cheeks. He has a thick amphysematous type of chest with the supraclavicular fossae grossly distended and typical barrel chest.

Chest measurements 35½" - 37" expansion 1½".

Voacl fremitus diminished.

Hyper resonant percussion note all over the chest.

Faint vesicular breath sounds all over the chest with no accompaniments.

Vocal resonance diminished.

Heart sounds normal.

Blood pressure 170/120 mms.Hg.

Sputum Examination: Negative.

In this case the whole picture is one of Emphysema.

-ooOoo-

CASE 37. Chronic Bronchitis - Emphysema.

John B. Age 57. Hewer 15 years then Screen worker.

Complains of cough and breathlessness.

Duration: 3 - 4 years.

History: This man had Lobar Pneumonia about 13 years ago from which he fully recovered. He had a second attack of Pneumonia some four years ago following which he had a chronic cough with dyspnoea. This cough is always present but he is liable to acute exacerbations especially in the winter. It is always worst in the mornings when he has to get up to cough his chest free. It is worst also when he lies down and as a result he frequently passes the night sitting up in a chair. Thick frothy sputum is expectorated - in water it appears to be of the nummular type. This man is also extremely breathless especially so on effort. He was a coal hewer (hand pick) for 15 years following which he was a scalloper for about a year. He then came to the surface and has worked at various forms of employment.

Examination: Small thin man. Obviously breathless. Frequent loose cough.

'Fullness' of apices but no barrel chest.

Chest measurements 32" - 33" expansion 1".

Cardiac dullness definitely diminished.

Resonant percussion note all over chest especially at apices.

Vesicular breath sounds with rhonchi on expiration all over the chest. Heart sounds healthy.

Blood pressure 160/110 mms.Hg.

Sputum Examination: Negative to Tuberculosis.

X-Ray Examination: Chronic Bronchitis and Emphysema.

CASE 38. Early Pulmonary Fibrosis.

George L. Age 35. Coal Cutter.

Complains of cough and pain in the chest.

Duration: 4 - 5 years.

History: This man has been working in the mine since the age of 14. For a long time he was a 'filler' but for the past 6½ years he has been a coal cutter using the compressed air machines. He first began to suffer from a cough five years ago, but it did not trouble him a great deal. Later, however, it became worse and it was especially troublesome at night. This cough has persisted and now he frequently developes a pain across the upper part of the chest and has to lie down until it goes off. This pain is most frequent during the night and is associated with a cough and he has to sit up in bed until he gets relief. He expectorates a good deal of blackish mucous.

Examination: Healthy appearance. Well formed chest which moves easily on respiration although the respiratory excursion is limited.. Chest expansion 1" - 36½" to 37½".

No diminution of the superficial cardiac dullness.

Resonant percussion note all over the chest with the exception of the apices where it is hyper resonant.

Vesicular breath sounds with an occasional rhonchus at both bases. Heart sounds normal.

Blood pressure 135/90 mms.Hg.

Sputum Examination: Negative.

X-Ray Examination: No abnormal Thoracic changes. This man has been incapacitated for several months despite the negative radiological findings.

-ooOoo-

CASE 39. Emphysema - Pulmonary Fibrosis.

Thomas P. Age 44. Coal Hewer.

Complains of cough and breathlessness.

Duration: 2 years.

History: This patient was always healthy until recently when he felt he was getting like an old man on account of his breathlessness. This has come on gradually over a period of between one and two years. He states that the breathlessness is worst on a Monday morning and that it gradually improves during the working week until, by Friday, his chest is fairly free. At the week-end, however, the chest becomes tight again and on the Monday morning he has to get up and cough. This produces a great deal of frothy mucous and thereafter he has a dry cough. He notices the breathlessness especially when he is coming 'outbye' and when walking home. He also states that he cannot take a deep breath - something prevents him. He works as a coal hewer with a pneumatic pick and has been doing this for three years. Before that he was a 'filler' and before that a coal hewer with a hand pick.

Examination: Well built short man.

Resting pulse 84 per minute. Respirations 20 per minute.

Chest moves easily on respiration which is of the thoraco-abdominal type. The chest, however, is definitely emphysematous.

Apex beat in 5th interspace in the nipple line.

Chest measurement 35" - 36½" expansion 1½".

Hyper resonant percussion note all over the chest but especially at both apices and bases.

Vesicular breath sounds, with accompaniments all over the chest

Heart sounds healthy.

Blood pressure 130/85 mms.Hg.

Sputum Examination: Negative.

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CASE 40. Chronic Bronchitis and Emphysema.

Thomas L. Age 62. Coal Hewer.

Complains of cough and breathlessness.

Duration: 10 years.

History: This man states he enjoyed good health all his life until about 10 years ago at the age of 52 when he developed an attack of Bronchitis. He recovered from this but was subject to attacks of bronchitis after this especially in the winter until finally he was never free from cough and was subject to more severe attacks of acute bronchitis from time to time. He has worked underground as a coal hewer in the mine for 49 years.

Examination: Slight build. Pale complexion. Dyspnoec but not to marked extent. Respirations 24 when removing his clothes. His body is well nourished. Breathing is almost entirely abdominal. Chest expansion is very poor $33\frac{1}{2}" - 34" = \frac{1}{2}"$.

Apex beat in 5th interspace in the nipple line.

