UNIVERSITY OF GLASGOW

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SILICOSIS AND TUBERCULOSIS IN SHEFFIELD METAL-GRINDERS.

(A Clinical - Statistical Study)

THESIS FOR THE DEGREE OF M.D.

presented by

ANDREW MEIKLEJOHN, M.B.,Ch.B. (with commendation) 1923.

First Assistant Medical Officer, Baguley Sanatorium, Corporation of Manchester.

lately Assistant Tuberculosis Officer, Corporation of Sheffield.

and Member-elect of Silicosis Board (Refractories and Sandstone Industries Scheme), Home Office.

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ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
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C O N T E N T S.

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INTRODUCTION.

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No branch of State Medicine has been more fruitful in practical application than that which deals with industrial diseases. Moreover, the prevention of these diseases is of paramount importance for, whereas sustained good health among the workers almost inevitably results in regular employment, improved nutrition and better social conditions, prolonged ill-health induces irregular employment, impaired nutrition and degraded social conditions for the worker and his dependents. Much has been achieved; there is yet much to do.

This is especially true of the diseases of the lungs associated with the inhalation of dust, to which Zenker gave the generic title "pneumonokoniosis".

Formerly it was believed that many kinds of dust, when inhaled or ingested, could give rise to pulmonary fibrosis, and as a result the disease was variously named anthracosis, siderosis, chalicosis or byssinosis. Modern research workers, notably Collis (1), however, have sought to establish the essential unity of these diseases by demonstrating that they are all due to the inhalation of free silica (SiO_2) in a finely divided state. The disease which results is silicosis.

In the lungs and air passages the silica dust causes diffuse fibrosis which varies in degree and extent with the amount of finely divided silica in the air breathed and the period of exposure. At the outset the fibrosis may give rise to no symptoms, but with its gradual advance the patient becomes increasingly breathless. This,

in itself, is not a grave disability, but such persons are very susceptible to acute infections of the lungs. Also, for some reason not yet wholly explained, the silicotic lung is exceedingly liable to invasion by the tubercle bacillus, and pulmonary tuberculosis is at once the most serious incapacitating and fatal complication of silicosis.

According to Collis and Greenwood (2), pulmonary tuberculosis implanted upon silicosis is correctly named tubercular silicosis.

Probably it was the wasting or phthisis of tubercular silicosis which impressed the people of the nineteenth century and so added to our language such phrases as potter's rot, grinder's rot, knapper's rot, and mason's phthisis.

Silicosis occurs extensively in many industries in this country and abroad. Its devastating effects have long been recognised among the gold miners in South Africa and Australia. In this country it is found among certain classes of pottery workers, metal-grinders, ganister miners and crushers and the makers of silica bricks - the so-called Refractories Industries, stone-masons, and certain groups of coal-miners. Its occurrence has recently been noted among girls employed in the making of abrasive soaps, and Cooke, McDonald and Oliver (3) have recorded pulmonary fibrosis among asbestos workers - "asbestosis". This, again, is probably a true silicosis, for asbestos is a magnesium silicate containing about 40% free silica.

It is interesting to note that the clue to the unity of the dust diseases of the lung was given by the small but interesting industry of flint-knapping which is carried on at Brandon in Suffolk. Flint is silica, and investigations of all the dusts in other

industries, in which dust-phthisis prevails, quickly disclosed that silica was the one constituent common to all the dusts.

During the two years 1926 - 1928, while engaged as an Assistant Tuberculosis Officer and Resident Medical Officer in charge of the city sanatorium at Sheffield, I had a very excellent opportunity of becoming thoroughly familiar with the diseases of the lungs as they occur in the Sheffield metal-grinders. My work at the tuberculosis dispensary brought me into intimate contact with these diseases in their earliest stages and, at the sanatorium, I was able to observe, over prolonged periods, cases of silicosis and tubercular silicosis, in all stages.

By frequent visits to the hulls, literally styes, as the grinders' workshops are called, I was able to study the actual grinding processes and the conditions under which they are carried on.

Increasing familiarity with these workers and their diseases led me to doubt the accuracy of many of the assertions which had hitherto been made regarding silicosis and tubercular silicosis. The following pages record the results of my observations and investigations into these diseases as they occur in metal-grinders in the Sheffield cutlery and allied industries.

As a preliminary, the clinical, radiological and pathological aspects were investigated by the intensive examination and prolonged observation of 37 consecutive cases of pulmonary disease in metal-grinders. These patients wereadmitted to the city sanatorium and remained under my care for periods varying from three to twleve months.

Meanwhile, study of the existing literature on dust diseases of the lungs revealed to me the fact that writers on the subject, even

at the present day, are almost unanimous in stating that tuberculosis, occurring as a complication of silicosis, is of an unusually rapid and fatal type. Collis (4), Nicholson (5), Thomson and Ford (6), and Heffernan (7), are only a few of those who advocate this opinion, while Beattie (8), so far as I can find, is the only author to question it. In none of their writings, however, have I been able to discover any data to substantiate their statement. My own experience did not seem to accord with the commonly accepted view, and so I decided to accumulate evidence which might throw some light on this aspect of the problem.

The medical records of 310 metal-grinders who died of pulmonary tuberculosis during the decennium 1919 - 1927 were carefully scrutinized and, as a control, the records of 1,361 other males who died of pulmonary tuberculosis during the same period.

From the data obtained, I submit that there are two distinct groups of cases of pulmonary tuberculosis among the metal-grinders. These I have named -

(a) natural group.

(b) industrial group.

The cases of true tubercular silicosis belong to the industrial group. It is a disease of late middle life, as originally indicated by Brownlee (9), and, in my opinion, is not more rapidly fatal in its course than tuberculosis in the average individual.

Greenwood in a private communication has argued that my statistics deal entirely with fatal cases and, accordingly, the results are not universally applicable to cases of tubercular

silicosis in metal-grinders.

To meet this criticism, the investigation was carried a further step.

The records of 71 cases of tuberculosis in metal-grinders notified during the years 1919, 1920 and 1921, and, as a control, 409 other males notified during the same period, were scrutinized. All these cases were traced up to the end of June 1928. The facts established in this examination of living cases are very similar to those determined in the series of fatal cases, and so I submit that my original conclusions are valid.

As a result of my observations, I suggest that tuberculosis still causes an excessive mortality among metal-grinders. This tuberculosis is divisible into two groups; a natural group in which the tuberculosis is not related to dust inhalation, and an industrial group - the cases of true tubercular silicosis.

This condition is a disease of late middle life and, contrary to the general opinion, is not more rapidly fatal in its course than is tuberculosis in the average individual.

From the Public Health point of view, the work seems to suggest that the problem of the grinding industry to-day is not silicosis but tuberculosis, and that our efforts to minimise the amount of dust are insignificant and futile in face of the larger and more pressing problem, viz., the elimination of tuberculosis from the grinding industry. The inclusion of metal-grinders under the Workmen's Compensation Act - The Metal Grinding Industries (Silicosis) Scheme, 1927. - is a step in the right direction. Crockett (10) and Philip (11), in recent speeches, have envisioned the day, 30

years hence, when tuberculosis in this country will be only a name. On the contrary, until the State by law eliminates the tuberculous worker from the grinding hulls, tuberculosis will continue to exact an excessive toll among the grinders and their colleagues, and these will form a constant reservoir of infection for the general community.

HISTORICAL.

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At what period in the history of mankind and in what countries dust diseases of the lungs first occurred is a matter on which we can only speculate. In the light of our present knowledge, however, it seems reasonable to suggest that these diseases musthave existed in prehistoric times in this country, when our primitive ancestors fashioned for themselves implements of flint.

Reference to the noxious effects of substances inhaled into the lungs is made in the works of some ancient classical writers. Hippocrates (460 - 357 B.C.) in his Epidemics (12) writes of the wan complexion and the difficulty in breathing of the metal diggers , and, in the early days of the Roman Empire, Pliny the Elder (23 - 79 A.D.) (13) records the use of respirators by polishers to prevent inhalation of red lead.

In the year 1557 A.D. was published De Re Metallica by Georgius Agricola, in which occurs the following interesting passage:

> "Other mines are very dry, and the constant dust enters the blood and lungs producing that difficulty of breathing which the Greeks call asthma. When the dust is corrosive it ulcerates the lungs and produces consumption; hence it is that in the Carpathian mountains there are women who have married seven husbands, all of whom this dreadful disease has brought to an early grave. At Altenburg in Meissen the men bind loose coverings (bladders) to their faces. By this means the dust is not carried into the lungs and blood, and does not hurt the eyes". (14)

Here for the first time wehave direct reference to the connexion between dust inhalation and asthma; how the so-called corrosive dusts produce consumption; the absence of similar

diseases among the wives of these workers.

The earliest medical treatise to deal with dust diseases of the lungs is the classical work of Ramazzini of Padua, "A Treatise of the Diseases of Tradesmen" translated into English in 1703. This shrewd physician personally made visits of inspection to the workshops to inform himself thoroughly of the trades about which he writes. In his work he records the prevalence of phthisisamong metal diggers, potters, workers in plaster of Paris, bakers and millers, starch makers, stone-cutters, masons, bricklayers and others (15) .

In 1713, Thomas Benson of Newcastle-under-Lyme, in seeking a patent for the grinding of flints used in the manufacture of white pots by a wet method, supports his claim by an interesting allusion to the baneful effects in the workers of this process, when carried out by the dry method (16).

The serious mortality from pulmonary disease, occurring among masons working with Craigleith sandstone, was noted by Alison in 1824, who drew attention to the now well-established fact that this variety of phthisis has its chief incidence in middle and advanced life (17).

In "My Schools and Schoolmasters" (1869), Hugh Miller, geologist, stonemason and author, has recorded for all time the risks of the mason's occupation and his own fortunate and timely escape (18).

P.236 Chapt. 11.

> My general health, too, had become far from strong. As I had been almost entirely engaged in hewing for the two previous seasons, the dust of the stone, inhaled at [redacted]y

breath, had exerted the usual, weakening effects on the lungs - those effects under which the life of the stone-cutter is restricted to about forty-five years; but it was only now, when working day after day with wet feet in a water-logged ditch, that I began to be sensibly informed, by a dull depressing pain in the chest, and a blood-stained mucoidal substance, expectorated with difficulty, that I had already caught harm from my employment, and that my term of life might fall short of the average one.

P.357 Chapt. 16.

The dust of the stone which I had been hewing for the last two years had begun to affect my lungs, as they had been affected in the last autumn of my apprenticeship, but much more severely; and I was too palpably sinking in flesh and strength to render it safe for me to encounter the consequences of another season of hard work as a stone-cutter. From the stage of the malady at which I had already arrived, poor workmen, unable to do what I did, throw themselves loose from their employment, and sink in six or eight months into the grave - some at an earlier, some at a later period of life; but so general is the affection, that few of our Edinburgh stone-cutters pass their fortieth year unscathed, and not one out of every fifty of their number ever reaches his forty-fifth year."

Thackrah of Leeds in his book "The Effects of Arts, Trades and Professions on Health and Longevity" - published 1832 - is the first author to mention specifically the occurrence of dust diseases among the Sheffield metal-grinders, and records the opinion of Knight that fork grinding ought to be confined to criminals (19).

This appalling scourge among the fork grinders had not escaped the attention of Sheffield's own physicians, industrialists, or the workers themselves. In his book "Diseases of Grinders" (1841), Calvert Holland started his crusade on their behalf and, by his clear statistical studies, demonstrated the terrible mortality from lung diseases incident to dry grinding (20 and 21). Under his leadership, a vigorous campaign for reform was initiated and later carried on by J.C. Hall whose pamphlet "On the Preventi[illegible]d

Treatment of the Sheffield Grinders' Disease" (1857) is full of shrewd observations and suggestions for improvements in the industry. Emphasis is laid by him on such aggravating causes of the disease as alcoholism, insanitary home and working conditions, and early marriages. Furthermore the following passage indicates the extreme youth of those then engaged in the grinding industry :

> "Boys are apprenticed to the lighter branches at from nine to thirteen years of age, and to the heavier branches of grinding at from twelve to fourteen. Some go to the trade even younger than this. In my recent visit to the wheels I met with one boy only eleven years old; he had been in the hull since the age of eight; he had a fearful cough, and, on examination, I found the upper portion of both lungs extensively diseased". (22).

Up to this time, all the references are to the clinical appearances of the disease associated with dust inhalation, and so it is interesting to note that Hall writes -

> "There is no necessary connexion between the Sheffield grinders' disease and thoracic consumption, although both affections may be present in the same individual." (22).

Here, for the first time, we have the definite assertion that the phthisis of dust disease may exist apart from thoracic consumption and that both conditions may be present simultaneously in the same individual. Scientific proof of this was not possible until the discovery of the tubercle bacillus by Koch in 1882.

This resulted in increased concentration on the pathological aspects of dust disease. Soon it became clear that, while dust in itself could lead to wasting and death, in a great many cases the lungs had been secondarily invaded by the tubercle bacillus.

As to how the dust predisposed to this complication was not wholly understood. Mechanical irritation of the lungs and air passages by the hard, gritty, irregular particles was offered as an explanation and widely accepted. Even so great an authority as Beattie proclaimed his adherence to this hypothesis so recently as 1916. (8).

The recent researches of Gye and Kettle (23, 24, 25) and of Heffernan (26) have rather displaced this hypothesis of mechanical causation and indicated that the action is of a complex chemical nature.

For long all kinds of dust were considered equally noxious, but careful observations by Collis (1) and others have now more or less established the fact that silica is the sole offending agent and that dust diseases of the lungs - "pneumonokoniosis" of Zenker - are synonymous with silicosis.

ETIOLOGY AND PATHOLOGY OF PNEUMONOKONIOSIS.

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The salient features of the etiology and pathology of the dust diseases of the lungs are best studied under the following headings :-

1. The nature of the dust.
2. The entrance of the dust into the body.
3. The effects of the dust in the lungs.
4. The mode of action of the dust.
5. Complications.

The Nature of the Dust.

Broadly speaking, dusts may be classified into organic and inorganic. Inhalation of any of these in excessive quantity is undoubtedly harmful but only a few are definitely injurious to health, e.g.,the poisonous effects due to inhalation of lead and arsenic.

Dust diseases of the lungs have long been recognized but it is only within recent times that the noxious agent has been defined. For years many inorganic forms of dust, coal, iron, cement, plaster of Paris, sandstone, horn, bone and emery were considered agents in the causation of lung diseases among workers, but the clinical studies of Collis (4) and the experimental animal researches of Beattie (27) in this country and Mavrogordato (28 and 29) in South Africa, have now established the fact that the true provocative substance is silica. Purdy (30) in a recent review of 103 contributions

on the subject shows that this opinion is the one most widely favoured at the present time.

All forms of silica, however, are not equally harmful. According to Collis (4) the element silicon is not in itself the important factor, for it occurs in cement and clay which are comparatively innocuous. The danger only really comes when the dust is in the form of free crystalline silica (SiO_2). As such, it is found abundantly in nature in the forms of quartz and flint. It is the chief component of granite, gneiss, ganister, gritstone and buhrstone. It is insoluble in water and in all acids except hydrofluoric, but it is soluble in alkalis and the alkaline body fluids - a fact which has been emphasized by Gye and Kettle (23 and 31) in their experimental work on the subject.

The Entrance of the Dust into the Body.

The dust reaches the lungs via the air passages in the inspired air. Other paths of entry to the lungs (e.g., via the intestinal tract) have been suggested, but the work of Findlay (32) has shown that this is not the case, and Watt, Irvine, Johnson and Steuart (33) state that silicosis is more common in mouth breathers than in nose breathers.

McCrae (34) was the first to demonstrate satisfactorily that only particles of very minute size are dangerous. By his investigations he shewed that the largest particles which gain access and become embedded in the lung proper have a maximum diameter of 10 microns and 70 % of all particles

are less than 1 micron in diameter.

The first defence of the body to be penetrated by the dust is the nasal vibrissae. These arrest some of the grosser particles and others are caught in the nasal mucus. According to Collis (4) and Middleton (35) these protective vibrissae, in patients, are usually stunted or absent, which observation I have been unable to confirm in my clinical studies among metal-grinders.

Progress of the dust along the air passages is resisted by the ciliated epithelium of their lining mucous membrane, and a good deal of the dust is expectorated in the mucoid secretions of these passages.

Effects of the Dust in the Lungs.

The fine dust which reaches the lung alveoli sets up a catarrhal process with proliferation of certain cells. Some of these cells are phagocytic - macrophages - and ingest the invading particles of silica. These are the so-called "dust cells". Mavrogordato (29), who has studied these cells carefully, believes they are of endothelial origin and, according to Pancoast and Pendergrass (36), evidence submitted by other observers would seem to favour this view. A large number of the "dust-cells" are expelled through the air-passages, the remainder collect in the alveoli and gain admission to the adjacent lymphatic vessels. From there, according to Mavrogordato (29), they show a distinct "pleural drift" and flock together to form pseudo-tubercles in and immediately

below the visceral pleura. Some of the "dust-cells" pass along the lymph channels toward the tracheo-bronchial glands. If the dust inhalation continues, the lymph channels become blocked and fibrosis of the lung results. Mavrogordato (29) is of the opinion that the dust cell becomes converted to a fibroblast while Gye and Purdy (24), on the other hand, consider that the silica acts as a direct cell poison which stimulates the connective tissue and results in its proliferation.

As the dust cells die, the particles of silica are liberated and aggregate in the fibrous tissue. In this way, areas of nodular fibrosis result. Meanwhile the tracheo-bronchial lymph-nodes enlarge and become fibrotic. Fibrosis increases along the line of the peribronchial lymphatics, in the interlobular zone and between the alveoli.

As the lymph flow at the hilum becomes increasingly blocked, there is a tendency for a back flow toward the sub-pleural lymphatics, and this results in fibrous nodules therein, thickened pleura and adhesions (Pancoast and Pendergrass (36)). Such adhesions between visceral and parietal pleura occur on the diaphragmatic surface of the lung and, as a result, the movements of the diaphragm greatly restricted. Increasing interstitial fibrosis causes collapse of numerous alveoli, while those in the less affected areas become distinctly emphysematous. In the same way, many of the minute blood vessels of the lung are obliterated. The visceral pleura becomes increasingly thickened and universally adherent to diaphragm and chest wall

and, in not a few cases, to the pericardium. Carbon particles associated with the fine silica result in marked irregular black mottling of the pleura, tracheo-bronchial glands and lung parenchyma itself. By coalescence of the fibrous nodules large irregular areas of dense fibrous consolidation occur and these, by interfering with the local circulation, may cause central necrosis and cavitation without the intervention of the tubercle bacillus (Collis (4)). The continued deposition of silica, with consequent fibrous tissue proliferation, renders the lung rigid and inelastic, and contraction of the fibrous strands leads to dilatation of bronchi and bronchioles and, in some instances, to actual bronchiectasis.

Undoubtedly, during the progress of this fibrosis, the lung is able to eliminate a certain amount of silica. That coal dust assists in this excretion is suggested by Mavrogordato (29) who states that -

> "Coal dust produces a catarrhal reaction and this reaction is helpful in eliminating inert silica dust along with coal dust."

Excessive mortality from Bright's disease among silica workers suggests that this dust or its derivatives, to some extent, may be eliminated by the kidneys and thereby cause inflammatory mischief in these organs (Collis (1)).

The Mode of Action of the Dust.

As to the actual method, by which the silica produces its deleterious action on the lung, there is as yet no unanimity.

Three explanations have been offered, viz.,

that the action is -

(1) mechanical.

(2) chemical.

(3) physico-chemical.

Early observers noted that the minute particles of dust were very hard and irregular, and this seemed to offer a very obvious explanation of the irritative action in the lungs. So recently as 1916 Beattie (8) wrote -

"My view still is that the size and shape of the particles is the important factor".

Gye and Kettle (23 and 31), however, questioned the validity of this view, pointing out -

(1) "that particles lose their sharpness when embedded in the phagocytic dust cells".
(2) "that the reaction (fibrosis) is noted at points remote from the presence of silica".

As a result of their experimental work on animals, these observers have shown that finely divided silica in the lungs slowly forms a silica sol, either directly or through the intermediate formation of sodium silicate, which is decomposed by carbonic acid, the sol formed acting as a cell poison (23 and 31).

More recently, Heffernan and Green (26) have questioned the above view that the silica acts as a direct cell poison. As a result of their observations, they suggest that -

"The harmful effects of silica appear to be due to its powerful potentialities as a colloid, rather than to any toxicity of the silica ion".

And further that,

"The activity of colloidal silica in the lungs may be

prevented by protecting the silica dust with a coating of a substance which is known to coagulate colloidal silica. Such substances are clay substance, carbon, shale dust, etc.".

A further explanation is that of Gardner (37) who considers that the lung lesion is due to a combined irritant action of the tubercle bacillus and the silica, which action neither of them alone can produce.

At the moment, the hypothesis of chemical action is the one most generally accepted.

Complications.

The silicotic lung, as shown by statistics (38), is exceedingly liable to invasion by pathogenic organisms (e.g.) pneumococcus, B.Pfeiffer, streptococci, etc. In some way not yet wholly understood, it forms a soil peculiarly favourable to the growth and multiplication of the tubercle bacillus and pulmonary tuberculosis is an almost invariable complication of advanced silicosis (8), and the most frequent terminal condition. Gye and Kettle (23 and 31) are of the opinion that this is due to the action of the silica, which causes a coagulation necrosis in which area the tubercle bacilli are protected from phagocytosis and so can multiply rapidly with impunity; and further to the disorganisation of the lymphatic drainage of the lung.

It has also been recorded that, when tuberculosis supervenes, it accelerates the development of the silicosis (39). Thus we have a vicious circle, the silica predisposing to implantation of the tubercle bacilli and these, in turn,

promoting further development of the silicosis.

Other respiratory diseases show an excessive incidence and mortality among the silica workers, (e.g.) pneumonia, pleurisy, asthma, bronchitis and emphysema. Now that increased attention is being paid to malignant disease of the lung, it will be interesting to note whether or not these workers suffer inordinately from this condition. If irritation, physical or chemical, plays a part in the etiology of new growths, then it is reasonable to expect such lesions to occur in the lungs of the silica worker. In a series of 15 cases of malignant disease of the lung which have come under my personal notice during 1927 and 1928, two at least have occurred in workers suffering from silicosis - both confirmed post-mortem - macro- and microscopically :

(1) primary carcinoma of right bronchus.

(2) mediastinal lympho-sarcoma infiltrating right lung.

An excellent technical description of metal-grinding in this country and abroad is given by Macklin in H.M. Report on The Grinding of Metals and Cleaning of Castings (40). The work is performed in workshops, called hulls or wheels. In many instances, these are dark and insanitary, often underground, where direct sunlight cannot penetrate. The grinding is done on gritstone or sandstone wheels of varying sizes, revolving in troughs, which may or may not contain water. Where no water is used the process is called dry-grinding (e.g.) fork and razor grinding; where water is used - wet-grinding (e.g.)

table-blade and pen-knife grinding. As the wheel revolves rapidly, the friction of the blade against it results in a fine spray of metal dust from the blade, silica from the stone and in wet-grinding, water from the wet surface of the stone and from the trough. A visit to a hull will readily convince one of the tremendous amount of dust produced. It is caked on the floor, walls, window frames and machinery. It adheres to the hair, skin and clothing of the workers. The windows are never glazed as the glass would immediately become obscured by the dust and so prevent the entrance of any light whatsoever. The volume of dust is considerably increased in the repeated preparation of the grinding surface of the stone by "hacking" and "redding" and more especially by "racing", which must be done dry. Some idea of the rapid attrition of a stone is conveyed by the fact that a saw grinder will wear down a stone 6 ft. in diameter by 10 inches thick to 2 ft. by 10 inches, in about eight weeks.

During recent years, "fannies" have been introduced as hoods to extract the dust. Composition wheels of carborundum bonded with silica have also been used extensively, but, for certain classes of work, sandstone is still indispensable.

In many of the heavier branches of the trade (e.g.) butcher blade and sheep shear grinding, the work is arduous. The omnipresent dust irritates the throat and the grinder spits into the trough beside him. As he cools the hot steel after grinding, he and his fellows dip their fingers into the sputum infected water. Then, while eating or smoking, these unwashed hands carry infection

direct to the mouth. The fast revolving wheel sprays the water as a fine cloud into the already vitiated atmosphere.

Silica unquestionably plays a vital part in promoting invasion of the lungs by the tubercle bacilli, but there are numerous contributory causes. Of these, the following deserve mention :

1. Alcoholism, Insanitary Housing Conditions and Early Marriages.

These were indicted by Hall in 1857 (22), but to-day they play a very insignificant part.

2. Child Labour.

Previous to the industrial legislation of the latter half of the nineteenth century, boys under 12 years were employed in these trades (Hall (22) vide quotation p. 10), and, as is well known, even to-day, pulmonary tuberculosis in children and adolescents is usually of a very acute and fulminating type. Nowadays apprenticeship to these trades seldom commences before the age of 16 years, and this factor alone may help to explain the great improvements in duration of life which have been recorded among these workers (Oliver (41)).

3. Insanitary Hulls.

Indubitably, this is a predisposing factor of the most vital importance. The great majority of the hulls are dark, damp and insanitary. Direct sunlight is excluded, and so the tubercle bacillus is allowed to flourish outside the body, protected by the dampness, absence of sunlight and the favourable temperature generated by the steam engines which drive the wheels. Year after year, Dr. F.E. Wynne (42)

proclaims, unheeded, that improvement in health conditions of the grinders is retarded by the retention of the antiquated system of "little maisters" who own these hulls and who are financially unable to erect factories of modern sanitary construction.

4. A Hereditary Occupation.

The natural deposits of gritstone used for makingthe wheels occur in the neighbourhood of Sheffield, and this has resulted in the concentration of the cutlery grinding industry in this centre. For centuries it has provided employment for a large number of the local inhabitants and long generations of families can be traced in the annals of the industry. A father already suffering from silicosis and perhaps tuberculosis takes his son to work with him. In the hull, during working hours, the lad is exposed to the dust and not infrequently to tubercle bacilli, and at other times, in his own home, in many instances, to tuberculous infection from his father or elder brothers.

In my study I have noted that the pathological picture described above is still to be observed among the metal-grinders but the lesions are seldom so gross or massive as those seen in ganister crushers or ganister miners - in one of whom I have seen the upper lobe of the right lung almost converted to a stony mass.

The following abbreviated post-mortem report is typical of many which I have seen and performed :

D.F. aged 59 years. Joiner's Tools' Grinder for 47 years. Body extremely emaciated. On opening the thorax the lungs did not retract or collapse.

Lungs. Adherent to the chest wall, diaphragm and pericardium; visceral pleura irregularly thickened and all surfaces covered with black mottling. Gritty nodules size of split-pea palpable just under pleura and throughout lung parenchyma; fairly uniformly distributed and many surrounded by areas of fibrosis from size of pea to size of walnut.

On section, knife grated and texture of lung was unusually tough; on cut surface fibrous masses and gritty particles stood out prominently; bronchioles and bronchi remained widely patent.

Chronic tuberculous cavity (tubercle bacilli present in pus) 1½ inches in diameter in right upper lobe; disseminated foci of caseous tuberculosis throughout both lungs. Hilum glands enlarged - size of brazil nut - very black and showing fibro-caseous areas on section.

Heart. Enlarged; muscle hypertrophy, especially right ventricle; fatty infiltration of muscle; small areas of atheroma immediately above aortic cusps; valves - no abnormality made out; slight sclerosis of coronary arteries.

Liver, spleen and kidneys congested - otherwise no abnormality.

Great omentum matted into a solid mass; enlarged caseous mesenteric glands; tuberculous ulcers in ileum.

Summary of pathological diagnosis :

Silicosis of both lungs; old tuberculous excavation in right upper lobe with recent acute tuberculous dissemination throughout both lungs; tuberculous enteritis and peritonitis.

Pathologist's Report (University of Sheffield).

Lung. Sections shew extensive silicosis and tuberculosis. The latter is apparently acute as the lesion is chiefly caseation with but little cellular reaction. (photomicrograph No 3)

Bronchial gland. Shews caseation.

Piece of Mesentery. Extensive tuberculous disease with early caseation.

PHOTOMICROGRAPHS ILLUSTRATING SILICOSIS AND TUBERCULOSIS

IN SHEFFIELD METAL-GRINDERS.

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1. Section of bronchial gland showing fibrosis (silicosis).

2. Section of lung showing fibrosis (simple silicosis).

3. Section of lung showing fibrosis (silicosis) and acute tuberculosis, very little cellular reaction.

4. Section of lung showing fibrosis (silicosis) and tuberculosis with caseation.

Note : Actual silica particles can only be demonstrated microscopically by use of polarised light.

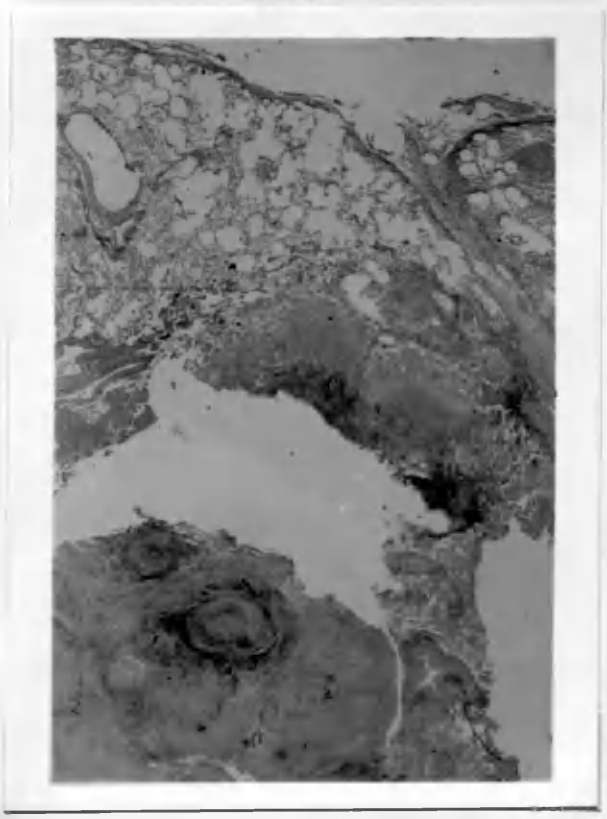
vide. Collis, E.L. "Industrial Pneumonoconioses, with special reference to Dust Phthisis". Milroy Lectures 1915, p.32.

H.M. Stationery Office, London, 1919.

No. 1

No. 2

No. 3



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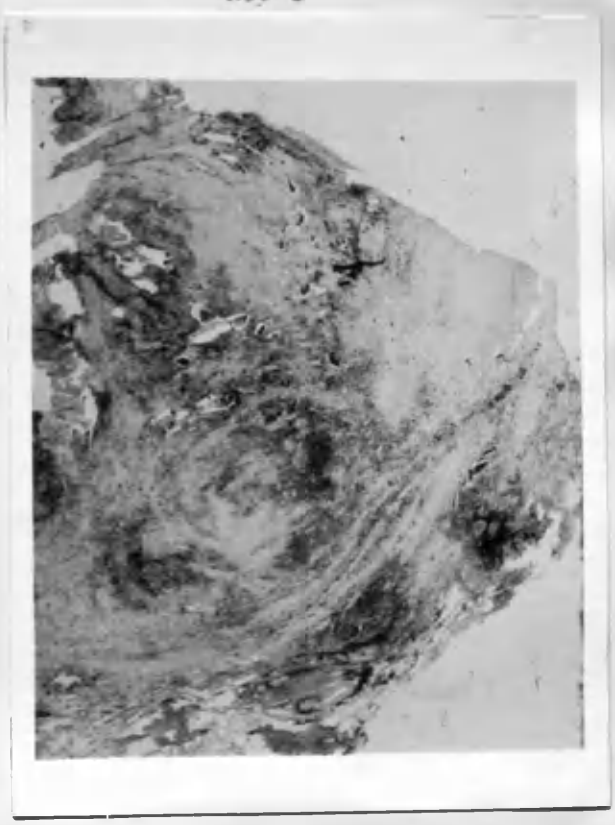

THE RELATION OF SILICOSIS TO PULMONARY TUBERCULOSIS IN SHEFFIELD METAL-GRINDERS.

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Mortality from Pulmonary Tuberculosis among Metal-Grinders.

In the preceding chapters, reference has been made to the unduly high incidence and mortality from pulmonary tuberculosis among workers exposed to silica dust. To this class belong the Sheffield metal-grinders, and the following figures have been extracted from the Annual Reports of the Medical Officer of Health for Sheffield.

Table 1.

	Mortality from Tuberculosis.	Rate per 1,000.	
	Grinders.	All persons over 15 years.	Ratio.
1923	6.9	1.2	5.75 : 1
1924	7.2	1.1	6.55 : 1
1925	6.3	1.1	5.73 : 1
1926	5.7	1.0	5.70 : 1
1927	7.8	1.0	7.80 : 1

This table clearly demonstrates that, at the present time, the mortality rate from pulmonary tuberculosis in metal-grinders is approximately six times greater than that for all persons over 15 years of age in the same city. The great majority of these persons are living under very similar housing and economic conditions to the grinders.

Table 2, which has been specially prepared, by the statistical department of the Medical Officer of Health of

Sheffield, shows the total number of deaths from all causes including pulmonary tuberculosis, and deaths from pulmonary tuberculosis alone, occurring among Sheffield metal-grinders for the years 1886 - 1925, arranged in periods of 5 years.

Table 2.

City of Sheffield.

Mortality among Grinders.

Quinquennia.

	1886-1890	1891-1895	1896-1900	1901-1905	1906-1910	1911-1915	1916-1920	1921-1925
All causes - Total Deaths	536	548	529	622	573	607	581	515
Pulmonary Tuberculosis - Total Deaths	191	207	199	291	295	322	224	201
Percentage of Total Deaths due to Pulmonary Tuberculosis	35.6	37.8	37.6	46.8	51.5	53.5	38.6	39.0

The predominance of pulmonary tuberculosis, as a cause of death in these workers, is once more apparent, for, whereas it accounts for 35% to 50% of total deaths in grinders, it accounts for only 10% to 20% (42) in all males over 15 years of age in Sheffield.

It is noteworthy that there has been practically no improvement in mortality from pulmonary tuberculosis among grinders during the past 40 years. In 1886 - 1890 pulmonary tuberculosis accounted for 35.6% of the total deaths; in 1921-1925 for 39%; this, despite the fact that the mortality from pulmonary tuberculosis in Sheffield steadily declined from 1.36 per 1,000 in 1896 to 0.745 in 1926, and the general death rate from 19.1 in 1896 to 11.3 in 1926.

Brownlee (43) has asserted that, in his experience, "The diagnosis of death from phthisis (is) among the most trustworthy of the diagnoses recorded by the Registrar General". Accordingly, the following conclusions from the foregoing data seem justified :

1. The mortality rate from pulmonary tuberculosis in metal-grinders is still greatly in excess of that in the general population.

2. An inordinately high proportion of deaths among metal-grinders is due to pulmonary tuberculosis, and no notable improvement in this respect has occurred in the past 40 years.

Does pulmonary tuberculosis in the metal-grinder run a more rapidly fatal course than in the average individual ?

The opinion of most writers on the subject, even at present, is that the tuberculosis which complicates silicosis is unusually rapid and fatal in its course. This is well illustrated by the following quotations :-

> "Personally I consider that the silicosis which precedes and predisposes to tuberculosis is slow in developing, but that tuberculosis implanted on silicosis is rapid in its course and unusually fatal in its termination".
>
> E.L. Collis (4)

> "The low infectivity of cases of pulmonary fibrosis is probably due to (a) the fact that many cases are never tuberculous at all, and (b) to the rapid termination of open infectious cases, leaving but a short period for infecting contacts."
>
> B.S. Nicholson (5)

> "When once tuberculosis supervenes in silica workers wasting and pyrexia are marked, and the disease progresses rapidly to a fatal termination."
>
> Hyslop Thomson and A.P. Ford (6)

"It (silicosis) is characterised, clinically, by a steadily increasing dyspnoea, and by a tendency to develop and succumb rapidly to pulmonary tuberculosis, in late middle life".

P. Heffernan (7)

Beattie, who is specially qualified to speak authoritatively of the condition as met in Sheffield metal-grinders, does not share the preceding opinions as shewn by the following :

"None, who has studied the pathological processes in the lung in cases of tuberculosis, can be anything but sceptical when he is told that fibrosis encourages the spread of the tuberculous process. Tubercle is healed by the production of fibrous tissue.. ..... The facts all seem to point to the suggestion I have made that tuberculosis, when combined with dust-fibrosis, is a slower process than tuberculosis alone, and certainly that the fibrosis does not in itself tend to shorten life."

J. M. Beattie (8)

The true state of our present knowledge of this aspect of tubercular silicosis is probably most accurately represented here:-

"With the onset of tubercular infection in the lungs, the latent silicosis becomes active. In other words, the tubercular infection appears to unmask the pre-existing, but more or less latent, silicosis. Exactly how the two conditions react upon each other is unknown."

(39)

In none of the contributions cited above are any statistics given in support of the opinion expressed. The problem seemed worthy of investigation, especially as my personal experience at the sanatorium rather inclined me to doubt the majority opinion. Clinically, pulmonary tuberculosis complicating silicosis in the grinder appeared to run its course in much the same way as in the average male person and at no more rapid rate.

Briefly the problem is :- "Does pulmonary tuberculosis in the metal-grinder run a more rapidly fatal course than in the average individual ?"

At the onset of the inquiry it was essential to formulate clearly certain definitions and to make these as rigid and unequivocal as possible :-

DEFINITIONS.

Metal-Grinder.

A man employed, during the whole of his working life, in the grinding of metals (cutlery and allied industries) in Sheffield.

Pulmonary Tuberculosis.

The diagnosis of pulmonary tuberculosis was held to be established on the first date on which tubercle bacilli were discovered in the sputum.

Fatality Period.

Period of time between first date of detection of tubercle bacilli in the sputum and date of death.

Morbidity Period.

Fatality period, plus the alleged period of the illness, which immediately preceded the discovery of tubercle bacilli in the sputum.

A definite standard for comparison had to be fixed; in other words, the average individual. For this purpose, statistics for the whole country as issued by the Ministry of Health, for various reasons, did not seem a just standard or yet those of the Medical Officer of Health of Sheffield. The most accurate control appeared to be all males employed in occupations other than grinding , and in which there was no silica risk, in the City of Sheffield during the period under consideration.

Average Individuals - referred to as Controls.

All males, aged 15 years andover, employed in occupations

other than the grinding of metals, not exposed to the silica risk, and resident in the City of Sheffield.

All the necessary data - (1) Name, (2) Age, (3) Occupation, (4) Duration of occupation, (5) Date of first positive sputum (T.B. +), (6) Alleged duration of illness, (7) Date of Death, (8) Cause of Death, could beobtained from the accurate records filed at the Tuberculosis Dispensary for the City of Sheffield. These records are very detailed and a record exists of all men certified as having died of pulmonary tuberculosis, whether the man has attended the dispensary at any time, or not.

All sputum examinations for tubercle bacilli for the city are carried out at the dispensary laboratory, and result and date carefully recorded.

No positive case (i.e.) a case in whose sputum tubercle bacilli have been found, has ever been discharged from supervision by the tuberculosis staff, and each week the Returns of theRegistrar of Births and Deaths are scrutinized to note the death, from any cause, of all patients belonging to the department.

The records of all males certified as having died of pulmonary tuberculosis during the ten years' period 1918-1927, both inclusive, were investigated, and, as a result, data were obtained of 310 GRINDERS, and, for comparison, 1,361 CONTROLS. These figures may appear small, but it must be remembered that they represent practically all the male deaths in the City of Sheffield (population 524,000) from pulmonary tuberculosis of

positive cases (T.B.+) for that period of ten years which is commensurate with the period of organised public health treatment of pulmonary tuberculosis and, in addition, a very considerable fraction of the time during which organised public facilities for the routine examination of sputum for tubercle bacilli have been available. Care was taken to eliminate all cases, few in number, in which the tuberculosis was not allowed to run its course to a fatal issue, e.g., by fatal accident, suicide, or acute intercurrent disease.

This part of the investigation provided information on a large group of fatal cases. The next problem was to obtain, if possible, similar details of a group of living cases. For this purpose, the records of the years 1919, 1920 and 1921 were searched and a list made of all male cases notified in each of these years. From this, a second list was prepared of the cases in whose sputum tubercle bacilli were found for the first time, in each of these years. All these patients were then followed up to the end of June, 1928. Data were thus assembled of 71 GRINDERS and 409 CONTROLS; 25 others who had left the city could not be traced for the full period and so were omitted from the analysis.

Is pulmonary tuberculosis a fatal disease ?

For the purpose of this study pulmonary tuberculosis has been restricted to those cases in whose sputum tubercle bacilli have been detected on at least one occasion.

Investigations into the after-histories of similar cases have been made in various sanatoria in this country and abroad, notably at the Brompton Hospital Sanatorium, Frimley (44), and at the Adirondack Sanatorium, U.S.A. (45).

From these, it would appear that theoutlook is quite hopeful and according to the Brompton figures, as many as 44.5% of the patients are alive and able to do full work four years after discharge from the hospital. But in these reports, two points call for comment :

1. The patients admitted are selected as likely to benefit by treatment. Those cases in which the immediate prognosis is grave are relegated to tuberculosis hospitals for advanced cases.

2. The after-histories are of cases discharged and the percentage calculations omit those cases who died in the sanatorium.

As a result, such investigations display the outlook in a particularly favourable light but are useless for application to the disease as it affects a community or a nation.

Nevertheless, there are still many who believe that a large percentage of cases of open pulmonary tuberculosis recovers, ultimately to die of some other disease. What then is the state of affairs regarding the fatality of pulmonary tuberculosis in an industrial community?

The following facts represent the case as it occurs in

Sheffield. This is a city of a population of 524,000, a fairly typical English industrial city. Dr. F.E. Wynne, writing of pulmonary tuberculosis in his Annual,Report of the City of Sheffield in 1927, states :

> "It will be seen that in 1927 Sheffield's death rate from Tuberculosis was less than that of any other town, very considerably less than most of them, and actually less than the death rate for England and Wales.
> The percentage reduction in Sheffield's death rate from Respiratory Tuberculosis during the past 10 years is also the highest of any town, and much higher than that for England and Wales". (46)

This highly commendable state of affairs ought if anything to brighten the prospect - at any rate it should not darken it.

The first part of the investigation deals with notified cases of pulmonary tuberculosis in whose sputum tubercle bacilli were reported for the first time in the years 1919, 1920 and 1921. The only selection made is, that the cases for study have been restricted to all males aged 15 years and over. The after-histories have been followed up to the end of June, 1928, that is an average period of eight years.

Table 3.

		1919	1920	1921	Total	Percentage
Died of Pulmonary Tuberculosis	Controls	98	115	144	357	87.3
do.	Grinders	22	13	34	69	97.0
Alive at 30/6/28	Controls	16	14	15	45	11.0
do.	Grinders	1	1	-	2	3.0
Died other causes	Controls	2	4	1	7	1.7
do.	Grinders	-	-	-	-	-
Left City		9	11	5	25	-
		148	158	199	505	

From the above table, it is seen that I was able to trace 480 of the 505 cases. Of the 409 controls 87.3% have died of pulmonary tuberculosis, 11% are still alive, and 1.7% died of other causes. Of the 71 grinders, 97% have died of pulmonary tuberculosis and 3% are still alive.