On percussion the chest is resonant all over.

Breath sounds harsh vesicular with prolongation of expiration and numerous rhonchi. No moist sounds.

Blood pressure 150/72 mms.Hg. Heart sound normal.

His sputum is thick and white with no muco pus. The typical 'crachets perles' of Laennec.

Sputum Examination: Negative.

This man in my opinion is suffering from typical Chronic Bronchitis with some accompanying Emphysema witness the poor -expansion and the dyspnoea.

-ooOoo-

CASE 41. Emphysema and Pulmonary Fibrosis with Hyperpiesis.

Sedman W. Age 64. Coke Burner.

Complains of cough and nocturnal dyspnoea.

Duration: 10 years.

History: This man has worked in a coke yard all his life some 51 years. His work entailed exposure to coal and coke dust and also to the noxious vapours from the coke ovens when they were opened up. For many years he was subject to attacks of winter cough and these winter coughs became worse being very troublesome and preventing sleep so that he lost work. Some six years ago he had what appeared to be a slight haemoptysis and this recurred two years later.

Examination: This man is of very poor physique thin and worn in appearance. Some dyspnoea on exertion and occasional loose cough with expectoration of thick sputum. Pulse is of the high pressure type and the artery is hard and tortuous. Chest moves poorly on respiration - thoraco-abdominal in type.

Expansion is 1" from 32½" - 33½". There is some bulging of the supraclavicular fossae. He has a wide costal angle.

Apex beat is in the 6th interspace 4¼" from the middle line and beyond the nipple line.

The liver is enlarged downwards about two fingersbreadth below the right costal margin.

The percussion note is hyper resonant all over the chest and is especially so in the apices.

Breath sounds are harsh vesicular - definitely harsher on the right side. No accompaniments. Heart sounds healthy.

Blood pressure 220/120 mms.Hg.

Sputum Examination: Negative to Tuberculosis.

X-Ray Examination: (1st) Showed no sign of Tuberculosis but a Pulmonary Fibrosis.

(2nd) Showed no present evidence of Tuberculous infection. Some early peribronchial infiltration.

This X-Ray would suggest an aortitis as there is some broadening of the aortic shadow. The blood pressure at this time was 240/110 mms.Hg.

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CASE 42. Chronic Bronchitis and Emphysema.

William F. Age 56. Coal Hewer.

Complains of cough and breathlessness. Faintness on exertion.

Duration: 5 years.

History: This man was quite healthy until he was 30 years of age when he states he got crushed in the chest by a fall of stone in the mine. After this, he states he was employed in a dusty seam and he started to have attacks of Bronchitis and was frequently off work with this. These attacks have gradually increased in frequency and duration until he has had to give up work belowground and come to the surface. He has been employed as a coal hewer all his life until some five years ago.

Examination: This man is of very poor physique, thin and poorly developed. His colour is good but he has some clubbing of the fingers and a frequent loose cough even when being examined. There is some bulging of his supraclavicular fossae. He has an emphysematous type of chest. The angle of Louis is well marked in the manubrium. His breathing is of the thoraco-abdominal type and he is definitely dyspnoec.

Chest expansion is $33\frac{1}{2}$" - 35" = $1\frac{1}{2}$".

The apex beat is in the 6th interspace $3\frac{3}{4}$" from the middle line.

Superficial cardiac dullness diminished by one fingers-breadth. Vocal fremitus diminished.

The chest is hyper resonant to percussion all over especially at the apices.

Breath sounds are vesicular all over. One or two rhonchi at the bases otherwise nil. Heart sounds healthy.

Blood pressure 160/100 mms.Hg.

Sputum Examination: Negative.

This man has reached the stage when he is rapidly becoming a chronic invalid. He will probably have a Pulmonary Fibrosis and traction by the contracting fidbrous tissue will cause the next stage that of Bronchiectasis which will cause complete invalidism with finally right heart failure and death.

-ooOoo-

CASE 43. Chronic Bronchitis and Emphysema.

Andrew G. Age 57. Coal Hewer & Stoneman.

Complains of cough and breathlessness.

Duration: 5 years.

History: This man states he had Pneumonia when very young and that this was followed by attacks of Bronchitis and Asthma ever since. He states he worked at the mines until he was 16 and then changed his work to that of a farm labourer at which he remained for eight years. During this time as a farm labourer, his cough, which had been troublesome when he was a miner, improved considerably and he had no asthmatic attacks except when stacking hay or corn when the dust made him cough. An attack is characterised by 'tight breathing' and 'wheezing' in the chest with spasmodic attempts to cough. Sometimes the dyspnoea lasts two to three days before he gets relief. He works down the mine in the 'airways' now - doing any odd work that is necessary. Originally he was a coal hewer and stoneman.

Examination: A small pale muddly complexioned man. Definitely dyspnoec on exertion. Some clubbing of the fingers. The supraclavicular fossae are ballooned out and the ribs are widely spaced. He has also a wide subcostal angle. Nutrition fair. Vocal fremitus diminished.

Chest expansion 1" from $33\frac{1}{2}$" - $34\frac{1}{2}$".

Apex beat in 5th interspace $3\frac{1}{2}$" from the mid line.

On percusion his chest is hyper resonant all over especially in the apices.

On auscultation there is diminished vocal resonance and the breath sounds are faint vesicular all over. No accompaniments

Blood pressure 140/90 mms.Hg. Heart sounds healthy.

Sputum Examination: Negative to Tuberculosis.

Emphysema causes serious obstruction to the Pulmonary circulation and may cause hypertiophy and dilatation of the right ventricle and general venous congestion throughout the body. The destruction of the elastic tissue of the alveoli which is one the significant changes in Emphysema helps to produce the next stage of Pulmonary Fibrosis so that when this contracts a condition of Bronchiectasis may become established.

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CASE 44. Pulmonary Fibrosis.

Anthony B. Age 42. Coal Hewer.

Complains of shortness of breath and cough.

Duration: 7 years.

History: This man was in good health until he had an attack of lobar pneumonia some eight years ago. He recovered slowly from this but eventually resumed work after three months. He found that he was unable to continue at his own work as a coal hewer and was given light work. After a year of this, however, he had to give up work altogether as the dust made him cough and he was unable to carry on at work owing to increasing breathlessness. He states that his cough is very troublesome especially at night and he coughs up much sputum. In the mornings he coughs so much that it takes him till midday to "get his lungs cleared". He has not worked for seven years.

Examination: Thin spare build. Muddy complexion. Definitely dyspnoec. Clubbing of the fingers. Chest is of the emphysematous type.

Respirations 20 per minute. Resting pulse rate 76 per minute

Air entry poor. Expansion $1\frac{1}{2}$" from $35\frac{1}{2}$" - 37".

Abdominal type of breathing.

Apex beat in 6th interspace 4" from the mid line.

Superficial cardiac dullness diminished by three fingersbreadths inside the nipple line i.e. $2\frac{1}{2}$" from the mid line.

Hyper resonance on percussion very marked especially at apices and bases.

The liver is enlarged three fingersbreadths below the right costal margin.

Vocal fremitus and resonance both diminished.

Vesicular breath sounds with occasionally fine crepitations at the apex of inspiration - heard at the bases. Heart sounds healthy

Sputum Examination: Repeatedly negative.

X-Ray Examination: Pulmonary Fibrosis and Emphysema.

This man complains of cough and breathlessness and pain over the Liver which is enlarged. On X-Ray examination there is Pulmonary Fibrosis and Emphysema. One concludes that the pain is due to Liver congestion due to weakness of the right side of the heart caused by the embarrassment of the pulmonary circulation brought on by pulmonary fibrosis.

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CASE 45. Fibroid Phthisis.

Charles H. Age 44. Stoneman previously Deputy.

Complains of coughing especially at night time.

Duration: 16 months.

History: This man has always been healthy until March 1943 when he developed Pleurisy. He had a cough, some pain on the right side of the chest and a friction rub which was, however, only present for about twenty-four hours. He had no pyrexia. After this he did not "pick up very well" and he found that he was short of breath and had a persistent cough. He also lost some weight and felt poorly generally. He was referred to the Tuberculosis Dispensary as a case of possible 'dust reticulation' but was found to have pulmonary Tuberculosis. He has always worked in the mines and was employed as a stoneman and previously to that as a 'deputy'. This man would be exposed to high concentrations of stone dust in both types of employment.

Family History: His father still alive aged 89. No history of chest trouble in the family.

Examination: Poorly developed chest with myotatic irritability on percussion.

Apex beat in 5th interspace 3" from the middle line that is one fingersbreadth medial to nipple line.

Resonant percussion note over most of chest except at left apex when a dull note was obtained and at both bases when it was hyper resonant.

Vesicular breath sounds with prolongation of expiration at apices and mid zone, and moist crepitations at the height of inspiration. Very definite cog-wheel breathing.

Blood pressure 125/70 mms.Hg. Heart sounds healthy.

X-Ray Examination: Early Tubercular changes in both lungs.

The writer has included this case in this series as for some time he was suspected to be a case of Silicosis - that is at the onset of his condition. The differential diagnosis between an early silicosis and early tuberculosis can be very difficult. In old retired miners Tubercle bacilli are often found in unsuspected cases.

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CASE 46. Emphysema.

George W.H. Age 58. Deputy Overman.

Complains of cough and shortness of breath.

Duration: 1 year.

History: This man has worked all his life in the mine with the exception of three years during ths last war. He has always enjoyed exceptionally good health. About a year ago, however, he started to have "trouble with his chest" and found himself getting very breathless on the least exertion. At the same time he developed a cough. He fell off work and after a week or two he felt much better and began work again but after a few days he again felt breathless and the cough recurred. His cough is always worst in the mornings and the sputum is worst at that time. The sputum is thick and yellowish green in colour. He states that his chest is full of "wheezles"

Family History: No history of chest trouble in the family.

Examination: Healthy appearance, colour good. Chest moves poorly on respiration - mainly abdominal type of breathing. Thick emphysematous type of chest. Marked bulging of apices in supraclavicular fossae.

Chest expansion 1" from 37½" - 38½".

Resting pulse rate 64 per min. Respirations 20 per min. (resting)

Cardiac border (left) 3" from the middle line and about 1½ fingersbreadths medial to the nipple line. There appears to be some diminution of the superficial cardiac dullness.

Percussion note resonant all over chest especially at apices and bases.

Vesicular breath sounds all over chest with numerous

rhonchi during expiration. Heart sounds healthy.