The following is a list of the seven cases who died of other causes and the fatality period in each :-

	G.H.B.	Syphilitic stricture of the oesophagus	1 week.
X	H.T.	Tabes mesenterica	40 weeks
	A.L.	Epithelioma of palate	168 weeks
X	G.N.T.	Bronchitis and Heart Failure	196 weeks
X	J.A.W.	Chronic bronchitis and syncope	53 weeks
	T.B.	Sarcoma of chest wall	9 weeks
X	W.R.	Bronchitis	23 weeks

In the cases marked (X) death was probably due to pulmonary tuberculosis although the practitioner in attendance did not certify so.

Study of the 45 cases still alive reveals the fact, that 23 are able to do full work and 3 part-time; the remaining 19 are wholly unfit for work and almost continually in sanatorium.

Further, it is interesting to note, that of the 23 able to do full work, the great majority belong to sheltered occupations, 4 have never received any form of institutional treatment, and 15 less than six months.

At the beginning of the year 1923 a register of all newly positive cases (T.B. +), occurring in the City of Sheffield,

was instituted. The annexed table sets forth in detail the number of cases recorded and the number of these alive at June 30th, 1928. :

Table 4.

	Number of new T.B. + cases	Alive at 30-6-28	Percentage alive	Percentage dead
1923	335	62	18.5	81.5
1924	340	87	25.6	74.4
1925	355	107	30.1	69.9
1926	364	155	42.6	57.4
1927	338	195	57.7	42.3

The figures in Tables 3 and 4 require no further elaboration. To my mind there is only one possible interpretation, viz., that open pulmonary tuberculosis (T.B. +) as seen in the City of Sheffield - a typical industrial community - is almost invariably fatal and, in the great majority of cases within a few years of the discovery of tubercle bacilli in the sputum for the first time. This occurs, irrespective of occupation, and in spite of the efforts of a highly skilled and efficient department for the prevention and treatment of the disease. This accords with the opinion of Brownlee (43) who states, "It would thus seem that phthisis is a disease which once established leads more or less uniformly to death".

Conclusions.

1. Open pulmonary tuberculosis almost invariably proceeds to a fatal termination within a few years of its onset.

2. Only a very small minority of patients recovers to die later of some other disease.

Fatality and Morbidity Periods (vide definitions p. 30).

Having now established the fact that open pulmonary tuberculosis, in the great majority of cases, progresses to a fatal issue, we are in a position toconsider the average duration of life after the original discovery of tubercle bacilli in the sputum. Moreover, this fact justifies us in basing this further part of the enquiry on a series of fatal cases. These, as already indicated, consist of all male deaths from open pulmonary tuberculosis occurring in Sheffield during the ten years 1918 - 1927, viz., 310 grinders and 1,361 controls.

Table 5 shows the average Fatality and Morbidity periods for Grinders and Controls for each of the years under consideration and for the complete series of cases.

Table 5.

To show average Fatality and Morbidity periods (in weeks).

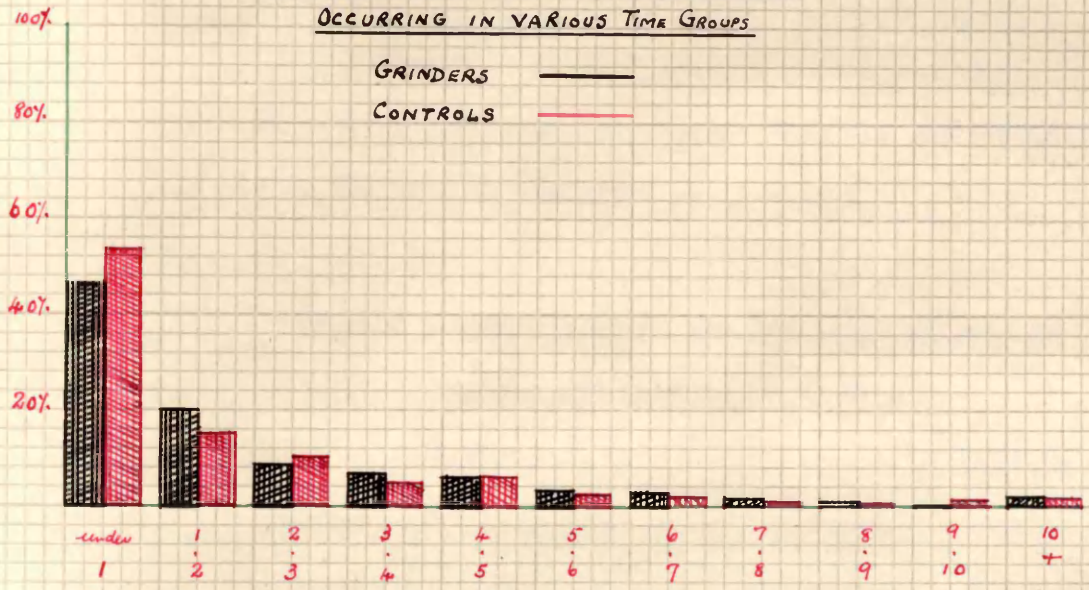
	GRINDERS			CONTROLS		
Year	Number of cases	Fatality	Morbidity	Number of cases	Fatality	Morbidity
1918	36	94	122	176	100	119
1919	30	84	95	129	89	117
1920	26	137	169	147	96	114
1921	30	160	170	137	99	114
1922	47	86	117	143	89	116
1923	21	78	89	111	85	126
1924	32	132	147	126	106	148
1925	24	120	149	131	99	140
1926	29	86	126	116	89	125
1927	35	133	160	145	110	147
1918 - 1927	310	110	135	1,361	97	126

In the Grinders the Fatality period varies from a minimum of 78 weeks in 1923 to a maximum of 160 weeks in 1921, and in the Controls from a minimum of 85 weeks in 1923 to a maximum of 110 weeks in 1927. The variations are rather gross in the case of the Grinders, but this is attributable to the small number of cases in a single year. In five of the years the average Fatality period in the Grinders exceeds that in the Controls and in the other five years vice versa. For the complete series of cases 1918-1927 the average Fatality period of the Grinders is 110 weeks as against an average of 97 weeks in the Controls.

But this average Fatality period is liable to a very serious fallacy. In a limited group of cases, as we are here considering, it is possible, that the great majority of the cases have a very short Fatality period, while the remaining few cases have an unduly long period. The average of all the cases is thus made to appear very favourable and the truth obscured. To deal with this possible error I haveprepared the following table and graphic representation.

1918-1927

To shew Percentage Number of Deaths occurring in various Time Groups



Time Groups	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
under 1 yr.	144	46·08	733	53·51
1 - 2	63	20·16	213	15·55
2 - 3	27	8·64	138	10·07
3 - 4	21	6·72	68	4·97
4 - 5	18	5·76	79	5·77
5 - 6	11	3·52	36	2·63
6 - 7	10	3·20	27	1·97
7 - 8	5	1·60	13	0·95
8 - 9	4	1·28	11	0·81
9 - 10	-	-	13	0·95
10 +	7	2·24	30	2·19
Total	310	ca 100	1,361	ca 100

From this it is immediately apparent that the incidence is practically identical in the two groups, and any slight advantage which may exist favours the Controls. This fallacy then does not apply.

In Table 6 are shown the average Fatality and Morbidity periods in the various specified age groups :

Table 6.

To show average Fatality and Morbidity periods in various age groups (in weeks).

Age Groups	GRINDERS			CONTROLS		
	Number of cases	Fatality	Morbidity	Number of cases	Fatality	Morbidity
15 - 20	4	60	67	99	38	77
20 - 25	12	63	82	129	68	94
25 - 30	17	124	130	147	82	108
30 - 35	18	89	105	157	115	135
35 - 40	37	160	200	169	108	142
40 - 45	40	157	194	169	111	144
45 - 50	62	96	115	167	103	143
50 - 55	47	118	146	133	112	143
55 - 60	36	103	121	89	85	124
60 - 65	30	54	76	61	102	120
65 - 70	6	29	79	33	99	110
70 & over	1	307	314	8	110	114
Average allages	310	110	135	1,361	97	126

The 70 and over group can be excluded as it only comprises 1 Grinder and 8 Controls. In the majority of the age groups up to 60 years the Fatality period in Grinders exceeds that in Controls, and in the remaining groups the approximation is fairly close. The Fatality period in the Grinder between the ages of 35 and 45 years is unusually high, but in view of the small number

of cases, I do not think, one would be altogether justified in concluding that fibrosis of the lungs, which is usually present at this age (i.e.) after 20 years occupation in grinding, impedes the progress of pulmonary tuberculosis, as is suggested by Beattie (8).

After the age of 60 years tuberculosis is apparently very much more rapidly fatal in the Grinders than in the Controls, and this may be cited, as proof of the alleged rapidly fatal nature of this disease as a complication of silicosis, which is undoubtedly present in grinders of this age, who have been employed most of their lives in the trade. However, those who are familiar with the clinical course of the disease in these groups will recall, that these patients do succumb rapidly, not because the tuberculosis has been of a fulminating type, but because the heart, already overburdened by the fibrotic lung, is soon overcome by the additional load of the tuberculosis toxaemia; and so death results from heart failure and, in not a few instances, suddenly from acute cardiac dilatation.

The very rapidly fatal course of pulmonary tuberculosis in the early groups 15 - 25 might possibly have made the average Fatality period for the Controls appear unduly low. Accordingly, these were excluded and it was then found that :

The average Fatality period in Grinders aged 25 years and over - 113 weeks

The average Fatality period in Controls aged 25 years and over - 105 weeks.

Among the Controls the longest Fatality period noted was in the case of a labourer who died on 15:12:27; tubercle bacilli were found

in the sputum for the first time on 15-11-07, i.e., a Fatality period of a little over 20 years; and among the Grinders in the case of a fork grinder (dry grinding) who died on 8-2-27. Tubercle bacilli were found in his sputum originally on 16-1-11, i.e., a Fatality period of a little over 16 years.

The following graphic representation gives a vivid mental picture of the Fatality period. It consists of three parts which show respectively the percentage number of deaths in each of the age groups within 1,2 and 5 years of the first discovery of tubercle bacilli in the sputum.

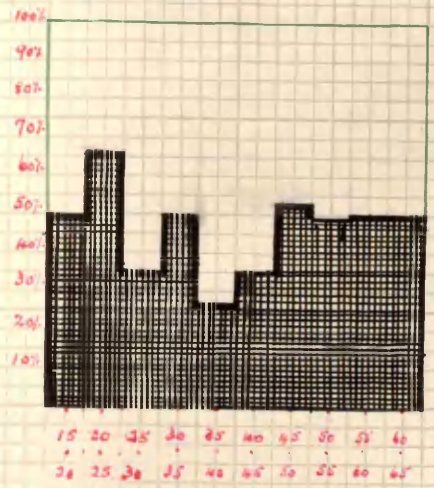
The shaded area represents the percentage of cases dead, the plain area the percentage still alive. The graphs Grinders and Controls, are practically identical; if anything the outlook for the Grinders is slightly the more favourable, at least up to the age of 60 years.

To show total Percentage of Deaths occurring within a certain Period of years from First Discovery of Tubercle Bacilli in Sputum.

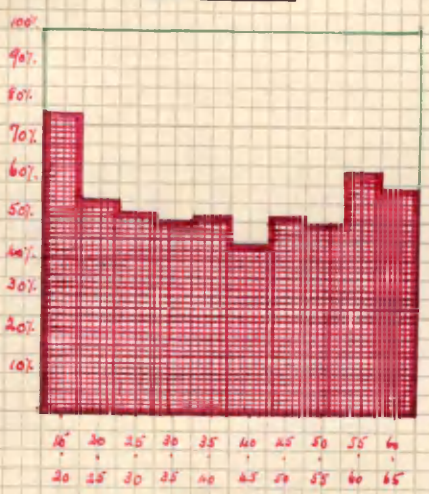
	Grinders			Controls		
Age Group	under 1 year %	under 2 years %	under 5 yrs. %	under 1 year %	under 2 years %	under 5 years %
15 - 20	50·00	75·00	100·00	78·00	91·00	100·00
20 - 25	66·66	74·99	100·00	55·80	76·73	97·65
25 - 30	35·28	58·80	88·20	52·05	67·60	93·29
30 - 35	50·00	72·24	94·48	50·32	64·97	86·00
35 - 40	27·00	45·90	81·00	51·33	69·62	88·50
40 - 45	35·00	52·50	75·00	44·84	60·77	86·73
45 - 50	53·13	75·67	86·94	51·51	65·89	90·45
50 - 55	48·99	57·51	91·59	49·63	64·67	87·98
55 - 60	50·00	77·80	88·92	62·89	76·36	89·84
60 - 65	50·00	86·63	100·00	59·04	68·88	86·92
65 - 70	100·00	100·00	100·00	51·51	72·72	84·84
70 - 75	-	-	-	-	-	-

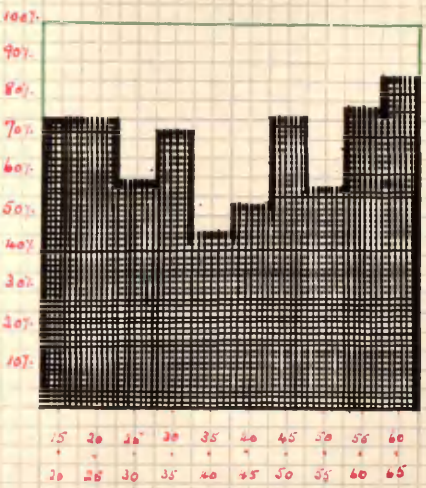
GRINDERS

CONTROLS

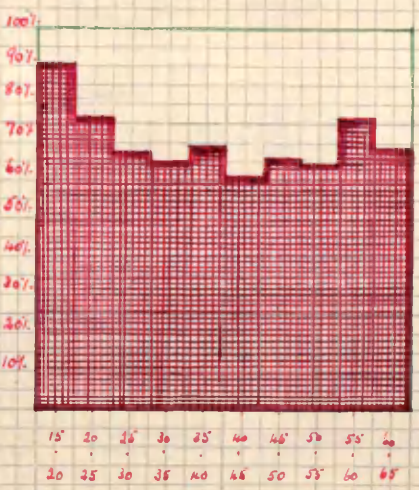


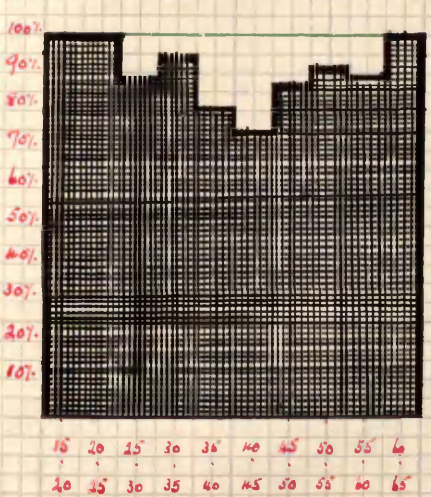
WITHIN 1 YEAR



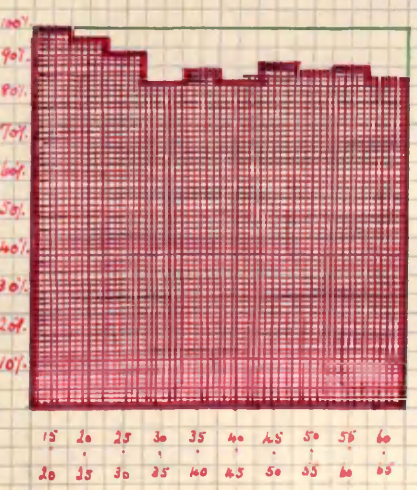


WITHIN 2 YEARS





WITHIN 5 YEARS



Morbidity Period.

For convenience Table 5 is repeated below.

Table 5.

To show average Fatality and Morbidity Periods (in weeks).

Year	GRINDERS			CONTROLS		
	Number of cases	Fatality	Morbidity	Number of cases	Fatality	Morbidity
1918	36	94	122	176	100	119
1919	30	84	95	129	89	117
1920	26	137	169	147	96	114
1921	30	160	170	137	99	114
1922	47	86	117	143	89	116
1923	21	78	89	111	85	126
1924	32	132	147	126	106	148
1925	24	120	149	131	99	140
1926	29	86	126	116	89	125
1927	35	133	160	145	110	147
1918 - 1927	310	110	135	1,361	97	126

In the Grinders the morbidity period varies from a minimum of 89 weeks in 1923 to a maximum of 170 weeks in 1921, and in the Controls, from a minimum of 114 weeks in 1920 and 1921 to a maximum of 148 weeks in 1924. For the whole series the average Morbidity period of Grinders is 135 weeks as aginst 126 weeks in the Controls. In other words, the illness which immediately precedes and leads to the discovery of tubercle bacilli in the sputum has an average duration of 25 weeks in the Grinders (135 - 110) as against 29 weeks in the Controls (126 - 97).

Grinders, it is acknowledged, are very prone to suffer from respiratory diseases other than tuberculosis, and as these are most common in winter time in this country, it was considered possible, that the seasonal incidence of deaths from tuberculosis

in Grinders might show some difference from the Controls.

In Table 7 are set forth details of the incidence of deaths from pulmonary tuberculosis arranged in calendar months :

Table 7.

To show percentage incidence of deaths occurring in each month.

Months	GRINDERS		CONTROLS	
	Number of cases	Percentage	Number of cases	Percentage
January	27	8.64	133	9.71
February	28	8.96	117	8.54
March	36	11.52	131	9.56
April	26	8.32	111	8.10
May	27	8.64	119	8.69
June	22	7.04	118	8.61
July	21	6.72	103	7.52
August	21	6.72	90	6.57
September	23	7.36	93	6.79
October	21	6.72	101	7.37
November	33	10.56	125	9.13
December	25	8.00	120	8.76
Totals	310	ca. 100.00	1,361	ca. 100.00

	Grinders.	Controls.
October - March (inclusive)	54.40 % of cases	53.07 % of cases
April - September (inclusive)	44.80 % of cases	46.28 % of cases

The two groups are practically identical and in each instance the winter months show a slight excess over the summer months. In this connection it is noteworthy that the Influenza Pandemic of 1918 - 1919 did not have any appreciable effect on the number of deaths amongst tuberculous patients.

So far we have been considering the cases of 310 Grinders in whose sputum tubercle bacilli were found; but that is not synonymous with 310 cases of silicosis complicated by tuberculosis, although the average period of occupation in the series exceeds

24 years, a period of time sufficiently long for the development of fibrosis of the lungs (silicosis). However, I have discovered that in 44 cases, silicosis was diagnosed on clinical grounds and, in an additional 36 cases on clinical evidence confirmed by X-ray examination. Details of these cases are set forth in Table 8.

Table 8.

Cases of tubercular-silicosis.

	Number of cases	Average Fatality period	Average Morbidity period
A Clinical cases	44	106	131
B X-ray cases	36	83	115
A plus B	80	96	123
All Grinders	310	110	135
Controls	1,361	97	126

The findings are very similar to those of the whole series and the Fatality and Morbidity are seen to be almost identical in the Grinders and Controls.

The foregoing data demonstrate fairly clearly, that open pulmonary tuberculosis in the Grinders and Controls alike runs a fatal course, on an average in about two years from the date of the first discovery of tubercle bacilli in the sputum. Although, so far as I can discover, no other worker has used the same terminus a quo this finding supports the opinions previously expressed by Watkins-Pitchford (47) and Porter (48). But contrary to the general opinion, alluded to at the outset, the course is no more rapid in the silica worker than that in the average individual.

Conclusion : Tuberculosis complicating silicosis, as seen in Sheffield Metal-Grinders, is not more rapidly fatal than tuberculosis in the average man.

Mean Age at Death.

Brownlee (9) as a result of his classical investigation into the "Epidemiology of Phthisis in Great Britain and Ireland", concluded that phthisis was divisible into three main types, each of which has its chief incidence at a definite age period, thus :-

(a) Young adult type : commonest age at death between 20 and 25 years.

(b) Middle age type : " " " " " 45 and 51 years.

(c) Old age type : " " " " " 55 and 65 years.

In Table 9 below are set forth details of the cases traced in each of the years under consideration and the mean age at death in these instances.

Table 9.

To show themean age at death.

Year	GRINDERS. No. of cases	GRINDERS. Mean age at death	CONTROLS. No. of cases	CONTROLS. Mean age at death
1918	36	45 years	176	37 years
1919	30	42 "	129	40 "
1920	26	44 "	147	41 "
1921	30	44 "	137	39 "
1922	47	46 "	143	36 "
1923	21	48 "	111	39 "
1924	32	47 "	126	39 "
1925	24	47 "	131	41 "
1926	29	49 "	116	40 "
1927	35	47 "	145	40 "
1918 - 1927	310	46 "	1,361	39 "

In every year, without exception, it is seen that the mean age at death for the Grinders is higher than that of the Controls. The average in the Grinders varies from a minimum of 42 years in 1919 to a maximum of 49 years in 1926, and, in the Controls,

from a minimum of 36 years in 1922 to a maximum of 41 years in 1920 and 1925.

For the total number of cases the mean age at death in the Grinders is 46 years as against 39 years in the Controls.

Reference to Table 10 on page 48, however, shows that in the Grinders only 5% of the total deaths occurred before the age of 25 years, whereas almost 17% of the Controls died before that age. This unduly high incidence in the early age groups of the Controls suggested that this was a possible explanation of the great difference in the averages of the two series. Accordingly, all cases under the age of 25 years were excluded and it was then found that -

The mean age at death of Grinders
aged 25 years and over - 47 years.