Blood pressure 180/100 mms.Hg.

Sputum Examination: No evidence of Tuberculosis.

This man has been exposed to coal dust for some 40 years and has only recently developed a cough and breathlessness. The cause appears to be a developing Emphysema.

-ooOoo-

CASE 47. Chronic Bronchitis with Emphysema.

James M. Age 54. Coal Hewer - with Pneumatic Pick 4 years.

Complains of cough and breathlessness.

Duration: 1 year.

History: This man has worked in the mine at the coal face for over 40 years. For most of that time he was a hewer with the ordinary hand pick but about four years ago he began to work with the pneumatic pick. He states he was always quite well until about a year ago when he had an attack of pneumonia from which he made a slow recovery and which left him with a chronic cough.. The 'pneumonia' he refers to seemed to be an Influenzal Bronchitis. Pulse, respirations and temperature were all up but there was no definite consolidation and no tubular breathing. Since this attack he has been troubled with some breathlessness especially on exertion.

Examination: Thick set figure. Barrel shaped chest with definite 'fullness' of supraclavicular fossae and thick chest wall. Abdominal type of respiration.

Respiratory excursion $1\frac{1}{4}$" from 38" - $39\frac{1}{2}$".

Resting pulse rate 104 per minute. Respirations 20 per min.

Apex beat in 6th interspace 4" from the middle line.

Border of cardiac dullness 1" within the nipple line.

Heart sounds normal.

Resonant percussion note all over chest. Hyper resonant at /apices

Vesicular breathing - no accompaniments.

Blood pressure 130/90 mms.Hg.

Sputum Examination: No Tubercle bacilli found.

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CASE 48. Chronic Bronchitis with Emphysema.

Peter P. Aged 62 Wasteman.-repairing the return airways.

Complains of cough and breathlessness.

Duration: 3 years.

History: This man has been working in the mine for 42 years most of the time as a 'bottom cutter', a hewer and other work. About three years ago he began to notice that he was getting very breathless and could not walk very far without stopping for a rest. He was working at the time on the machines (coal cutters) on the arc walls and he finally had to give up this work on account of his breathlessness. After a period of some months off work he resumed again, but this time in the lighter work that he has at present. He also has been complaining of a cough which is very troublesome at nights. Sputum is thick and yellow.

Examination: Thin somewhat senile wasted appearance.

Resting pulse rate 64 per minute.

Resting respirations 20 per minute.

Chest emphysematous and marked angularity of the Sternum with scoliosis of the vertebral column.

Chest expansion $1\frac{1}{2}$" i.e. 33" - $34\frac{1}{2}$".

Apex beat in 5th interspace $4\frac{1}{2}$" from the mid line.

Apparent diminution of superficial cardiac dullness one fingersbreadth within nipple line.

Breathing chiefly abdominal type as there is little movement of the chest and the costal cartilages seem to be ossified.

Percussion note hyper resonant all over chest.

Vesicular breath sounds with rhonchi on deep expiration

all over the chest.

Heart sounds normal.

Blood pressure 150/95 mms.Hg.

Sputum Examination: Negative.

An evident case of Chronic Bronchitis with Emphysema.

-ooOoo-

CASE 49. Chronic Bronchitis.

James F. Age 38. Conveyor Filler.

Complains of cough and breathlessness.

Duration: 5 years.

History: This man was quite well until he had Broncho-pneumonia some five years ago. Since then he has had a cough but has not lost much working time through it. His cough was worst when he woke up especially if he had to wake up at night to go out to work i.e. at about 3 a.m. He only produced sputum in the mornings. He is not very breathless except when he has been exposed to high concentrations of dust and is coming 'out-bye' after his shift has ended. His work is very dusty and a lot of shot-firing takes place and he has been exposed to this for over 20 years. He states that in addition to the shot-firing the 'set' (of tubs) raises a great amount of dust - he is employed as a Filler filling coal on to a conveyor.

Examination: Well developed man who has a short frequent cough. Nutrition good.

Resting pulse rate 84 per minute.

Respiratory rate 16 per minute.

Thoraco-abdominal type of breathing.

Respiratory excursion 2" i.e. from $35\frac{1}{2}$" - $37\frac{1}{2}$".

Apex beat in 5th interspace $3\frac{1}{2}$" from the middle line.

Area of superficial cardiac dullness not diminished, the left border of the heart being 1" from the nipple line.

Resonant percussion note all over chest.

Vesicular breath sounds without accompaniments all over the chest.

Blood pressure 140/95 mms.Hg.

The emphasis here is on the cough - Chronic Bronchitis due to inhalation of dust over a long period.

-ooOoo-

CASE 50. Emphysema.

George P. Age 50. Stoneman.

Complains of shortness of breath and morning cough.

Duration: 3 years.

History: Patient has always been very healthy until three years ago when he gradually developed shortness of breath. After being off work some weeks he improved and went back to his own work as a 'stoneman'. He worked for a few weeks then his breathing again became troublesome and he was off work again. This time he started work 'at bank' and worked there for seven weeks in an atmosphere relatively free from dust, He was quite well during this time. He was then ordered down the pit again and worked for a week or two but was unable to continue and again came to the surface. He had another attempt at work belowground this time at the shaft bottom but his overman ordered him 'in bye' and again he had a sharp attack of 'Asthma' - the worst attack he had. Since then he has been quite unable to work belowground and has been at work on the surface in the brickyard ever since.