The mean age at death of Controls
aged 25 years and over - 42 years.

The difference, with slight modification, persisted and so one is probably justified in concluding that the mean age at death from pulmonary tuberculosis in metal-grinders is much higher than in the average male individual, the difference being from 5 to 7 years.

Table 10 shows the cases arranged according to age in groups of 5 years and, to facilitate comparison, the numbers are converted to a percentage basis. And, further to impress this distribution in age groups, graphic representations have been added.

Table 10.

To show distribution of cases in age groups of 5 years.

Age Group	GRINDERS		CONTROLS	
	No. of Cases	%	No. of Cases	%
15 - 20	4	1.28	99	7.23
20 - 25	12	3.84	129	9.42
25 - 30	17	5.44	147	10.73
30 - 35	18	5.76	157	11.46
35 - 40	37	11.84	169	12.34
40 - 45	40	12.80	169	12.34
45 - 50	62	19.84	167	12.19
50 - 55	47	15.04	133	9.71
55 - 60	36	11.52	89	6.50
60 - 65	30	9.60	61	4.45
65 - 70	6	1.92	33	2.41
70 - 75	1	0.32	8	0.58
All ages	310	ca.. 100.00	1,361	ca.100.00

Graph No. 1 represents the whole series of cases in the decennium 1918 to 1927 (both inclusive). The succeeding graphs illustrate the cases in the individual years under consideration.

GRAPHS - EXPLANATORY.

-o-o-o-o-o-o-o-o-o-o-o-o-o-

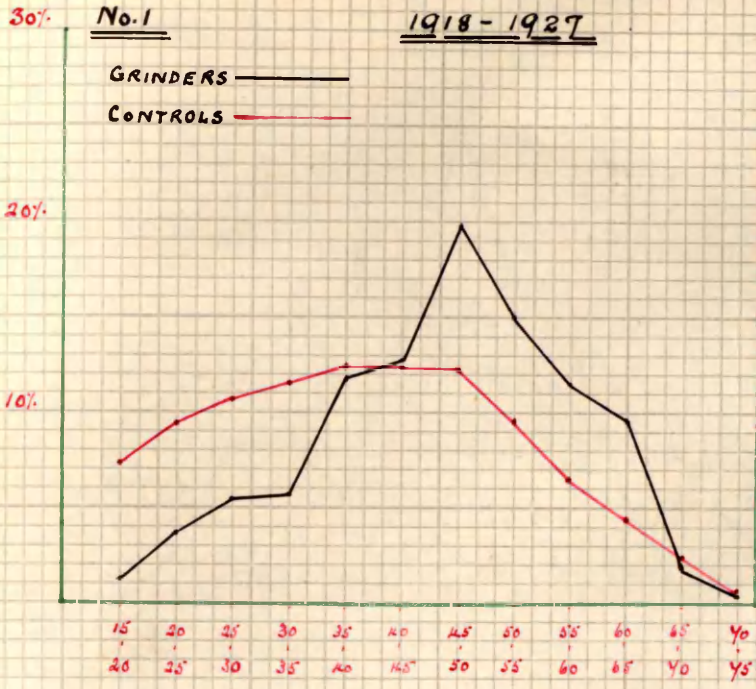
Graphs to show % distribution of the cases in defined age groups.

1. To each graph is appended the appropriate table of data.
2. Grinders are shown in BLACK.

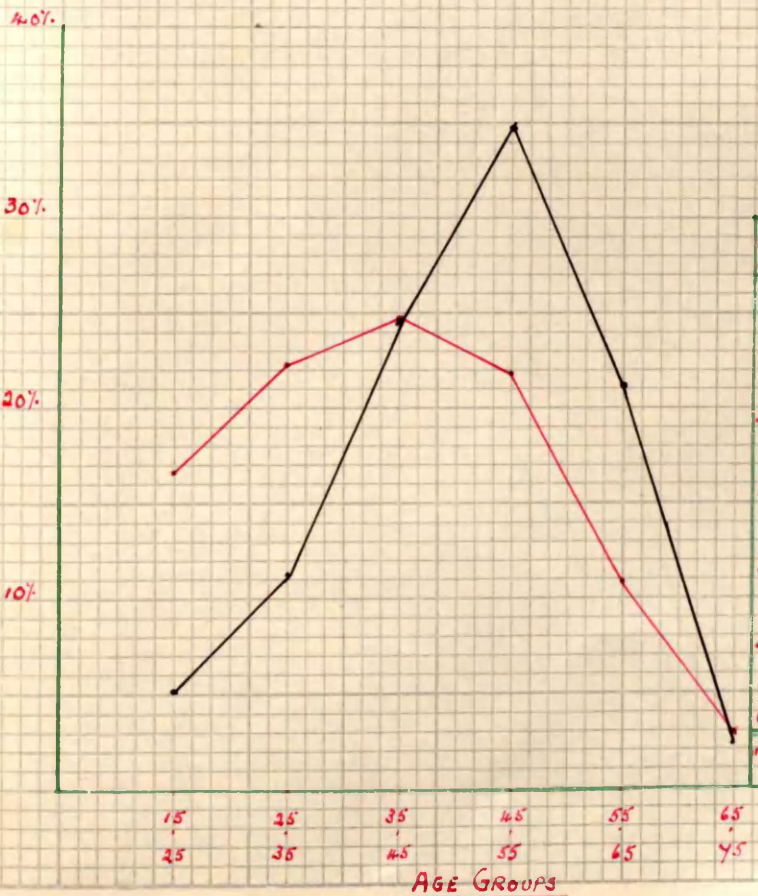
   Controls are shown in RED.
3. Percentages are on the ordinate; age groups on the abscissa.
4. The upper graph shows the % distribution in age groups of 5 years.

   The lower " " " " " " " " " 10 years.
5. Age group 15 - 20 years means 15 years and under 20 years, and so on for each of the other groups.

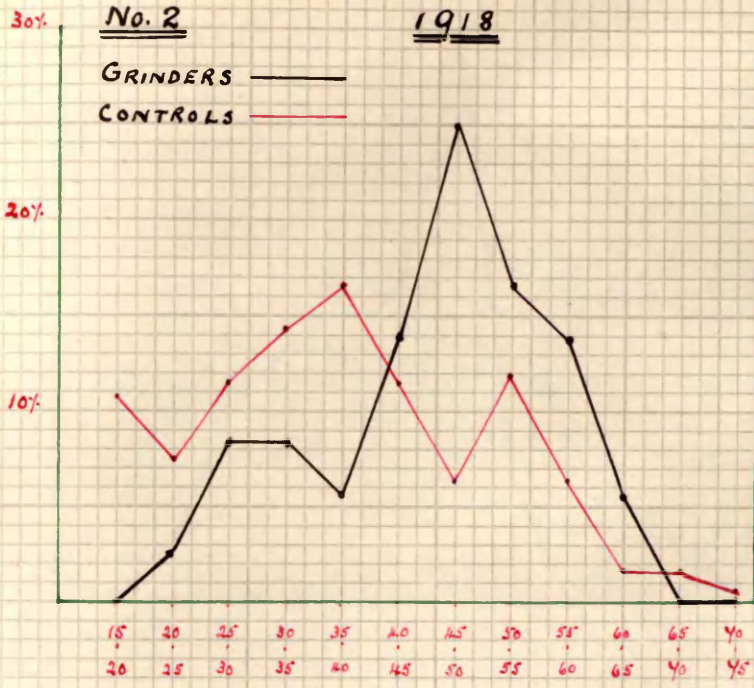
No. 1 1918 - 1927



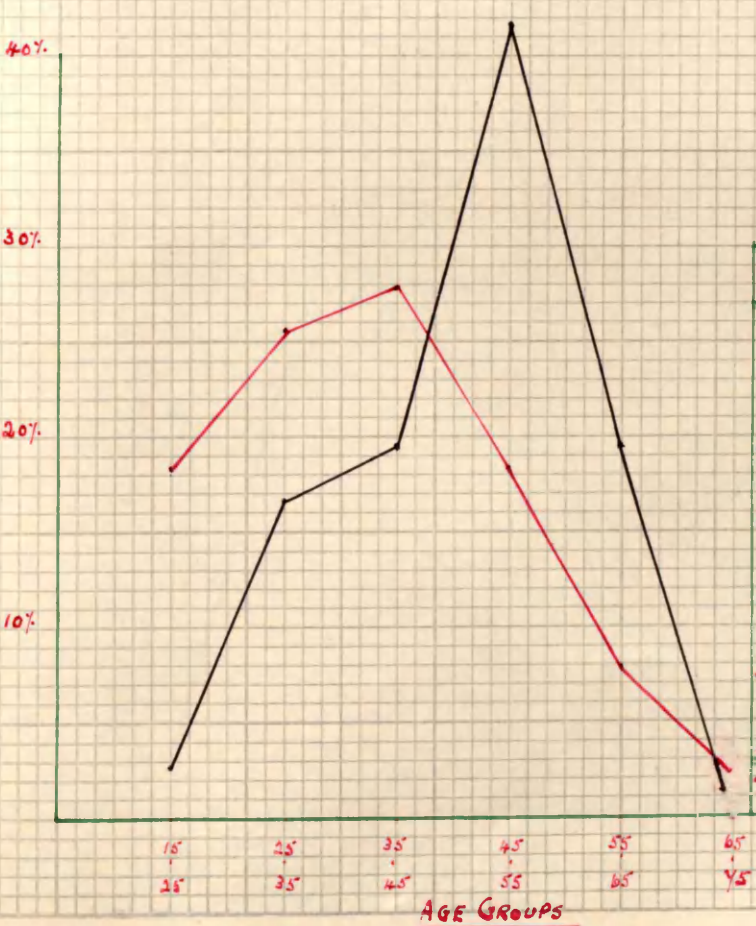
Age Group	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
15-20	4	1·28	99	7·23
20-25	12	3·84	129	9·42
25-30	17	5·44	147	10·73
30-35	18	5·76	157	11·46
35-40	37	11·84	169	12·34
40-45	40	12·80	169	12·34
45-50	62	19·84	167	12·19
50-55	47	15·04	133	9·71
55-60	36	11·52	89	6·50
60-65	30	9·60	61	4·45
65-70	6	1·92	33	2·41
70-75	1	0·32	8	0·68
TOTAL	310	ca 100	1.361	ca 100



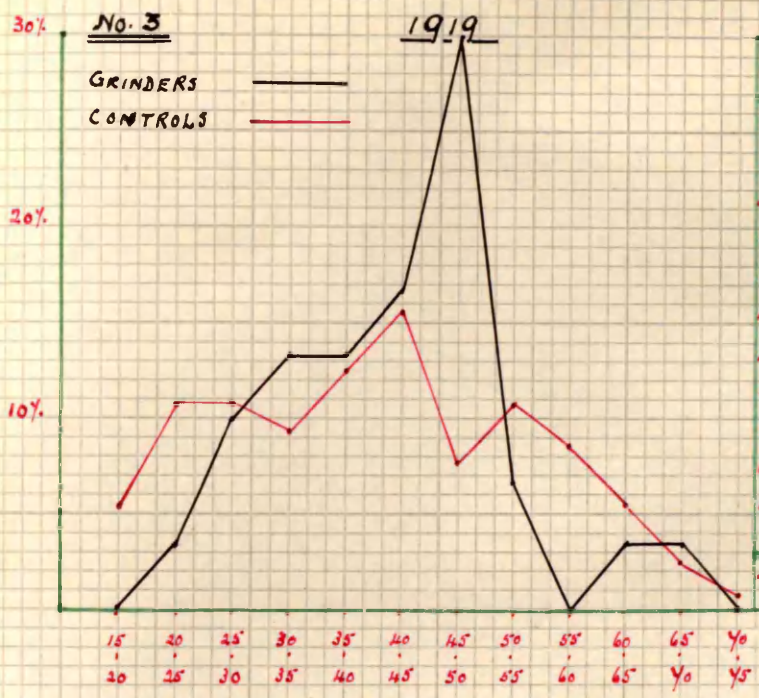
Age Group	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
15-25	16	5·12	228	16·65
25-35	35	11·20	304	22·19
35-45	77	24·64	338	24·68
45-55	109	34·88	300	21·90
55-65	66	21·12	150	10·95
65-75	7	2·24	41	2·99
TOTAL	310	ca 100	1.361	ca 100



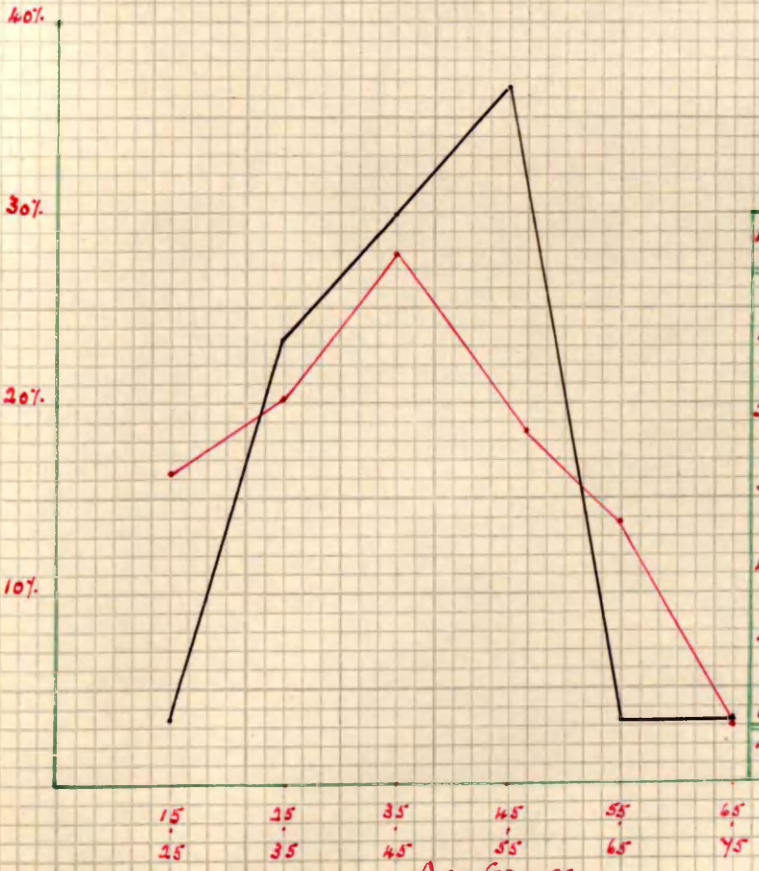
Age Group	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
15-20	-	-	19	10.83
20-25	1	2.78	13	7.41
25-30	3	8.34	20	11.40
30-35	3	8.34	25	14.25
35-40	2	5.56	29	16.52
40-45	5	13.90	20	11.40
45-50	9	25.02	11	6.27
50-55	6	16.68	21	11.97
55-60	5	13.90	11	6.27
60-65	2	5.56	3	1.71
65-70	-	-	3	1.71
70-75	-	-	1	0.57
Total	36	ca 100	176	ca 100



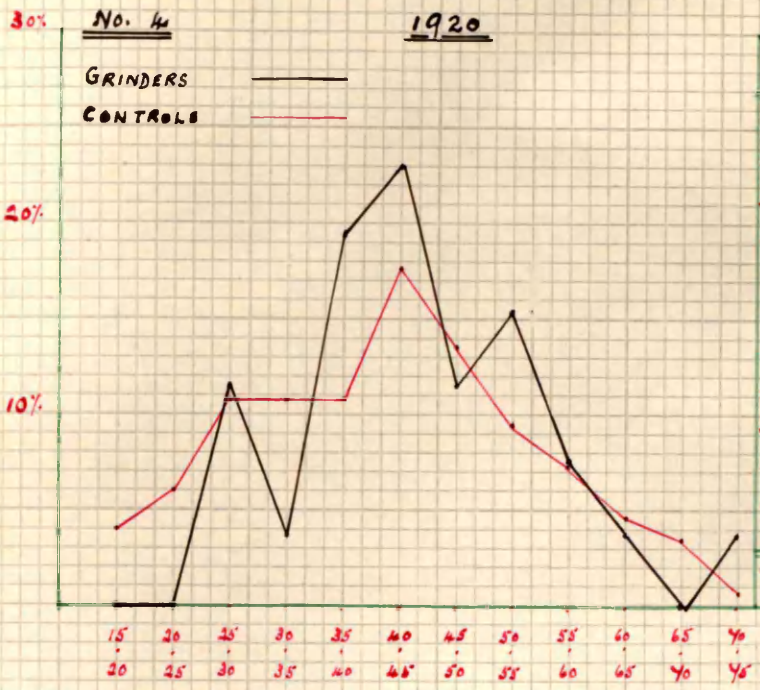
Age Group	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
15-25	1	2.78	32	18.36
25-35	6	16.68	45	25.68
35-45	7	19.46	49	27.93
45-55	15	41.70	32	18.24
55-65	7	19.46	14	7.98
65-75	-	-	4	2.29
Total	36	ca 100	176	ca 100



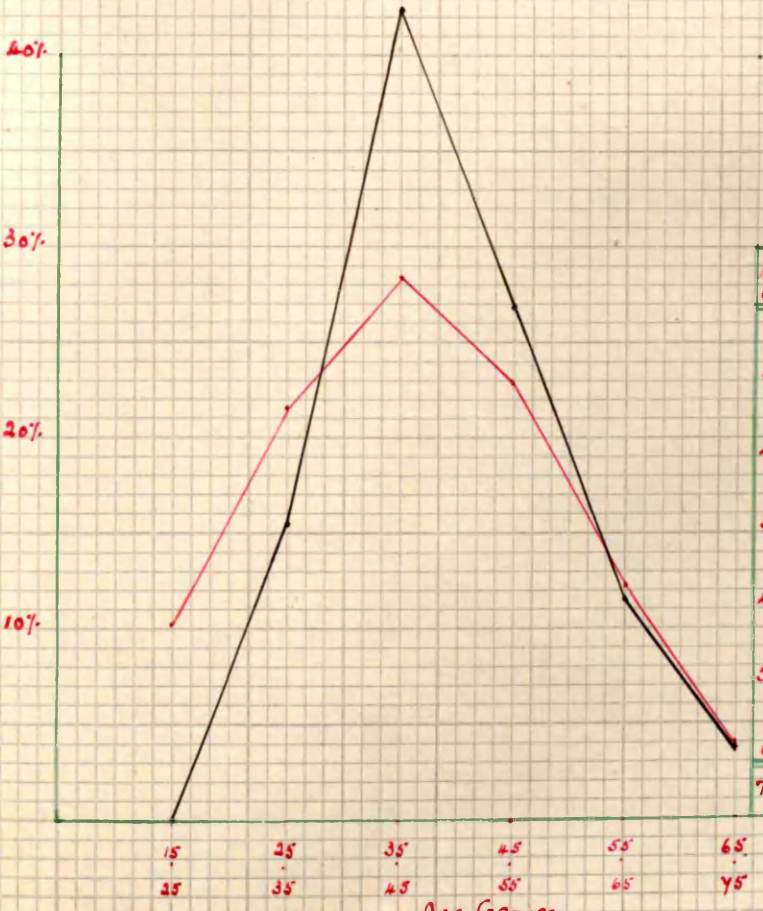
AGE GROUP	GRINDERS No. of CASES	GRINDERS %	CONTROLS No. of CASES	CONTROLS %
15-20	-	-	7	5·43
20-25	1	3·33	14	10·85
25-30	3	9·99	14	10·85
30-35	4	13·32	12	9·30
35-40	4	13·32	16	12·40
40-45	5	16·65	20	15·50
45-50	9	29·97	10	7·75
50-55	2	6·66	14	10·85
55-60	-	-	11	8·53
60-65	1	3·33	7	5·43
65-70	1	3·33	3	2·33
70-75	-	-	1	0·78
TOTAL	30	ca 100	129	ca 100



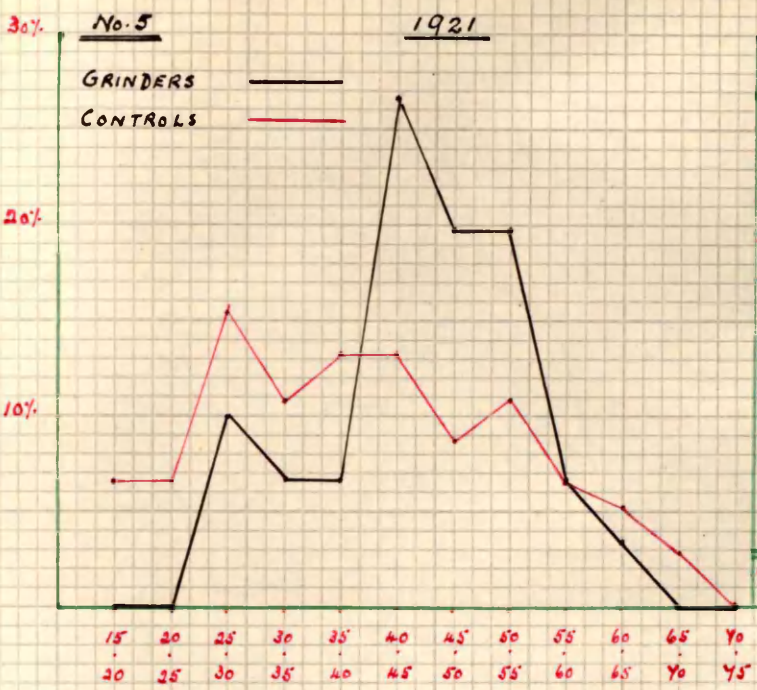
AGE GROUP	GRINDERS No. of CASES	GRINDERS %	CONTROLS No. of CASES	CONTROLS %
15-25	1	3·33	21	16·28
25-35	4	13·31	26	20·15
35-45	9	29·97	36	27·90
45-55	11	36·63	24	18·60
55-65	1	3·33	18	13·75
65-75	1	3·33	4	3·11
TOTAL	30	ca 100	129	ca 100