Examination: This man is dyspnoec especially on slight exertion. Well formed chest with however some bulging of the apices and wide costal angle. Rather barrel type of chest.

Resting pulse rate 88 per minute.

Respirations 28 per minute.

Chest measurements 37" - 38½" - expansion 1½".

Apex beat is in the 5th interspace 4½" from the middle line.

The percussion note is resonant all over the chest except

for the apices where it is hyper resonant.

Heart sounds normal and regular.

Breath sounds are vesicular all over the chest. An occasional rhonchus at the bases.

Blood pressure 130/90 mms.Hg.

Sputum Examination: Negative.

This case illustrates the onset of an Emphysema. The history is interesting.

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Summary of Cases.

Case.	Name.	Age.	Nature of Work.	History of Illness or Accident.	Dyspnoec.	Cough.	Sputum.	Duration of Exposure.	Nature of Incapacity.	Incapacitated. Wholly.	Incapacitated. Partially.
1.	Anthony H.	63	Coal Hewer.	Influenza.	+-+	+-+	Pos.	50 years.	Koniophthisis.	+	
2.	Peter C.	67	Coal Cutter.	Bronchitis,Pleurisy.	+-+	+-+	Pos.	52 years.	Koniophthisis.	+	
3.	Jos.E.R.	43	Stoneman,Master Shifter.	Pleurisy.	+-+-+	+-+	Pos.	28 years.	Silicosis & Tuberculosis.	+	
4.	Ralph K.	31.	Coal Cutter.	Spinal Injury,Bronchitis.	+-+	+-+	Neg.	15 years.	Dust Reticulation.	+	
5.	Edmund W.	47	Stoneman.	Bronchitis.	+-+-+	+	Neg.	32 years.	Dust Reticulation.		+
6.	Robert S.E	47	Filler,Stoneman.	Pain in chest.	+	+	Neg.	32 years.	Dust Reticulation.	+	
7.	Wm. J.	35	Stoneman.	"Coughs and Colds".	+-+-+	+-+	Neg.	20 years.	Dust Reticulation.		+
8.	John D.	48	Coal Cutter.	---	+-+	+-+	Neg.	20 years.	Non Specific Reticulation.		+
9.	John O.K.	52	Boiler Cleaner.	---	+-+	+-+	Neg.	38 years.	Non Specific Reticulation.		+
10.	Wm. L.	41	Stoneman.	Pleurisy.	+-+	+-+	Neg.	25 years.	Pulmy.Fibrosis,Early Reticulation.		+
11.	Thomas E.	57	Coal Hewer.	Crushed chest,Bronchitis.	+-+-+	+-+	Neg.	12 years.	Fibroid Phthisis.	+	
12.	Thomas H.	45	Coal Cutter.	Bronchitis,Haemoptysis.	+-+-+	+-+	Neg.	30 years.	Ch.Bronchitis,Emphysema.	+	
13.	Henry H.	32	Coal Filler.	Fractured Rib.	+	+-+	Neg.	17 years.	Pulmy.Fibrosis,Emphysema.	+	
14.	John G.B.	36	Coal Cutter.	---	+-+	+	Neg.	20 years.	Miners Asthma,Emphysema.	+	
15.	W.H.B.	33	Coal Cutter.	Head Colds.	+-+	+	Neg.	13 years.	Miners Asthma,Fibrosis.	+	
16.	John W.	39	Coal Filler.	Pneumonia.	+-+	+	Neg.	24 years.	Pulmonary Fibrosis.	+	
17.	Ambrose M.	57	Coal Hewer.	---	+-+-+	+-+	Neg.	40 years.	Emphysema,Ch.Bronchitis.	+	
18.	Jas. D.	58	Overman.	---	+	+	Neg.	44 years.	Pulmonary Fibrosis.		+
19.	Edward A.	48	Coal Cutter.	Influenzal Bronchitis.	+-+	+-+	Neg.	34 years.	Emphysema.		+
20.	Thomas C.	62	Stoneman.	Bronchitis,Fracture Ribs.	+-+	+-+	Neg.	39 years.	Ch.Bronchitis, Emphysema.	+	
21.	James C.	52	Coal Hewer.	Acute Bronchitis.	+-+	+-+	Neg.	32 years.	Ch.Bronchitis, Emphysema.	+	
22.	W.T.J.	58	Coal Cutter.	---	+-+-+	+-+-+	Neg.	42 years.	Pulmy.Fibrosis,Emphysema.	+	
23.	Andrew H.	49	Stoneman,Timber Drawer.	---	+	+-+	Neg.	33 years.	Pulmonary Fibrosis.		+
24.	John T.	45	Scalloper.	---	+	+	Neg.	28 years.	Pulmonary Fibrosis.		+
25.	Clement F.	56	Stoneman.	Acute Bronchitis.	+-+	+	Neg.	30 years.	Emphysema.		+
26.	Ralph E.	55	Coal Hewer.	Gassed (1914-1918)	+-+	+-+-+	Neg.	28 years.	Bronchiectasis.	+	
27.	John W.T.	46	Coal Cutter.	---	+-+	+	Neg.	38 years.	Pulmonary Fibrosis.		+
28.	John H.	51	Coal Cutter,Stoneman.	Ch.Bronchitis,Emphysema.	+-+	+	Neg.	25 years.	Ch.Bronchitis, Emphysema.		+
29.	F.G.G.	45	Coal Cutter,Stoneman.	---	+-+	+	Neg.	30 years.	Emphysema.		+
30.	James S.	16	Coal Separator Att.	---	+-+	—	Neg.	1 year.	Dust Bronchitis.		+
31.	Robert M.	55	Coal Hewer.	Pneumonia.	+-+-+	+-+	Neg.	30 years.	Bronchiectasis.	+	
32.	W.H.E.	45	Coal Cutter,Stoneman.	---	+-+-+	+-+	Neg.	28 years.	Emphysema.	+	
33.	W.R.Y.	50	Coal Hewer,Putter.	---	+	+	Neg.	25 years.	Bronchitis with Emphysema.		+
34.	Percy N.	41	Putter,Stone Duster.	Pneumonia.	+-+-+	+-+-+	Neg.	20 years.	Emphysema(Miners Asthma)	+	
35.	Peter D.	41	Coal Cutter.	---	+-+	+	Neg.	26 years.	Pulmonary Fibrosis.		+
36.	W. S.	59	Coal Cutter.	---	+-+	+-+	Neg.	45 years.	Emphysema.	+	
37.	John B.	57	Coal Hewer.	Pneumonia.	+-+	+-+	Neg.	30 years.	Ch.Bronchitis,Emphysema.		+
38.	George L.	35	Coal Cutter.	---	+-+	—	Neg.	20 years.	Pulmonary Fibrosis.		+
39.	Thomas P.	44	Coal Hewer,Filler.	---	+-+	+	Neg.	28 years.	Emphysema.		+
40.	Thomas L.	62	Coal Hewer.	Bronchitis.	+-+	+-+	Neg.	49 years.	Ch.Bronchitis,Emphysema.		+
41.	Sedman W.	64	Coke Burner.	Bronchitis,Haemoptysis.	+-+	+-+	Neg.	50 years.	Emphysema,Pulmy.Fibrosis.		+
42.	William F.	56	Coal Hewer.	Crushed Chest,Bronchitis.	+-+	+-+	Neg.	40 years.	Ch.Bronchitis,Emphysema.		+
43.	Andrew G.	57	Coal Hewer,Stoneman.	Pneumonia.	+-+	+-+	Neg.	35 years.	Ch.Bronchitis,Emphysema.		+
44.	Anthony B.	42	Coal Hewer.	Pneumonia.	+-+	+-+-+	Neg.	20 years.	Pulmonary Fibrosis.	+	
45.	Charles H.	44	Deputy,Stoneman.	Pleurisy.	+-+	+	Pos.	27 years.	Fibroid Phthisis.	+	
46.	George W.H.	58	Deputy.	---	+	+	Neg.	40 years.	Emphysema.		+
47.	James M.	54	Coal Hewer.	Pneumonia.	+	+	Neg.	40 years.	Ch.Bronchitis,Emphysema.		+
48.	Peter P.	62	Coal Cutter, Hewer.	---	+-+	+-+	Neg.	42 years.	Ch.Bronchitis,Emphysema.	+	
49.	James F.	38	Coal Filler(Conveyor).	Broncho-Pneumonia.	+	+	Neg.	20 years.	Chronic Bronchitis.		+
50.	George P.	50	Stoneman.	---	+-+	+-+	Neg.	26 years.	Emphysema.		+