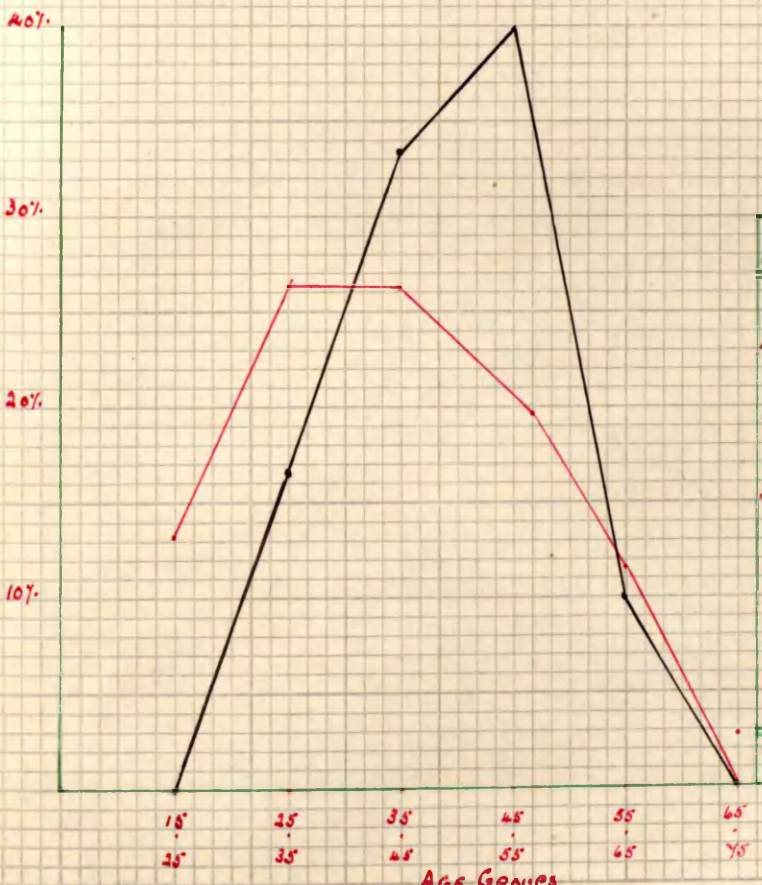
	GRINDERS		CONTROLS	
AGE GROUP	No. of CASES	%	No. of CASES	%
15-20	-	-	6	4·06
20-25	-	-	9	6·09
25-30	3	11·54	16	10·82
30-35	1	3·85	16	10·82
35-40	5	19·23	16	10·82
40-45	6	23·08	26	17·58
45-50	3	11·54	20	13·52
50-55	4	15·38	14	9·46
55-60	2	7·69	11	7·44
60-65	1	3·85	7	4·73
65-70	-	-	5	3·38
70-75	1	3·85	1	0·68
TOTAL	26	ca 100	147	ca 100



	GRINDERS		CONTROLS	
AGE GROUP	No. of CASES	%	No. of CASES	%
15-25	-	-	15	10·14
25-35	4	15·38	32	21·63
35-45	11	42·31	42	28·39
45-55	7	26·92	34	22·98
55-65	3	11·54	18	12·17
65-75	1	3·85	6	4·06
TOTAL	26	ca 100	147	ca 100

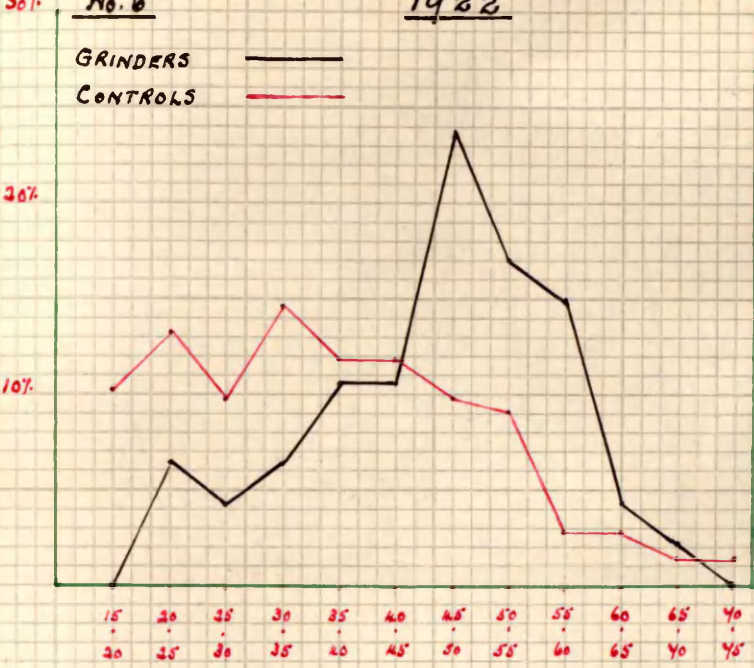


Age Group	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
15-20	-	-	9	6·57
20-25	-	-	9	6·57
25-30	3	9·99	21	15·33
30-35	2	6·66	15	10·95
35-40	2	6·66	18	13·14
40-45	8	26·64	18	13·14
45-50	6	19·98	12	8·76
50-55	6	19·98	15	10·95
55-60	2	6·66	9	6·57
60-65	1	3·33	7	5·11
65-Yo	-	-	4	2·92
Yo-Y5	-	-	-	-
Total	30	ca 100	137	ca 100

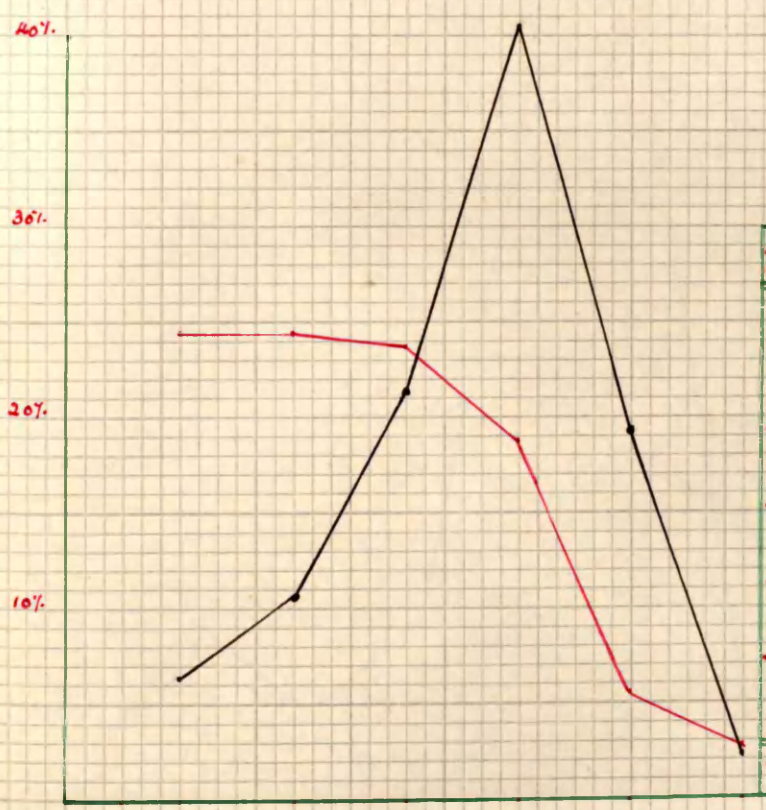


Age Group	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
15-25	-	-	18	13·14
25-35	5	16·65	36	26·28
35-45	10	33·30	36	26·28
45-55	12	39·96	27	19·71
55-65	3	9·99	16	11·68
65-75	-	-	4	2·92
Total	30	ca 100	137	ca 100

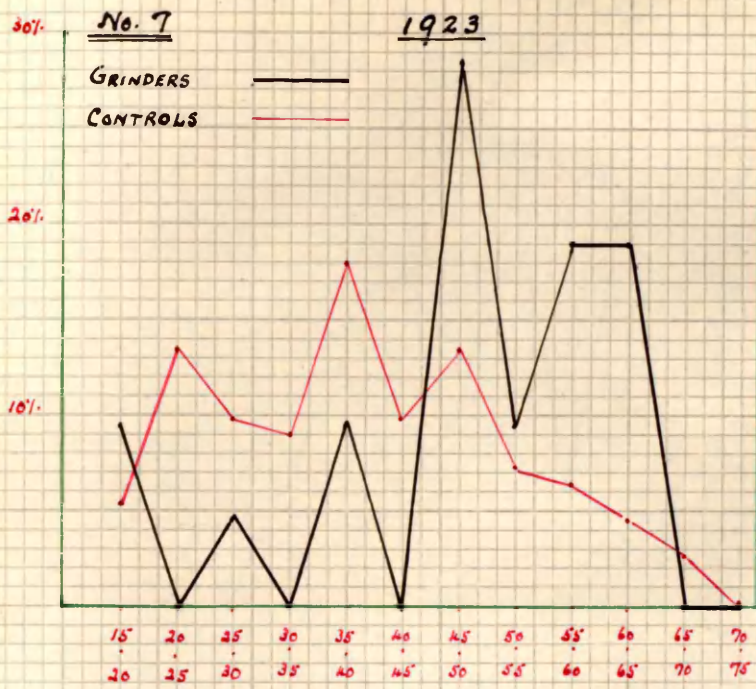
No. 6 — 1922



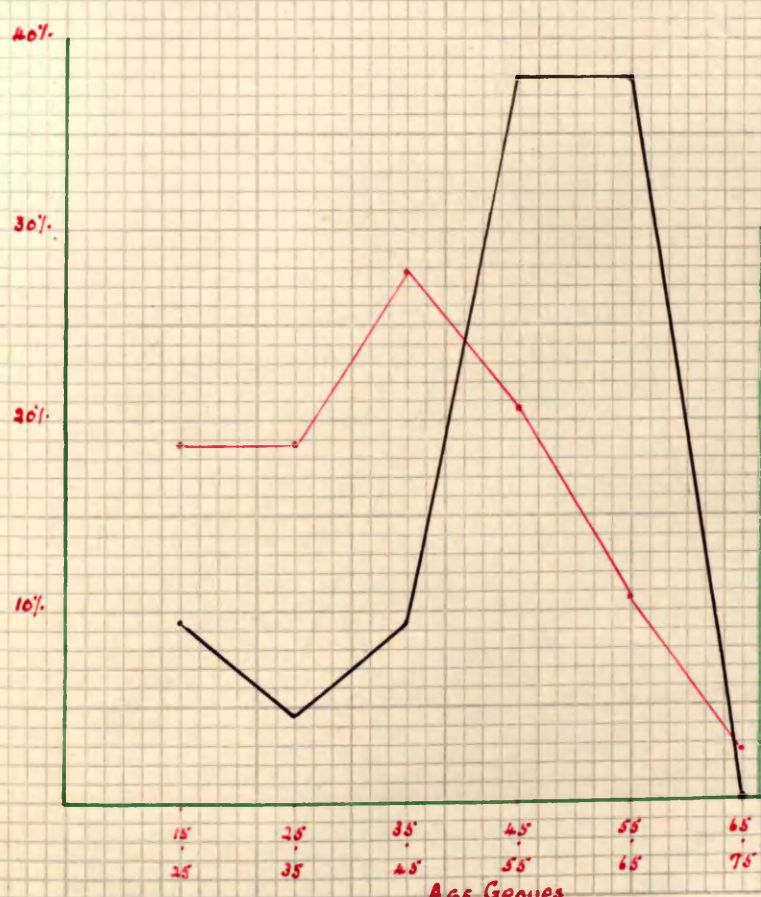
Age Group	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
15-20	-	-	16	11.2
20-25	3	6.39	19	13.3
25-30	2	4.26	14	9.8
30-35	3	6.39	21	14.7
35-40	5	10.65	17	11.9
40-45	5	10.65	17	11.9
45-50	11	23.43	14	9.8
50-55	8	17.04	13	9.1
55-60	7	14.91	4	2.8
60-65	2	4.26	4	2.8
65-70	1	2.13	2	1.4
70-75	-	-	2	1.4
TOTAL	47	ca 100	143	ca 100



Age Group	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
15-25	3	6.39	35	24.5
25-35	5	10.65	35	24.5
35-45	10	21.30	34	23.8
45-55	19	40.47	27	18.9
55-65	9	19.17	8	5.6
65-75	1	2.13	4	2.8
TOTAL	47	ca 100	143	ca 100



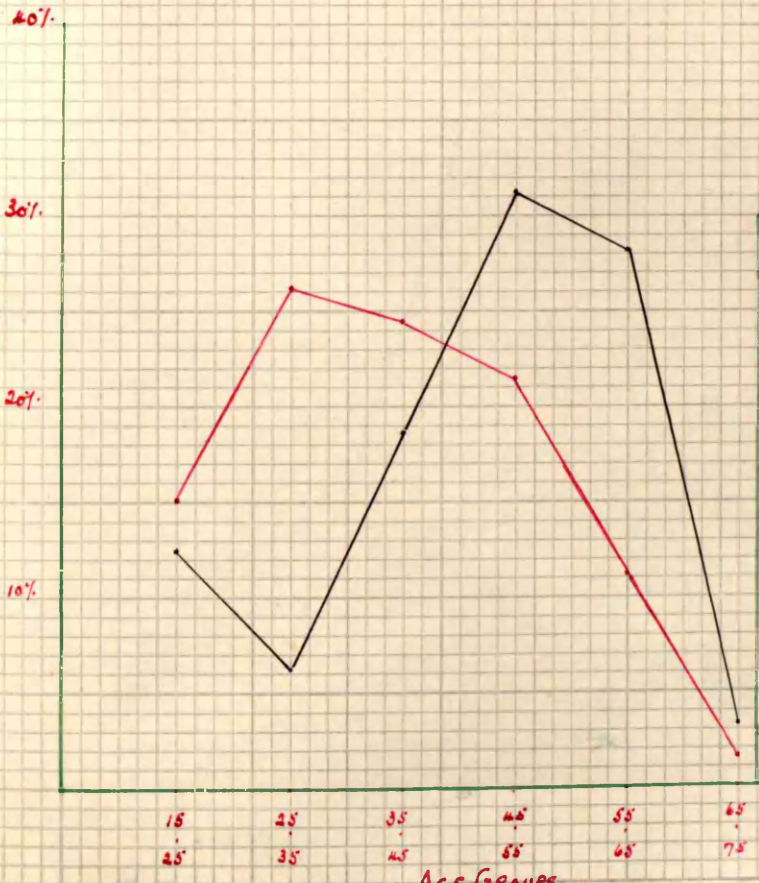
Age Group	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
15-20	2	9.52	6	5.4
20-25	-	-	15	13.5
26-30	1	4.76	11	9.9
30-35	-	-	10	9.0
35-40	2	9.52	20	18.0
40-45	-	-	11	9.9
45-50	6	28.56	15	13.5
50-55	2	9.52	8	7.2
55-60	4	19.04	7	6.3
60-65	4	19.04	5	4.5
65-70	-	-	3	2.7
70-75	-	-	-	-
Total	21	ca 100	111	ca 100



Age Group	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
15-25	2	9.52	21	18.9
26-35	1	4.76	21	18.9
35-45	2	9.52	31	27.9
45-55	8	38.08	23	20.7
56-65	8	38.08	12	10.8
65-75	-	-	3	2.7
Total	21	ca 100	111	ca 100

No 8 — 1924

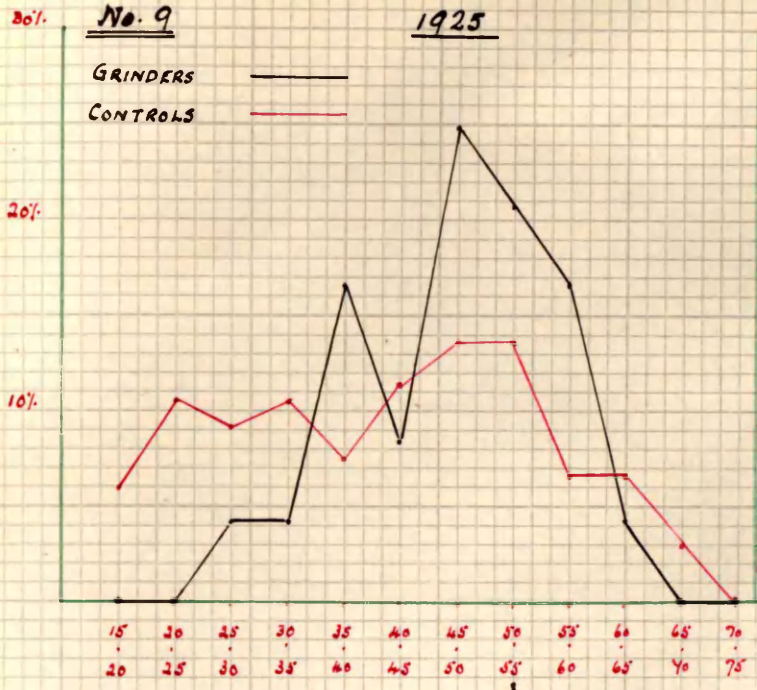
Age Group	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
15-20	2	6·25	9	7·15
20-25	2	6·25	10	7·94
25-30	-	-	14	11·17
30-35	2	6·25	19	15·09
35-40	4	12·50	16	12·70
40-45	2	6·25	15	11·91
45-50	3	9·38	16	12·70
50-55	7	21·88	11	8·73
55-60	2	6·25	9	7·15
60-65	7	21·88	5	3·97
65-70	1	3·13	2	1·59
70-75	-	-	-	-
Total	32	ca 100	126	ca 100



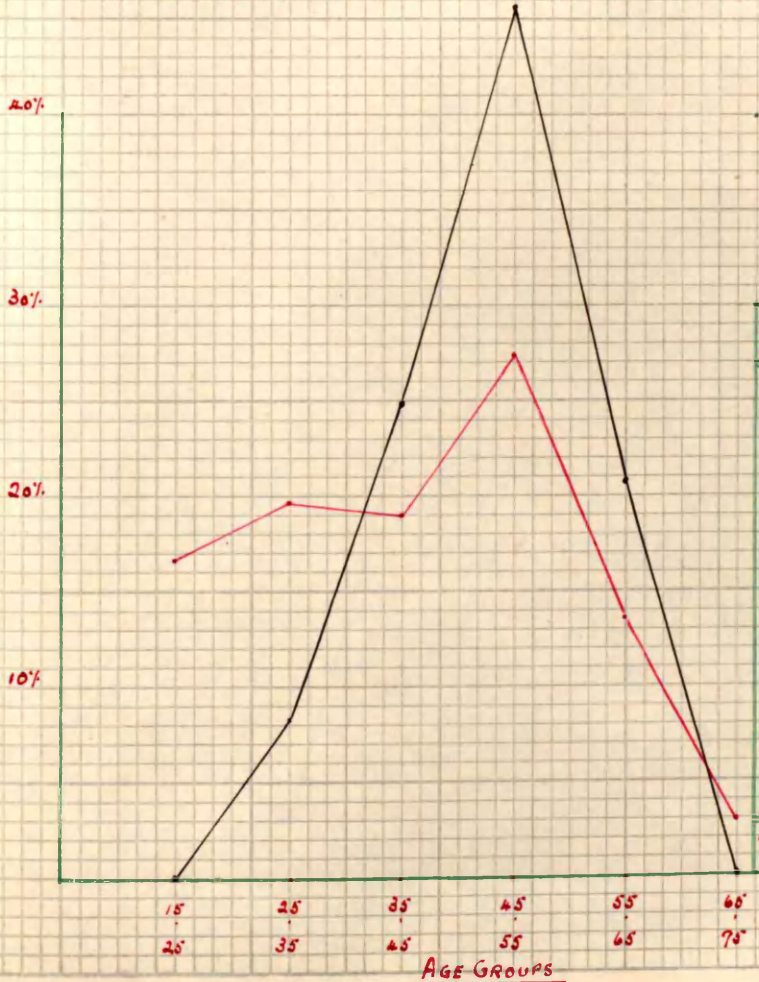
Age Group	Grinders No. of Cases	Grinders %	Controls No. of Cases	Controls %
15-25	4	12·50	19	15·09
25-35	2	6·25	33	26·20
35-45	6	18·75	31	24·61
45-55	10	31·25	27	21·44
55-65	9	28·13	14	11·17
65-75	1	3·13	2	1·59
Total	32	ca 100	126	ca 100

No. 9 1925

GRINDERS
CONTROLS



AGE GROUP	GRINDERS No. of CASES	GRINDERS %	CONTROLS No. of CASES	CONTROLS %
15-20	-	-	8	6·08
20-25	-	-	14	10·64
25-30	1	4·16	12	9·12
30-35	1	4·16	14	10·64
35-40	4	16·64	10	7·60
40-45	2	8·32	15	11·40
45-50	6	24·96	18	13·69
50-55	5	20·80	18	13·68
55-60	4	16·64	9	6·84
60-65	1	4·16	9	6·84
65-70	-	-	4	3·04
70-75	-	-	-	-
TOTAL	24	ca 100	131	ca 100



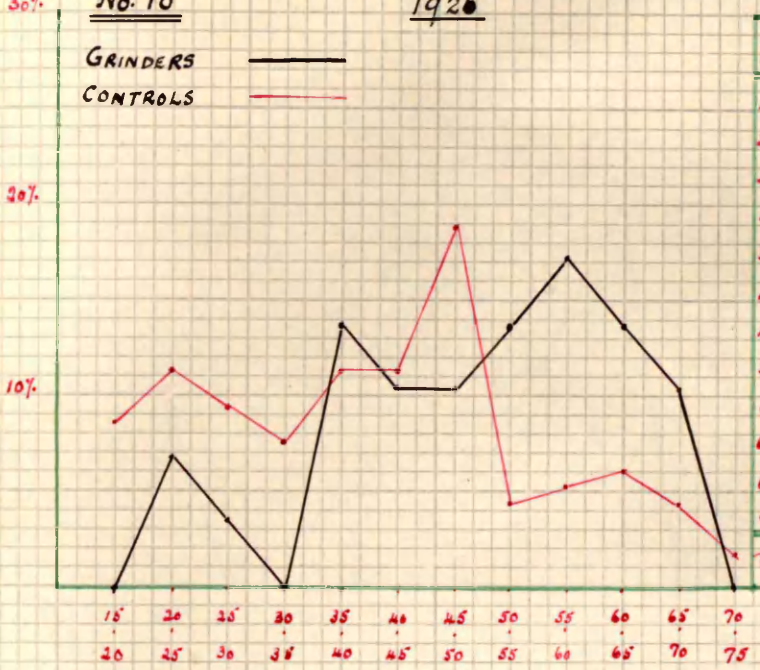
AGE GROUP	GRINDERS No. of CASES	GRINDERS %	CONTROLS No. of CASES	CONTROLS %
15-25	-	-	22	16·72
25-35	2	8·32	26	19·76
35-45	6	24·96	25	19·00
45-55	11	45·76	36	27·36
55-65	5	20·80	18	13·68
65-75	-	-	4	3·04
TOTAL	24	ca 100	131	ca 100

AGE GROUPS

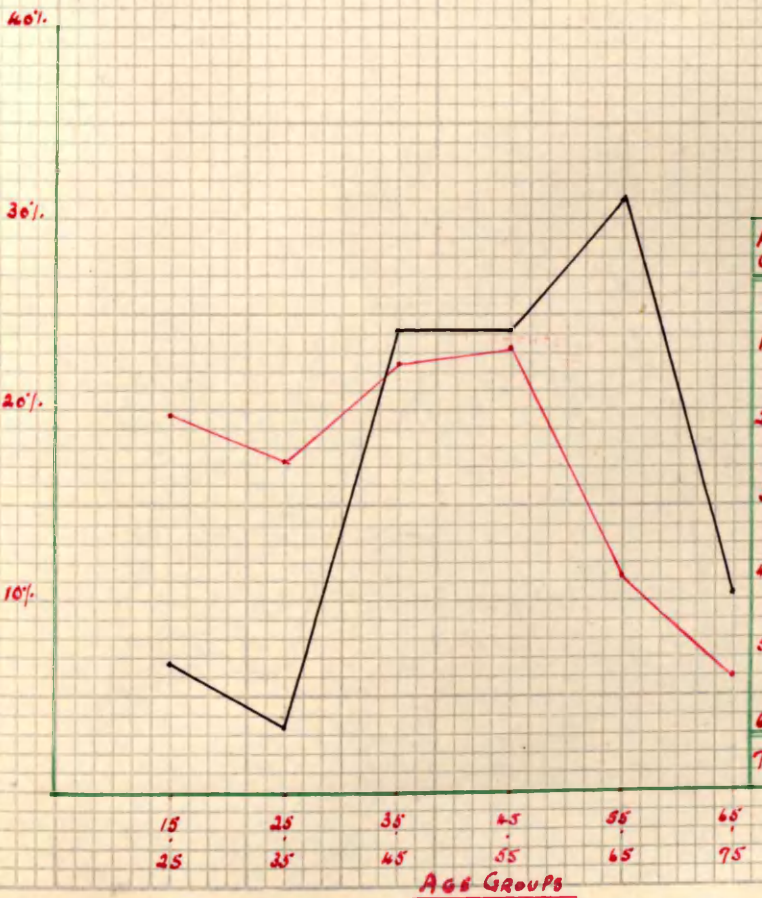
No. 10 1926

GRINDERS

CONTROLS



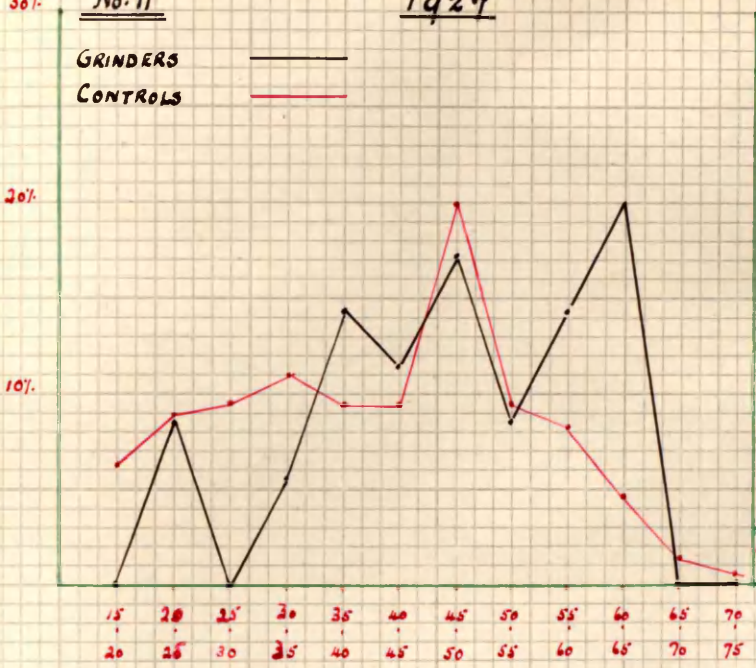
AGE GROUP	GRINDERS No. of CASES	GRINDERS %	CONTROLS No. of CASES	CONTROLS %
15-20	-	-	10	8.62
20-25	2	6.90	13	11.21
25-30	1	3.45	11	9.48
30-35	-	-	9	7.76
35-40	4	13.80	13	11.21
40-45	3	10.34	13	11.21
45-50	3	10.34	22	18.96
50-55	4	13.80	5	4.31
55-60	5	17.24	6	5.17
60-65	4	13.80	7	6.03
65-70	3	10.34	5	4.31
70-75	-	-	2	1.72
TOTAL	29	ca 100	116	ca 100



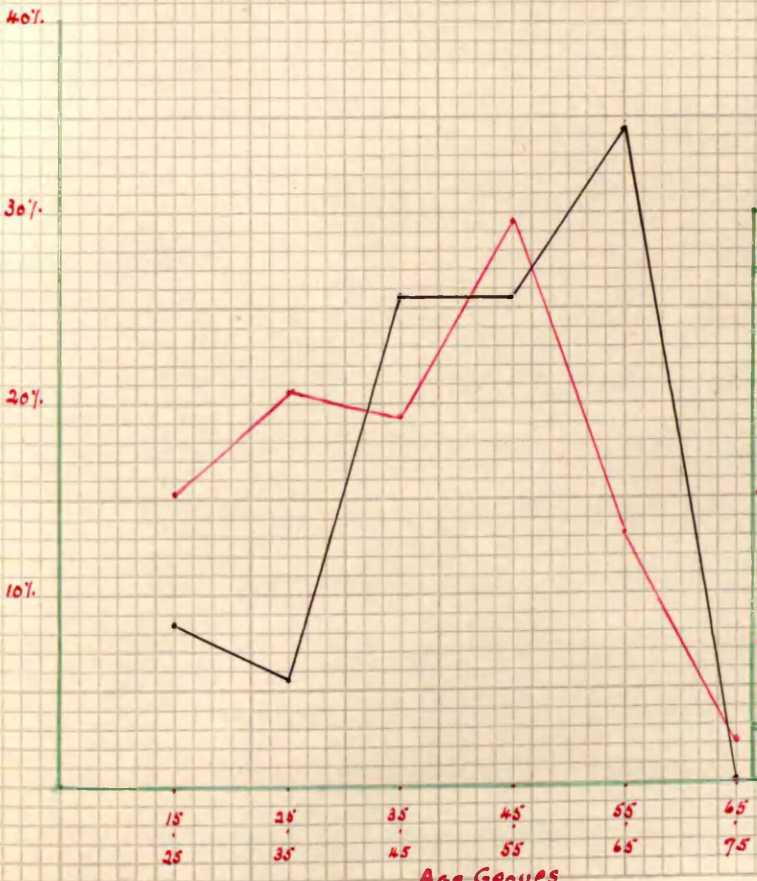
AGE GROUP	GRINDERS No. of CASES	GRINDERS %	CONTROLS No. of CASES	CONTROLS %
15-25	2	6.90	23	19.83
25-35	1	3.45	20	17.24
35-45	7	24.14	26	22.41
45-55	7	24.14	27	23.27
55-65	9	31.03	13	11.21
65-75	3	10.34	7	6.03
TOTAL	29	ca 100	116	ca 100

No. 11 — 1927

GRINDERS
CONTROLS



Age Group	GRINDERS No. of Cases	%	CONTROLS No. of Cases	%
15-20	-	-	9	6.21
20-25	3	8.58	13	8.97
25-30	-	-	14	9.66
30-35	2	5.72	16	11.04
35-40	5	14.30	14	9.66
40-45	4	11.44	14	9.66
45-50	6	17.16	29	20.01
50-55	3	8.58	14	9.66
55-60	5	14.30	12	8.28
60-65	7	20.02	7	4.83
65-70	-	-	2	1.38
70-75	-	-	1	0.69
TOTAL	35	ca 100	145	ca 100



Age Group	GRINDERS No. of Cases	%	CONTROLS No. of Cases	%
15-25	3	8.58	22	15.18
25-35	2	5.72	30	20.70
35-45	9	25.74	28	19.32
45-55	9	25.74	43	29.67
55-65	12	34.32	19	13.11
65-75	-	-	3	2.07
TOTAL	35	ca 100	145	ca 100

Analysis of Curves.

The relatively small number of Grinders in the separate years rather minimises the value of the curves for the individual years, but their analysis is not without value. The graph of all the cases for the whole ten years (1918 - 1927) merits the most serious consideration.

Graph No. 1 (1918 - 1927 - Age groups of 5 years).

Controls.

This shows that a little over 7% of the total cases die between the ages of 15 and 20 years. In the succeeding groups there is a steady gradual rise untilthe 40 - 45 group is reached. This is the maximum point, viz., a little over 12% of the total cases. There is a slight fall in the 45 - 50 group and, thereafter, the decline is rapidly progressive and zero is almost reached in 70 - 75 group.

Grinders.

Only 1.28% of the cases fall in the 15 - 20 group. The curve rises slowly to 30 - 35 group after which there is a sharp rise in the 35 - 40 group. There is a further slight rise in the 40 - 45 group, at which point the curves (Grinders and Controls) practically coincide. In the next group 45 - 50 there is a further sharp rise to a maximum of approximately 20% of the total cases. The curve now declines slowly to 60 - 65 group which still accounts for 10% of total cases. Thereafter, the fall is rapid and in 70 - 75 group the curve almost reaches zero.

For all practical purposes, the 70 - 75 group may be neglected. This then makes the 40 - 45 group the central axis of the graph.

In the case of the Controls, the graph takes the form of a simple symmetrical curve (the downgrade is slightly steeper than the upgrade), with its apex on the central axis. The Grinders' graph, however, is skew and apparently composite. A first peak is reached in the 40 - 45 group and almost coincides with the apex of the curve of the Controls. Then follows a second and higher peak in the 45 - 50 group. Thereafter, the curve declines but, up to the 60 - 65 group, it remains at a very much higher level than that of the Controls.

A scrunity of the curves for the individual years is now of great interest.

In the Controls it is seen that, in every case, the graph is essentially a simple curve starting off at a fairly high level but, from 1918 - 1923 inclusive, the apex tends to occur before the 40 - 45 group and from 1924 onwards to occur after the 40 - 45 group.

In the case of the Grinders (always remembering the small number of cases, which results in rather gross variations), it is again seen that the curve is skew and the maximum incidence after the 40 - 45 group (i.e.) beyond the central axis.

The composite nature of the curve is also fairly apparent as in practically every instance, a primary peak occurs at or before the 40 - 45 group and a second higher after 40 - 45 group.

Graph No. 1 (1918 -,1927. Age groups of 10 years).

For the Controls, this seems to confirm the previous findings, viz., a simple symmetrical curve withits apex in 35 - 45 group.

In the case of the Grinders, however, this re-arrangement

of age groups converts the graph to the form of a simple curve. The skewness is still present; the apex occurs about 45 - 55 group, (i.e.) beyond the central axis.

The composite character is slightly obscured, but study of the curve reveals that, in most instances, there is a sharp rise in 35 - 45 group followed by a further sharp rise to the maximum in 45 - 55 group. In addition, it isagain interesting to note in the 1918 - 1927 graph that the curves coincide in 35 - 45 group, a feature which can be seen in individual years, e.g., 1919 and 1922.

From this analysis of the graphs I think one is justified in concluding that, in the caseof the Controls, there is a definite and fairly considerable incidence of fatal cases of pulmonary tuberculosis in adolescence and early adult life (15 - 25 group). The incidence increases in succeeding age groups till the maximum is reached in middle life (35 - 45 group). Thereafter, there is a steady and progressive decline towards zero.

The case of the Grinders is very different from the above. In the adolescent and early adult ages (15 - 25 group), the percentage incidence of fatal cases of pulmonary tuberculosis is very low. This is, probably, to be explained in some measure by the fact that grinding is known to involve much hard manual labour, with the result that only those who are robust and physically fit choose it as an occupation. Unfortunately, in many cases, a son, whether physically suitable or not, follows in the steps of his father. If, perchance, the father is an infectious case of pulmonary tuberculosis, the son runs a very

grave risk of falling a victim to this disease early in his working life. The maximum percentage incidence of fatal cases occurs after the age of 45 years. It is maintained at an unusually high level in late middle life and onwards (45 - 65 groups), after which the incidence declines rapidly.

Two peaks have been noted in the Grinders' curves. The explanation of these is not obvious and so far as I can find, have not been noted hitherto. I suggest that the two peaks are to be interpreted as indicating two distinct groups of fatal cases of pulmonary tuberculosis among these workers. These I will call :

(a) Natural group.

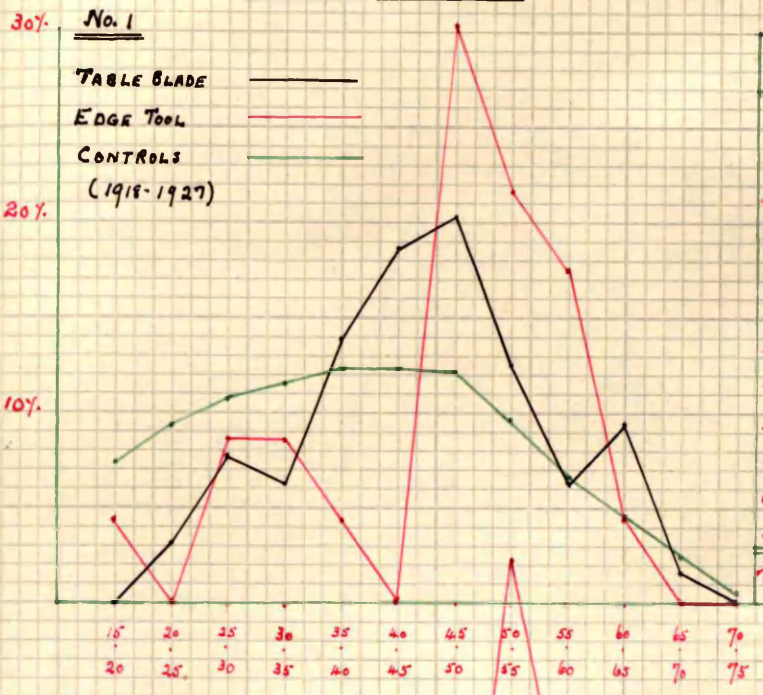
(b) Industrialgroup.

The Natural group includes those Grinders who, by heredity and (or) intimate and frequent contact in private life with an infectious case of pulmonary tuberculosis would have died from tuberculosis irrespective of theoccupation they had chosen to follow. These cases account for the peak at or before 40 - 45 group and to the point where the curves of the Grinders and Controls coincide.

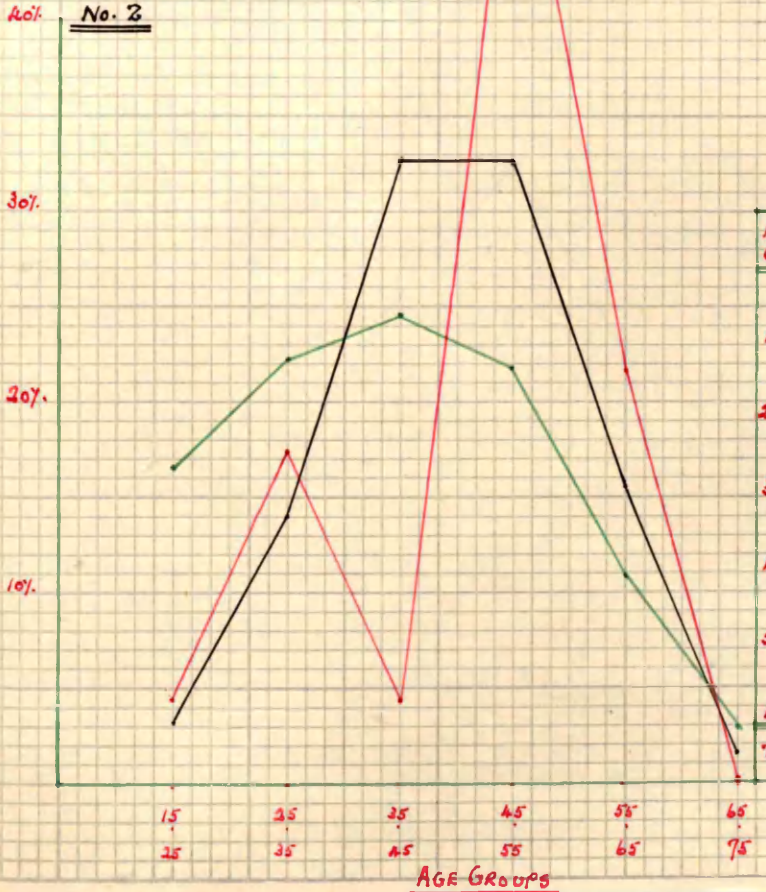
The Industrial group comprises the Grinders who acquire pulmonary tuberculosis as a result of their occupation, and these account for the second peak (i.e.) the heavy percentage incidence in late middle life onwards (45 - 65)groups). These are the cases of true tubercular silicosis.

Six graphs, illustrating the percentage incidence of the cases in various classes of Grinders, are now added. These help to

CLASSES of GRINDERS



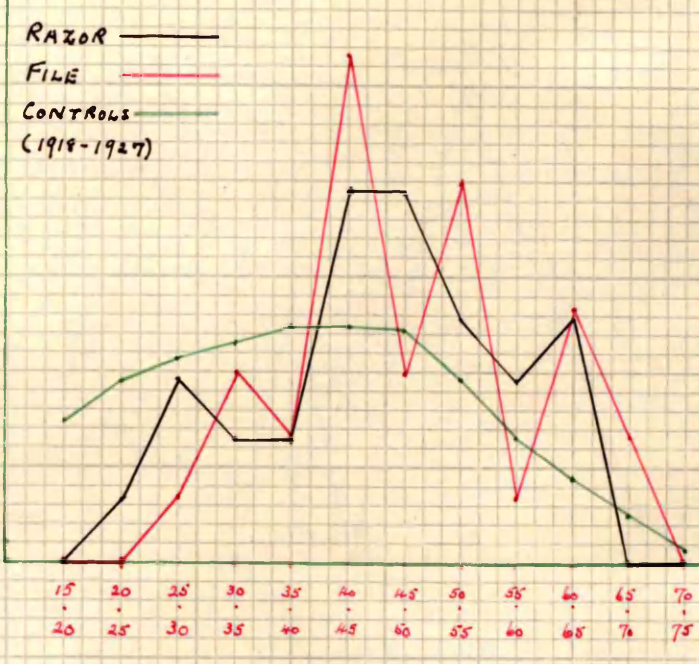
AGE GROUP	TABLE BLADE No. of CASES	%	EDGE TOOL No. of CASES	%
15-20	-	-	1	4.35
20-25	2	3.12	-	-
25-30	5	7.80	2	8.70
30-35	4	6.24	2	8.70
35-40	9	14.04	1	4.35
40-45	12	18.72	-	-
45-50	13	20.28	7	30.45
50-55	8	12.48	5	21.75
55-60	4	6.24	4	17.40
60-65	6	9.36	1	4.35
65-70	1	1.56	-	-
70-75	-	-	-	-
TOTAL	64	ca 100	23	ca 100



AGE GROUP	TABLE BLADE No. of CASES	%	EDGE TOOL No. of CASES	%
15-25	2	3.12	1	4.35
25-35	9	14.04	4	17.40
35-45	21	32.76	1	4.35
45-55	21	32.76	12	52.20
55-65	10	15.60	5	21.75
65-75	1	1.56	-	-
TOTAL	64	ca 100	23	ca 100

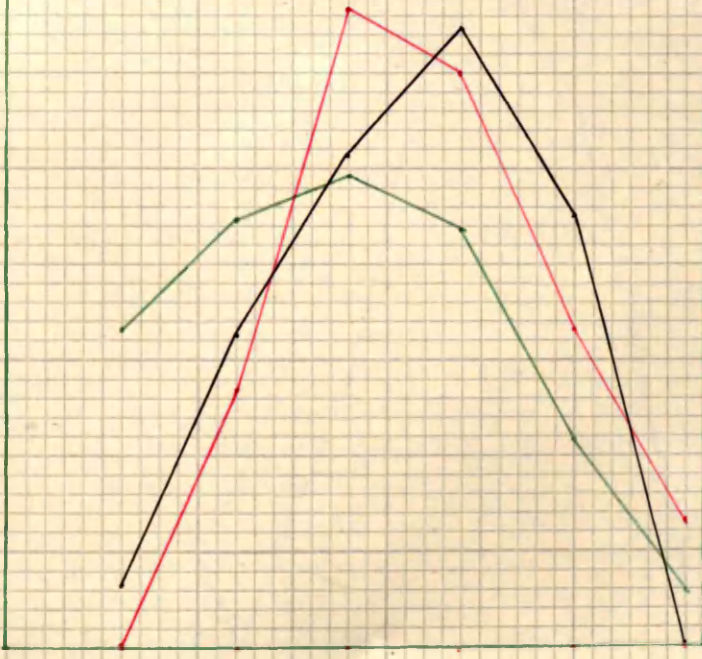
CLASSES of GRINDERS

No. 3.