SUMMARY AND CONCLUSIONS.

Summary.

As a general practitioner in a coal mining district the writer has had occasion to examine many hundreds of miners in the course of routine and other examinations during the last ten years. The foregoing cases (fifty in all) are the only cases of true chronic pulmonary disease that I have encountered and investigated. The number of miners employed in this district is just over 3,000 and from this one can conclude that chronic incapacitating pulmonary disease is not common. Silicosis is exceedingly rare - in all these fifty cases only three cases of Silicosis and one of dust reticulation (or pneumoconiosis) were accepted by the Silicosis Board. That this disease does exist among bituminous, coal miners is indisputable but it does not occur on anything like the scale that it does among Anthracite miners.

Minor degrees of pulmonary disability do occur, however, among bituminous miners and it is the writer's impression that this has increased during recent years. The duration of exposure is of great importance as it is apparent that men exposed to the dust hazard for a period of fifty years frequently exhibit gross fibrosis of the lungs on radiological examination and tubercle bacilli may be found in their sputum. It was in fact the finding of tubcle bacilli in the sputum of men over sixty that first drew the writers interest to this condition.

After practising for some years among coal miners one comes to the conclusion that after the fourth decade nearly

all of them, especially coal cutters are emphysematous. The reason which one would advance for this is that the peri-bronchial lymphatics become packed with dust so that the lymphatic circulation is interfered with and a foreign body reaction is set up. This causes a pulmonary fibrosis which is so often seen on radiological examination. The fibrosis itself by causing traction on the lung lobules distorts them and as a result the air is no longer thrown into the atrium as a jet. This jet of air as a result of the distortion no longer ventilates the lobule and as a result Emphysema occurs. This was the theory of Christie (Brit.Med.J. 1944).