	RAZOR		FILE	
AGE GROUP	No. of CASES	%	No. of CASES	%
15-20	-	-	-	-
20-25	1	3·23	-	-
25-30	3	9·69	1	3·33
30-35	2	6·46	3	9·99
35-40	2	6·46	2	6·66
40-45	6	19·38	8	26·64
45-50	6	19·38	3	9·99
50-55	4	12·92	6	19·98
55-60	3	9·69	1	3·33
60-65	4	12·92	4	13·32
65-70	-	-	2	6·66
70-75	-	-	-	-
TOTAL	31	ca 100	30	ca 100

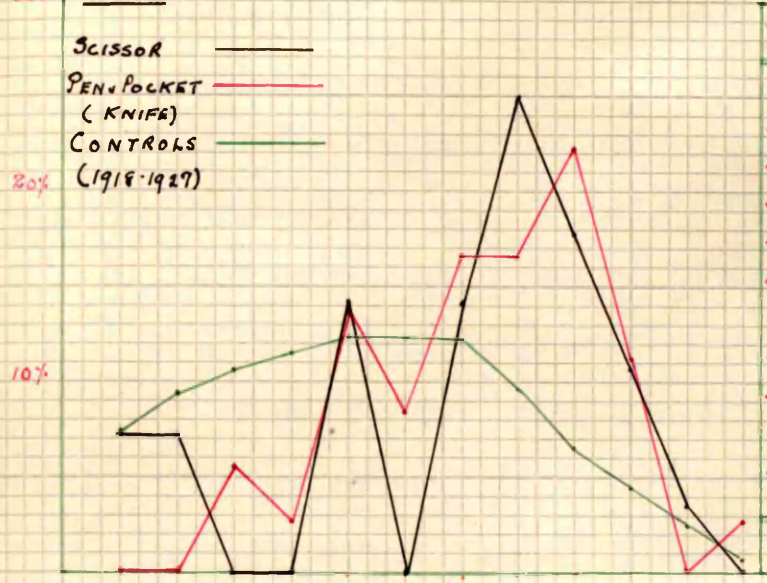
No. 4



	RAZOR		FILE	
AGE GROUP	No. of CASES	%	No. of CASES	%
15-25	1	3·23	-	-
25-35	5	16·15	4	13·32
35-45	8	25·84	10	33·30
45-55	10	32·30	9	29·97
55-65	7	22·61	5	16·65
65-75	-	-	2	6·66
TOTAL	31	ca 100	30	ca 100

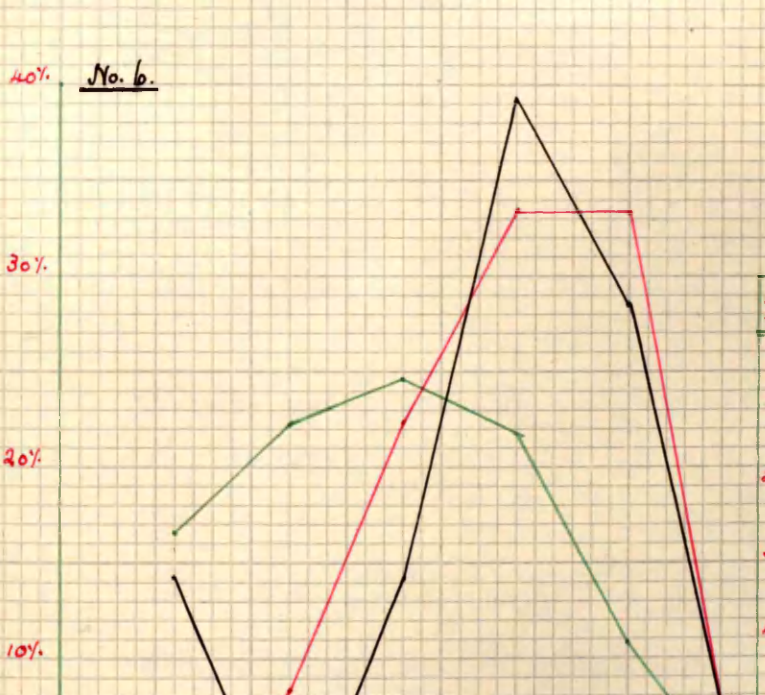
CLASSES of GRINDERS

No. 5



AGE GROUP	SCISSOR No. of CASES	SCISSOR %	PEN & POCKET No. of CASES	PEN & POCKET %
15-20	2	7.14	-	-
20-25	2	7.14	-	-
25-30	-	-	2	5.56
30-35	-	-	1	2.78
35-40	4	14.28	5	13.90
40-45	-	-	3	8.34
45-50	4	14.28	6	16.68
50-55	7	24.99	6	16.68
55-60	5	17.86	8	22.24
60-65	3	10.71	4	11.12
65-70	1	3.57	-	-
70-75	-	-	1	2.78
TOTAL	28	ca 100	36	ca 100

No. 6.



AGE GROUP	SCISSOR No. of CASES	SCISSOR %	PEN & POCKET No. of CASES	PEN & POCKET %
15-25	4	14.28	-	-
25-35	-	-	3	8.34
35-45	4	14.28	8	22.24
45-55	11	39.27	12	33.36
55-65	8	28.56	12	33.36
65-75	1	3.57	1	2.78
TOTAL	28	ca 100	36	ca 100

confirm the three characteristics of the Grinders' curves already emphasized.

(a) the relatively small incidence of cases in early age groups (15 - 25 years).

(b) the skewness of the curves (i.e.) maximum incidence beyond the age of 45 years.

(c) the composite nature of the curves (i.e.) a primary peak at or before 45 years and a second higher peak at or beyond 45 years.

Table 11.

Same as Table 10 but age groups re-arranged.

	GRINDERS		CONTROLS.	
Age groups	No. of Cases	%	No. of Cases	%
under 45 years	128	40.96	870	63.52
over 45 years	182	58.24	491	35.84
All ages	310	ca. 100.00	1,361	ca. 100.00
25 - 45 years	112	35.84	642	46.87
45 - 65 years	175	56.00	450	32.85
other ages	23	7.36	269	19.64
All ages	310	ca. 100.00	1,361	ca. 100.00

This conveys very clearly the fact that, in the Controls, the chief percentage incidence of fatal cases of pulmonary tuberculosis is in early middle and middle life (25 - 45 years), but, in the Grinders, it is in late middle life and onwards (45 - 65 years).

It may be urged, however, that the mean age at death is,

statistically, an almost worthless constant for public health purposes because it is mainly fixed by the age constitution of the population at risk. To take a well known illustration; the mean age at death of curates is much lower than that of bishops, not because it is specially hygienic to wear knee breeches instead of trousers, but because living bishops, as a class, are much older than living curates. This line of argument of course applies only to those trades or professions in which men proceed mainly by seniority (e.g.) subalterns to generals, curates to bishops, captains to rear-admirals. But it does not apply to an industry, such as grinding, into which the men enter as apprentices and, throughout their lives, are referred to as grinders, irrespective of skill or experience. Furthermore, this industry has long since reached its maximum expansion in this country and there has been no sudden influx of young men to its ranks within recent years, or yet any material alteration in the numbers of men of mature years. In short, the age constitution of the trade in this century has remained fairly constant.

Nevertheless, with a view to throwing further light on the age incidence of tuberculosis in Sheffield grinders, I have investigated the series of living cases for the years 1919, 1920 and 1921 already cited on p. 34. On this occasion, instead of the mean age at death, I have determined the mean age at the date of the first discovery of tubercle bacilli in the sputum.

The results are set forth in Table 12 :-

Table 12.

Mean age at date of first discovery of tubercle bacilli in the sputum.

	1919	1920	1921	(1919 - 1921)
Grinders	40 years	43 years	45 years	43 years
Controls	41 "	40 "	39 "	39 "

Again, it is seen that the mean age in the Grinder is higher than in the Control.

The following graph shows the percentage distribution of these cases in various age groups.

The curves are very similar to those found in the series of fatal cases analysed, and once more it is clear that pulmonary tuberculosis in the Grinders is predominantly a disease of late middle life. Furthermore, the hypothesis of two groups (2) natural and (b) industrial is, I think, still tenable.

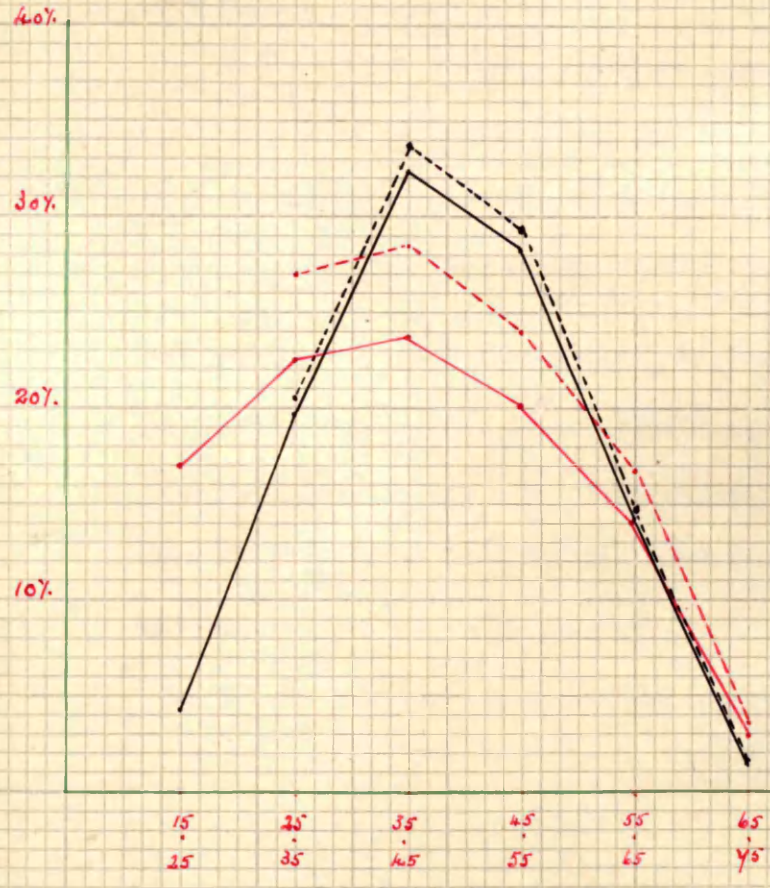
CASES 1919-1921

AGE at DATE of FIRST POSITIVE SPUTUM (T.B.+)

TO SHOW % DISTRIBUTION IN VARIOUS AGE GROUPS.

ALL CASES { GRINDERS ——— / CONTROLS ———

OMITTING ALL UNDER 25yrs. { GRINDERS - - - - - - / CONTROLS - - - - - -



No. of CASES in VARIOUS AGE GROUPS

AGE GROUPS	GRINDERS	CONTROLS	GRINDERS	CONTROLS
			OMITTING ALL under 25yrs.	
15-25	3	68		
25-35	14	90	14	90
35-45	23	95	23	95
45-55	20	80	20	80
55-65	10	56	10	56
65-75	1	12	1	12
75+	-	1	-	1
ALL AGES	71	402	68	334

% IN VARIOUS AGE GROUPS

AGE GROUPS	GRINDERS	CONTROLS	GRINDERS	CONTROLS
			OMITTING ALL under 25yrs.	
15-25	4.23	17.00		
25-35	19.74	22.50	20.58	27.00
35-45	32.43	23.75	33.81	28.50
45-55	28.20	20.00	29.40	24.00
55-65	14.10	14.00	14.70	16.80
65-75	1.41	3.00	1.47	3.60
75+	-	0.25	-	0.30
ALL AGES	ca 100	ca 100	ca 100	ca 100

N.B A RECORD of the AGE EXISTED only in 402 of the 409 CONTROLS.

The table and graphic representation which follow have been constructed from figures recently published by the Registrar General (38). Here a distinction is made between cutlery and metal grinders (the Sheffield Grinders are chiefly engaged in cutlery manufacture) and the figures prove beyond any shadow of doubt that the chief incidence of fatal pulmonary tuberculosis in these workers is between the ages of 45 and 55 years.

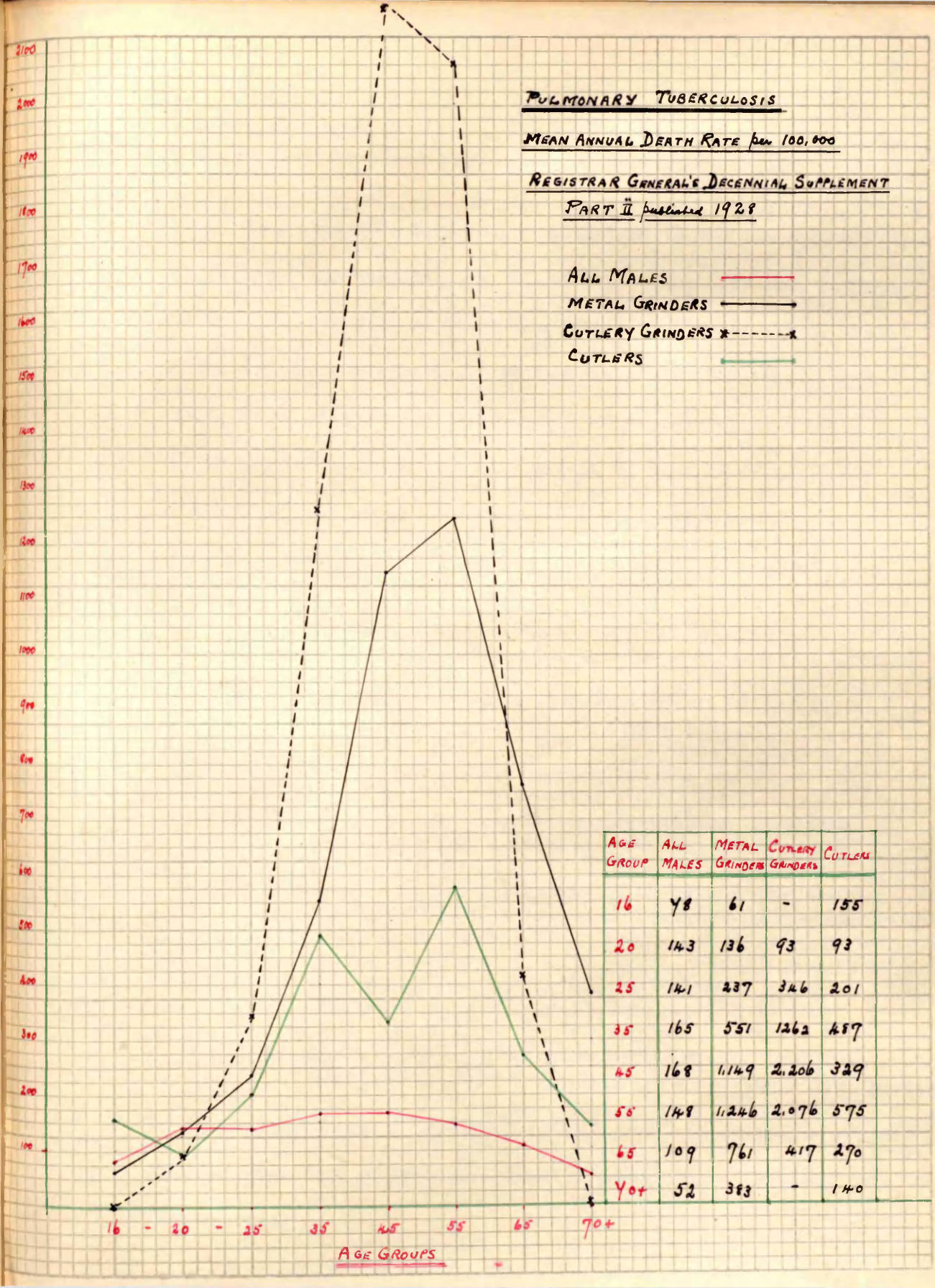
I have now adduced evidence from three sources :

(1) from aseries of fatal cases.

(2) from a series of living cases.

(3) from statistics for the whole country by the Registrar General,

and I feel justified in asserting that pulmonary tuberculosis, in Sheffield grinders, occurs most frequently inmiddle life onwards - between the ages of 45 and 55 years, (i.e.) this form of industrial tuberculosis conforms to Brownlee's middle age type.



AGE GROUP	ALL MALES	METAL GRINDERS	CUTLERY GRINDERS	CUTLERS
16	48	61	-	155
20	143	136	93	93
25	141	237	346	201
35	165	551	1262	487
45	168	1.149	2.206	329
55	148	1.246	2.076	575
65	109	761	417	270
70+	52	383	-	140

THE INFECTIVITY OF PULMONARY TUBERCULOSIS COMPLICATING SILICOSIS.

Many observers have called attention to the low infectivity of tuberculosis complicating silicosis. Wheatley (49) records that in the parishes of Clive and Grinshill of Shropshire, where there are quarries, half the stoneworkers die of phthisis, yet there is no excess of phthisis among women and young persons in the parishes. Collis (50) speaks of "the immunity from phthisis of the wives and widows of the flint knappers". Several explanations of this observation have been offered :

Nicholson (5) attributes it -

(a) to the fact that many cases are never tuberculous at all.

(b) to the rapid termination of open infectious cases, leaving but a short period for infecting contacts.

Wheatley (49) suggests the following reasons :

(1) Many of the cases returned as phthisis may have been non-tuberculous lithosis.

(2) Those cases in which infection with tubercle intervened may have been of a type with little breaking down of the lung and consequently little infection given off.

(3) The home conditions and general surroundings may be unfavourable to the spread of consumption.

(4) Consumption under fairly good conditions may have even a much less degree of infection than is generally supposed.

Cobbett (51) adds, "On the other hand there is reason to suppose that in this form of phthisis there is a scarcity of bacilli expectorated".

My experience does not lead me to agree with this last statement, for in about 1,500 consecutive sputum examinations

carried out at the Sheffield sanatorium, I found that tubercle bacilli were just as numerous and constant in cases of tuberculosis complicating silicosis as in other cases of tuberculosis.

With Nicholson's second explanation, of a short infective period, I cannot agree, for, in the foregoing pages, I have adduced evidence to show that, so far as the Sheffield metal-grinders are concerned, the infective period is just as long, if not longer, as in the average tuberculous individual.

On the incidence of tuberculosis among the wives and families of the grinders, I have no adequate data on which to base any conclusion, but I would point out that marital or conjugal infection - husband to wife - is very rare - even in tuberculosis without silicosis (Fishberg (52)).

Assuming the correctness of the views, expressed by the authors quoted, as to spread of infection to children, I think my investigations suggest a possible explanation not hitherto advanced. My statistics show that open pulmonary tuberculosis among metal grinders is predominantly a disease of middle life onwards (45 - 55 years). By this age, the majority of the children of the Grinders will have reached an age when that intimate contact with the infective person, which is so essential a factor in the spread of tuberculous infection, is highly improbable. The older ones will have married and have left home, while the younger ones will have reached an age that they require sleeping accommodation, apart from their parents.

OBSERVATIONS ON THE CLINICAL ASPECT OF SILICOSIS IN METAL GRINDERS.

(37 illustrative cases)

-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-o-

When one recalls the great variety of pathological changes, fibrosis, emphysema, areas of fibrotic consolidation, cavitation and irregular thickening of the pleura, which occur in the lungs following the inhalation of silica dust, it is little wonder that the diagnosis of silicosis still presents to the ablest physician a problem of extreme difficulty. And this difficulty is increased when he is required to determine the presence or absence of coincident tuberculosis.

Formerly a good deal of importance was attached to the history of exposure to dust over a prolonged period. But at best this could only be considered presumptive evidence of pneumonokoniosis, and it was not always known that such dusts, as coal and lime, were more or less innocuous. However, the symptomatology of silicosis and tubercular silicosis are now fairly well defined and by clinical examination of the chest, together with a good skiagram of the lungs, and repeated careful examination of the sputum for the tubercle bacillus, it is possible to arrive at a fairly accurate diagnosis.

From my investigations among the Sheffield metal grinders I have found that the symptoms and signs in these workers are identical with those occurring in other workers exposed to silica dust. Any difference which exists is entirely one of

degree and dependent on the intensity and duration of the exposure; well marked silicosis in the grinder requiring on the average about twenty years' occupation in the industry. Further, I amconvinced, that an accurate diagnosis of the degree of fibrosis, or the presence or absence of tuberculosis, cannot be made entirely by clinical examination but that a technically good skiagram of the chest is absolutely essential, and tuberculosis should never be excluded, until the sputum has been repeatedly examined for tubercle bacilli with negative result.

In South Africa silicosis has been divided into three clinical stages thus :- (29)

1. ante-primary - in which the earliest detectable physical signs are present but there are no roentgenological changes.