It will be seen from the table summarising these cases at the end of the clinical section that these men have frequently suffered from Pleurisy, Bronchitis and atypical Pneumonia. This is due again to the lymphatics being packed with dust and so interfering with the lymphatic circulation that a dry pleura results and hence friction. It is a fact that at autopsy coal miners appear to have grossly thickened and adherent pleurae.

Then again the dust in the bronchioles stimulates the exudation of mucous to entangle the dust and remove it by means of the ciliated epithelium. There is a limit to this, however, and when infection occurs the ciliated epithelium is destroyed and replaced by columnar epithelium and the dust is then only removed when a cough reflex is initiated. This leads to stagnation in the bronchioles and then by the traction of the fibrous tissue and frequent coughing a condition of Bronchiectasis may become established. Thus

Bronchiectasis may be an occupational disease.

All these changes hitherto described viz:- Chronic Bronchitis, Pleurisy, Bronchiectasis and Pulmonary Fibrosis are ascribed to the dust which is inhaled in the course of their work as coal miners. This dust is of a mixed nature the analysis of which has already been mentioned and only contains about 1% Silica Dioxide. It does, however, in association with coal dust appear to produce chronic pulmonary disease in coal workers. In addition nitrous fumes appear to accelerate this process as has been proved by animal experiments, notably the Toronto experiments mentioned earlier in this thesis.

'Stonedusting' has been used extensively during recent years as a guard against explosions in coal mines - the stone dust being liberally sprinkled about prior to shot-firing. In addition the coal cutter is exposed to the dust from the coal cutting machines, especially if these are pneumatic machines. It can be of little surprise therefore to find that the old Miners Asthma is showing a tendency to return after an absence of some sixty years. Originally this condition would be caused by defective ventilation aided no doubt by coal dust, and now it may be caused by excessive dust from modern coal cutting machines plus the nitrous fumes from explosives. Three cases have come to my notice - all in young men, and all three have been associated with coal cutting machines - Cases 14, 15 and 34. On radiological examination there is little to find beyond evidence of Emphysema and Chronic Bronchitis. One rather thinks that

these cases are likely to increase in numbers.

Thus one would like to see added to modern textbooks on medicine the fact that Anthracosis, as so called, is not a harmless condition for a pure Anthracosis would be hard to find, but that the inhalation of coal dust with a certain proportion of stone dust plus exposure to nitrous fumes over a period of many years produces the variety of conditions which may be called a 'Coal Miners Lung'. This may be described as an Emphysematous lung with thickened pleura and pulmonary fibrosis. This type of pulmonary fibrosis has come to be called Pneumoconiosis of coal workers both from a clinical and radiological standpoint.

In addition there is no mention in modern textbooks of the condition now called Koniophthisis. This is a mixture of a dust reticulation and Tuberculous necrosis in association with a Silicotic Fibrosis. The tuberculous process is so modified that it may hardly be recognised. The Silica tends to augment the fibrotic response while the coal dust exerts some inhibitory effect on the tubercle. Thus it is that the Koniophthisis so produced tends to be a very chronic condition. The first two cases of these series are considered to be cases of Koniophthisis.

Then again there is the extraordinary phenomenon of the 'cricket ball masses' of which also there is no mention in the standard text books. Case 3 of these series illustrates such a case and his skiagram illustrates the appearance seen radiologically. A micro-photograph shows the appearance on microscopic section. There has been much discussion as to

whether they are Tuberculous or inflammatory in origin. In this case (Case 3) one can only say that Tubercle bacilli were finally isolated in the pus from the abscess which pointed externally. The diagnosis of this condition from a Bronchogenic Carcinoma is by no means easy as the patient exhibits all the characteristics of a malignant cachexia and the X-Ray gives little help in the differential diagnosis although Bronchoscopy may settle the matter.

'Finger-print markings' too are sometimes seen in these coal miners lungs. They too closely simulate secondary deposits of cancer in the lung but are actually due to coal dust in emphysematous bullae which have become compressed. They resemble the markings of fingerprints on the lung field when seen on X-Ray.

Although these fifty cases herein described are practically the only sufferers from chronic pulmonary disease that one has found in the course of several years, the remark of one patient "everyone on the coal cutters has a cough" has some significance when viewed in the light of modern attempts to step up the output of the coal mines. The application of modern machines and modern methods are dust provoking and new methods of counteracting the dust should be introduced.

From the fifty cases here described, some twnety-three were completely incapacitated - the remaining twenty-seven were partially incapacitated and by this one means that they had some light form of employment but even so, were frequently off work for prolonged periods.

Few cases of Silicosis proper are found in bituminous

coal miners and the few cases that do arise have been mainly employed as coal cutters.

One might ask why if all underground workers are exposed to the same dust hazard some develope dust reticulation, pulmonary fibrosis or other chronic pulmonary disability and others do not. The only answer that the writer would advance is that very frequently the worker afflicted by dust reticulation has a history of earlier attacks of Bronchitis, Influenza, Pleurisy, Pneumonia or chest injury and possibly these disorders devitalised his lung tissues to some extent. Possibly they caused damage to his dust excretory mechanism - the ciliated epithelium of the bronchi so that the dust was not eliminated efficiently and so a pulmonary fibrosis was initiated. When one looks at the cases summarised at the end of the clinical section it will be seen that no fewer than twenty-six men had suffered from one of the above complaints on one or more occasions. There has been much speculation as to whether naso-pharyngeal obstruction would predispose to pulmonary fibrosis or pneumoconiosis but such has not been the writer's experience and one must remember that men working hard in a dusty atmosphere would have their mouths open and be breathing through them from sheer exertion.

Coal cutters and stone workers seem to be the classes most afflicted with chronic pulmonary disease. From the list of cases summarised it will be seen that although some men had changed their occupation some seventeen had been coal cutters and fourteen had been stone workers, although

some had worked at both occupations in the course of their time underground.

Thus these two classes of workers amount to over thirty of the total of fifty cases described. In addition the term coal hewer is frequently applied to the men who work with the pneumatic pick which operates in much the same fashion as a coal cutter.

Three cases only had Tubercle bacilli in their sputum although two other cases were strongly suspected to be Tuberculous but no tubercle bacilli were ever isolated from their sputum.

The older the worker the more likely he was to have extensive pulmonary fibrosis or Koniophthisis although coal cutters in their thirties were very liable to show reticulation but the older men had doubtless not been exposed to the concentrations of dust that the young coal cutter now experiences. In the old days the coal was hewed by hand which is not such a dusty occupation and explosives were not used to the extent that they are to-day.

Only four of these cases were accepted by the Silicosis Board.

Electrocardiograph: The two cases of Koniophthisis (Cases 1 and 2) one case of dust reticulation(Case 4)and one case of Pulmonary Fibrosis(Case 16) were investigated with the Electrocardiograph. There were no definite cardiac abnormalities in any of them but one feature was noticed in three of them, namely, a high peak in the T waves in Lead II.

The only exception was Case 2. The writer merely carried out these electrocardiographs for curiosity as there were no physical signs of any cardiac abnormality. Originally only two were examined this way but as they both had a high peak in T_2 a further two were examined as controls - in Case 2 this high peak did not occur.

One would conclude this thesis by saying that all cases of Chronic Bronchitis, Emphysema, Bronchiectasis and even Chronic Fibroid Phthisis be viewed with suspicion in coal mining districts. In many cases an X-Ray will show up dust reticulation, pulmonary fibrosis or even Silicosis. The cases of Silicosis that are coming to light now are not all new cases. Many of them have been called Chronic Bronchitis for years and it is only when the practitioner becomes aware of this pitfall and has a good radiological examination carried out that the true facts come to light.

Much research remains to be done on the subject of chronic respiratory disease in coal miners and the Government have recently become aware of this fact and are now proposing to set up a Pneumoconiosis Bureau and a research centre for the investigation and further study of this condition, because as they state in their report on the rehabilitation of coal miners suffering from Pneumoconiosis -"not enough is known of this condition".

The Coal Trade is becoming ever more important as regards post war planning and with new methods of mining coming into vogue the dust hazard should not be forgotten. Coal miners should have frequent clinical and radiological examinations

carried out by industrial medical officers and on the first sign of cough, dyspnoea or pulmonary fibrosis they should be removed from the dusty places where they are employed and for a time at least, employed in the open air.

Better ventilation where the concentration of dust is heaviest would be helpful or alternatively extractor fans for use after shot-firing should be employed.

Shot-firing should only be done in the shift where the least number of men are working and then extractor fans could remove the dust and fumes before the men returned to the working place.

It is by such methods as these that improvement in the health of coal miners will be realised, and it is by means of such improvement in the working conditions that men will be attracted to the industry, for it is on the export of coal and its by-products that much of our post-war foreign trade will depend.

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Conclusions.

1. Fifty cases of Chronic Pulmonary disease in coal workers are described - twenty of them with X-Ray plates as illustrated.

2. Some degree of Emphysema is almost universal in coal miners over 45 years of age.

3. Pulmonary Fibrosis is an incapacitating disease due to the inhalation of mixed dust and yet little may be found on clinical examination.

4. This condition is found chiefly in coal cutters and stoneworkers.

5. Pleurisy and Pneumonia frequently precede the onset of Pulmonary Fibrosis and predispose to it.

6. Pneumoconiosis of coal workers (dust reticulation) may be very easily missed unless a good radiological examination is carried out.

7. Pulmonary Tuberculosis is not of frequent occurrence among coal miners - rather less in fact than among the rest of the population, but in the older age groups of coal miners, one finds cases of Koniophthisis.

8. Koniophthisis is a condition in which dust reticulation and Tuberculous necrosis are found in association with a Silicotic Fibrosis.

9. Occasionally one finds an extraordinary condition in which Pulmonary Tuberculosis and Silicotic fibrosis

Form a 'cricket ball mass' which is necrotic in the centre and contains a tarry fluid. This may even rupture externally.

10. Miners Asthma which has been defunct for about 60 years shows signs of returning and modern methods of mechanical mining are believed to be responsible for this.

11. Stone dust which is liberally scattered around the coal face before shot-firing, is not so innocuous in the writer's opinion as was formerly believed - although it only contains 1% Silica Dioxide.

12. There is no cardiac abnormality found on electroccardiographic examination in cases of chronic pulmonary disease of coal miners.

13. Frequent clinical and radiological examinations of coal miners should be carried out by industrial medical officers. Extractor fans, mist projectors etc should be used at the coal face to minimise the dust hazard and shot-firing should be done in the shift where the least number of men is working.

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