2. primary - definite physical signs are present; capacity for work is impaired though not seriously or permanently.

3. secondary - capacity for work is seriously and permanently impaired.

The recognition of the ante-primary stage is of extreme difficulty, but Sutherland and Bryson (53) in their report on silicosis in the Pottery Industry in England, indicate that they were able, in these workers, to diagnose fibrosis of the lungs ("fibrosis of the lungs includes silicosis") at a period of employment earlier than that at which silicosis has been found by radiological examination inthe same occupational groups.

Roentgenological stages corresponding to the clinical and pathological stages have been defined. In America the classification into three stages, as presented by Lanza and Childs (54) in 1917 is the one most usually adopted.

Stewart (55) in this country has described four stages, and these I have followed in the series of clinical cases appended.

1. Enlargement of root shadows with thickening of the trunk or "bronchial" shadows, and an increase in the linear markings towards the periphery of the lungs, giving rise to a reticulated appearance of the lung field.

2. The fine nodular stage. The skiagram shows mottling coarser than that of miliary tuberculosis, distributed uniformly over the lung fields.

3. The coarse nodular stage. The skiagram now shows a nodular mottling as in the previous stage but the mottling is coarser and the nodules are fewer in number.

4. The fourth and last stage is characterised by aggregation of the nodules, haziness round them, and large clouded areas irregularly distributed.

As to the diagnosis of complicating tuberculosis, there does not seem to be any definite agreement on the essential criteria, and, apart from the presence of tubercle bacilli in the sputum, it may be impossible to state whether it is present or not. In my experience, however, where one finds dense dulness over the upper portions of the lungs associated with an X-ray picture, which shows marked confluence of the shadows in these areas, the presumption of the presence of tuberculosis is very strong if not altogether definitive.

Judged by the subcutaneous tuberculin test I have found that practically 100 % of the grinders give evidence of tuberculous infection.

The following series of 37 cases indicates the salient clinical characteristics of silicosis and tubercular silicosis in the Sheffield metal-grinders, and at the same time serves to illustrate some of the features already described in the previous

sections.

The series is divisible into two groups -

(a) in cases I to XIV - tubercle bacilli were present in the sputum.

(b) in cases XV to XXXVII - no tubercle bacilli have ever been found in the sputum.

The early symptoms is a subject to which a gooddeal of attention has been paid and Table 13 below shews the earliest symptom and its frequency of occurrence in this series.

Table 13.

Earliest Symptoms.

Symptoms	T.B. Neg.	T.B.	Total	Percentage
Cough	7	5	12	32.4
Asthma	6	2	8	21.6
Haemoptysis	3	1	4	10.8
Pleurisy	2	1	3	8.1
Dyspnoea	1	1	2	5.4
Anorexia	1	1	2	5.4
Sore Throat	1	-	1	2.7
Hoarseness	-	1	1	2.7
Burning in Chest	-	1	1	2.7
Adenitis neck	1	-	1	2.7
Loss of Weight	-	1	1	2.7
Nil	1	-	1	2.7
	23	14	37	ca. 100.00

From this, cough appears as the outstanding initial symptom. Middleton (35) in his series of 57 cases found dyspnoea the most frequent, and I think there is no doubt that shortness of breath is the most dominant symptom of silicosis alone or complicated by tuberculosis, though it is not necessarily the one which first impresses the individual patient.

Haemoptysis occurred in several of the cases and varied from streaked sputum to frank haemorrhage. One man (Case XXI) coughed up 2½ pints of blood and recovered. Table 14 summarises the occurrence of this sign.

Table 14.

Haemoptysis.

The first sign in 4 cases.

	T.B. Neg.	T.B.	Total	Percentage
Streaked sputum	8	7	15	40.5
Frank haemorrhage	4	1	5	13.5
Nil	11	6	17	46.1
	23	14	37 ca..	100.00

Sputum.

The sputum in metal grinders is in newise pathognomonic. In the early stages of silicosis it is scant, mucoid, full of carbon particles and difficult to expectorate. On no occasion have I been able to identify particles of silica. In the later stages especially where tuberculosis has supervened it becomes abundant and purulent.

Dyspnoea.

At first this is slight and only noted on exertion, but as the fibrosis of the lungs advances the breathlessness becomes increasingly greater and in very advanced cases of silicosis it occurs even at rest in bed. The dyspnoea is associatedwith loss of expansion of the chest, and the breathing may ultimately be carried on entirely by the diaphragm and accessory muscles of

respiration. All writers on the subject emphasise this dyspnoea and suggest that it is due to the loss of eleasticity of the lungs resultant on the fibrosis. This undoubtedly is part of the explanation,but to my mind a factor of much greater importance is, that as the fibrosis is established, the lungs become less contractile and more voluminous and the chest has to expand to accommodate them. In this way the position of rest of the chest becomes one which is somewhere between the normal positions of expiration and inspiration. The more advanced the fibrosis the more nearly is the position of full inspiration approached. Consequently, the expansion of the chest becomes more and more restricted and pari passu the volume of "tidal air". Confirmation of this explanation can be noted at autopsy where as previously described the lungs are found to be large and rigid and do not recede from the chest wall when the thorax is opened.

In the actual clinical examination of the chest in these workers there are two points to which I would like to allude :

Percussion.

This should be carried out rapidly and the strength of stroke varied according to the individual case. The typical feature is a high-pitched resonance without actual flatness. This is more or less uniform over the whole chest and is accompanied by a feeling of marked resistance under the pleximeter finger. Collis (4) states that it is sometimes possible to detect isolated patches of dulness, but in my experience such is impossible. If the percussion note over the upper lobe tends to become flat and in this way

distinguishable from the high-pitched resonance of the remainder of the lungs, then, I think, one is amply justified in suspecting invasion by the tubercle bacillus.

Auscultation.

Middleton (35) states that "The breath sounds are frequently diminishedimintensity in various areas, especially at the bases posteriorly; they are typically of a high-pitched "whiffing" quality in more advanced cases and over some areas are broncho-vesicular". In my experience the characteristic feature is the extreme weakness of the respiratory murmur, and in many cases the expiratory portion is quite inaudible. Any change towards a bronchial or "whiffing" character - especially over the upper lobes - is again very suspicious of tuberculous invasion.

Finally it must be insisted that the diagnosis of silicosis or tubercular silicosis must be arrived at only after consideration of all the available evidence; occupation of the patient, the clinical symptoms and signs, a good skiagram and repeated examination of the sputum for tubercle bacilli. Furthermore, every case of so-called simple silicosis diagnosed must be kept under strict supervision as a potential case of open tubercular silicosis.

Among other diseases unduly prevalent in Sheffield metal-grinders Hall (22) mentions acute rheumatic fever. Accordingly, it is interesting to note that in the following 37 clinical cases no less than five (VI;XV;XVI;XVIII & XXXI) give a history of one or more attacks of this condition.

Collis (1) recently has associated Bright's disease with the 'silica dust risk'. In two of my cases (V. & IX) I found a faint

trace of albumin in the urine, but no other evidence of nephritis; and in case IX there was a definite history of renal colic due to a calculus..

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37 ILLUSTRATIVE CLINICAL CASES.

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Explanatory notes :

Occupation - Sandstone (W) = wet grinding
(D) = dry grinding

Sputum - Tubercle bacilli present 5:1 indicates an average of 5 tubercle bacilli per field - $\frac{1}{12}$ oil immersion.

X-ray report- Stage is according to definitions of Stewart quoted on p. 62.

T.A.F. - Tuberculin albumose free.
The subcutaneous method of test was followed.

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Case No. 1.

L.S. age 32 years.

Occupation. Table-blade grinder, sandstone, (W) 4½ years.

Social State. Married, abstainer, 15 cigarettes per day.

Family History. Father and three brothers - table-blade grinders - alive and well. No history of tuberculosis. One child - bronchial gland tuberculosis.

Previous History. Trench fever, fistula-in-ano (1918) and neurasthenia.

Present illness. Earliest symptom : dyspnoea on exertion.
Symptoms : Cough, occasional blood-streaked sputum, night sweats, anorexia, general lassitude, dyspnoea, loss of weight, and neurasthenia.

Physical examination. Ht. 5'6¼", Wt.opt. 12 st. 4 lbs., now 10 st.
Chest - expiration 36", inspiration 38".

Right lung. Resonance : dulness, over upper lobe, high-pitched note over remainder of lung, R.M. harsh vesicular accompanied by loud snoring rhonchus; weak vesicular at base.

Left lung. Resonance : slight dulness over left apex, especially behind, high-pitched over remainder of lung.
R.M. harsh vesicular.

Clinical diagnosis.
Chronic tuberculous infiltration of the right upper lobe and left apex. Industrial fibrosis.

Other systems. Haemorrhoids, otherwise normal.

Sputum. Tubercle bacilli present 5:1, (first time 27-7-26).

Urine. S.G. 1,023, acid, no abnormal constitutents.

Blood pressure. Systolic 142, diastolic 80.

X-ray report. Probable extensive disseminated tuberculosis both upper lobes, old and becoming definitely hard in places; some silicosis (stage I).

Remarks. Only short period of exposure to silica risk; presence of a tuberculous condition (fistula-in-ano) previous to his occupation in grinding industry; father and brothers grinders.

Case No. II.

F.P. age 52 years.

Occupation. Jobbing grinder, sandstone (W) 38 years.

Social State Married, practically abstainer, 2 ozs. tobacco per week.

Family History. No history of tuberculosis.

Previous History. "Colic".

Present illness. Earliest symptom : burning sensation in chest (October 1927). Symptoms : Cough at night, blood-streaked sputum (December 1927), occasional night sweats and dyspnoea on exertion.

Physical examination Ht. 5'5", Wt.opt. 11 st. 11 lbs., now 9 st. 9 lbs.
Chest - expiration 34", inspiration 35½".

Right lung. Resonance : marked dulness over upper lobe with high-pitched note over remainder of lung especially in axillary region. R.M. high-pitched bronchial with consonating rales and creaking rhonchi and whispering pectoriloquy over upper lobe; very weak vesicular, expiration almost inaudible in axillary region; puerile over remainder of lung.

Left lung. Resonance : slight dulness over apex with high-pitched note over remainder of lung.
R.M. vesiculo-bronchial with fine consonating rales in supraspinous fossa; puerile over remainder of lung.

Clinical diagnosis.
Chronic tuberculous infiltration of the right upper lobe, and early active infiltration of the left apex; silicosis both lungs with emphysema at both bases.

Other systems. Myocardial weakness, response to effort very poor; fundus oculi shows definite arterio-sclerosis.

Sputum. Tubercle bacilli present - isolated bacilli - (first time 11-11-2

Urine. S.G. 1,015, acid; no abnormal constitutents.

Blood pressure. Systolic 152, diastolic 94.

X-ray report. Tuberculous infiltration upper two-thirds of right lung, and left apex; moderately well marked silicosis both lungs (Stage 4).

Remarks. This patient was discharged from sanatorium but returned six weeks later. The tuberculous lesion had not advanced but signs of failing heart had appeared, viz., slight oedema of legs and orthopnoea.

Case No. III.

P.M. age 55 years.

Occupation. Sheep shear grinder, sandstone (W) 22 years.

Social State. Married, moderate beer drinker, 2 ozs. tobacco per week.

Family History. No history of tuberculosis.

Previous History. Never off work on account of illness.

Present illness. Earliest symptom : intractable cough (October 1927).
Symptoms : Cough all day, anorexia, loss of energy, dyspnoea on slight exertion, and loss of weight.

Physical examination. Ht. 5'1", Wt.Opt. 10 st. 5 lbs., now 8st. 8½ lbs.
Chest - expiration 32", inspiration 33¼".

Right lung. Resonance : dulness over upper lobe with slight heightening of pitch and increased resistance at right base.
R.M. rasping vesicular with prolonged expiration over upper lobe; vocal resonance increased, air entry to base very good.

Left lung. Resonance : dulness over upper lobe and almost normal over remainder of lung.
R.M. rasping vesicular over upper lobe. Vesicular with coarse bubbling rales over base especially behind.

Clinical diagnosis.
Chronic tuberculous infiltration both upper lobes; possible slight bronchiectasis at left base.

Other systems. No abnormality made out.

Sputum. Tubercle bacilli present - isolated bacilli, (first time 5-12-27)

Urine. S.G. 1,015, acid; no abnormal constitutents.

X-ray report. Tuberculous infiltration both apices especially right; silicosis (Stage 4).

Remarks. A case of tubercular silicosis. This man was very ill on admission to the sanatorium; recurrent attacks of faintness, orthopnoea, and cyanosis; after three months' sanatorium treatment he is now able to walk about comfortably for six hours each day.

Case No. IV.

J.H.W. age 35 years.

Occupation. Edge tool grinder, sandstone (W) 17 years.

Social State. Married, moderate beer drinker, 5 cigarettes per day.

Family History. Sister - in-law who lived at his home died of pulmonary tuberculosis (tubercle bacilli present in sputum). One child tuberculous caries of dorsal spine.

Previous History. Never off work on account of illness.

Present illness. Earliest symptom : intractable cough.
Symptoms : Cough all day, haemoptysis 10 ozs., night sweats, anorexia, fatigue, dyspnoea on slightest exertion, occasional complete aphonia.

Physical examination. Ht. 5' 7", Wt.opt. 9 st.. 5 lbs., now 9 st.. 2 lbs.
Chest - expiration 34", inspiration 35".

Right lung. Resonance : dulness over right upper lobe with very marked heightening of pitch and increased resistance at base; resonant zone just below root of lung behind.
R.M. harsh vesicular with prolonged expiration over upper lobe.

Left lung. Resonance : dulness over apex and extending outwards from the root behind.
R.M. vesicular over apex, bronchial with soft medium rales in root zone behind; very weak vesicular, expiration practically inaudible over base.

Clinical diagnosis.
Tuberculous infiltration both upper lobes with recent acute spread in left root zone; silicosis especially left base .

Other systems. Myocardial weakness, response to effort very poor.

Sputum. Tubercle bacilli present 10:1 (first time 28-8-26).

Urine. S.G. 1,025, neutral; no abnormal constitutents.

Blood pressure. Systolic 128, diastolic 84.

X-ray report. Tuberculous infiltration both upper lobes; extensive silicosis (Stage 4).

Remarks. Always considerably under average physical development; known contact with infectious case of pulmonary tuberculosis; one child suffering from tuberculous caries of spine.

Case No. V.

H.S. age 59 years.

Occupation. Scissors grinder, sandstone (W) 44 years.

Social state. Married, occasional glass of beer; non-smoker.

Family History. Father and one brother - scissors grinders - died of pulmonary tuberculosis (tubercle bacilli present in sputum in both instances).

Previous History. Facial erysipelas 1920.

Presnet illness. Earliest symptom : intractable cough.
Symptoms : cough, night sweats, anorexia, fatigue, constant dyspnoea, and loss of weight.

Physical examination. Ht. 5' 4", Wt.opt. 9st. 5 lbs., now 7st. 13 lbs.
Chest - expiration 29¼", inspiration 29¾".

Right lung. Resonance : slight dulness to second rib in front with very marked heightening of pitch and increased resistance to light percussion; dulness to root of scapula behind with high-pitched note in vault of axilla and over inferior angle of scapula.
R.M. bronchial over upper lobe with coarse consonating rales at sterno-clavicular joint; weak vesiculo-bronchial with creaking rhonchus at base.

Left lung. Resonance : impaired note to level of third rib in front with great heightening of pitch and increased resistance over remainder of lung.
R.M. bronchial over upper lobe with amphoric breathing in second and third interspaces in front (whispering pectoriloquy and bubbling rales in this area); weak vesicular with breaking rhonchi over base.

Clinical diagnosis.
Acute caseating tuberculosis of the left upper lobe, broncho-pneumonic consolidation of the right upper lobe, general silicosis with emphysema at the bases.

Other systems. Myocardial weakness, pulse rapid, regular, poor volume and tension; haemorrhoids.

Sputum. Tubercle bacilli present 20:1 (first time 17-7-27).

Urine. S.G.1,020, neutral, trace of albumen.

Blood pressure. Systolic 130, diastolic 78.

X-ray report. Extensive caseating tuberculosis both upper lobes, general silicosis with emphysema at both bases (Stage 2).

Died 5-8-27.

Remarks. Died suddenly of acute cardiac dilatation.
N.B. worked with father and brother (positive cases).

Case No. VI.

H.L. age 68 years.

Occupation — Saw grinder, sandstone, (W) 37 years.

Social State. — Married, alcoholic, non-smoker.

Family History. — No history of tuberculosis.

Previous History. — Rheumatic fever.

Present illness. — Earliest symptom : Persistent cough.
Symptoms : Cough, sputum contained clots of blood on several occasions, fatigue, dyspnoea on exertion, and loss of weight.

Physical examination. — Ht. 5'6", Wt.opt. 11 st.. 6 lbs., now 10 st.. 4½ lbs.
Chest :- expiration 34¾", inspiration 35¼".

Right lung. — Resonance : dull to level of third rib in front, and to middle of vertebral border of scapula behind. High-pitched over remainder of lung.
R.M. harsh vesicular with consonating rales over right upper lobe; very weak vesicular expiration inaudible, numerous creaking rhonchi over base.

Left lung. — Resonance : dull to level of second rib in front, and extreme apex behind.
R.M. harsh vesicular with prolonged expiration over apex, very weak with short expiration over remainder of lung.

Clinical diagnosis.
Chronic tuberculous infiltration of the right upper lobe and left apex; general silicosis both lungs with thickened pleura at right base.

Other systems. — Well marked arterio-sclerosis, arcus senilis, small cyst of thyroid gland.

Sputum. — Tubercle bacilli present 10:1 (first time 8-5-26).

Urine. — S.G. 1,018, acid, no abnormal constitutents.

Blood pressure. — Systolic 110, diastolic 70.

X-ray report. — Tuberculous infiltration of the right upper lobe, generalised coarse silicosis both lungs with thickened pleura over right base.(stage 4).

Remarks. — A case of tubercular silicosis (awarded compensation).
N.B. Early age - 14 years - at which he entered the occupation.

Case No. VII.

H.S. age 33 years.

Occupation. Table-blade grinder, sandstone (D) 13 years.

Social State. Married, occasional beer, 10 cigarettes per day.

Family History. No history of tuberculosis.

Previous History. "Rheumatic pains".

Present illness. Earliest symptom : Hoarseness (April 1927).
Symptoms : Cough, night sweats, anorexia, fatigue, hoarseness.

Physical examination. Ht. 5'1", Wt.opt. unknown, now 6 st.. 13 lbs.
Chest - expiration 31½", inspiration 32¾"

Right lung. Resonance : very marked dulness over right upper lobe with marked heightening of pitch over base posterially. R.M. tubular at right apex in front with coarse crackling rales, V.R. whispering pertoriloquy at apex in front, high-pitched bronchial over remainder of lung but very weak over base.

Left lung. Slight dulness over left apex, hyper-resonance over remainder of lung.
R.M. bronchial over upper lobe, harsh vewicular with prolonged expiration over remainder of lung.

Clinical diagnosis.
Sub-acute tuberculous infiltration of the right upper lobe, (cavity in right apex) and left apex. Industrial fibrosis (?) right base, emphysema of left lung.

Other systems. Tuberculous laryngitis, myocardial degeneration.

Sputum. Tubercle bacilli present 5:1 (first time 13-7-27).

Urine. S.G. 1,022, acid; no abnormal constitutents.

Blood pressure. Systolic 120, diastolic 74.

X-ray report. Extensive tuberculosis of the right upper lobe with acute spread in left root zone.

Died 8-10-27, acute progressive pulmonary tuberculosis.

Remarks. A case of pulmonary tuberculosis of acute type, no definite evidence of silicosis.

Case VIII.

A.W. age 42 years.

Occupation. Edge tool grinder, sandstone (W) 15 years; then carborundum wheel 10 years.

Social State. Married, occasional beer, 10 cigarettes per day.

Family History. One brother and two sisters probably died of pulmonary tuberculosis.

Previous History. Influenza 1918.

Present illness. Earliest symptom : pain over left upper lobe.
Symptoms : cough, occasional blood-streaked sputum for past two years, night sweats, fatigue, slight loss of weight.

Physical examination. Ht. 5'6", Wt. opt. 10 sts. 4 lbs., now 9 sts. 13 lbs.
Chest - expiration 36¾", inspiration 38".

Right lung. Resonance : dull over right upper lobe especially in front and in axilla, slightly high-pitched over remainder of lung.
R.M. harsh vesicular over upper lobe and weak vesicular over base.

Left lung. Resonance : very slight impairment apex, otherwise normal.
R.M. puerile all over.

Clinical diagnosis.
Tuberculous infiltration of the right upper lobe and left apex, silicosis at right base.

Other systems. No abnormality made out.

Sputum. Tubercle bacilli present, isolated bacilli (first time 1-6-27).

Urine. S.G. 1,012, acid; no abnormal constitutents.

Blood pressure. Systolic 144, diastolic 86.

X-ray report. General silicosis (stage 3) with acute spread of tuberculosis in left root zone.

Remarks. Bad family history; tuberculosis did not appear till ten years after he had given up work on sandstone (silica risk); on discharge from sanatorium he returned to his usual work in the grinding hull.

Case No. IX.

A.F. age 36 years.

Occupation. Machine-file grinder, sandstone (W) 15 years.

Social State. Married, occasional beer, 8 cigarettes per day.

Family History. No history of tuberculosis.

Previous History. Accident - amputation of left arm 1907.
Renal calculus (?).

Present illness. Earliest symptom : rapid loss of weight. (September 1926).
Symptoms : cough, occasional blood-streaked sputum, night sweats, anoraxia, fatigue, dyspnoea on slight exertion, loss of weight, hoarseness.

Physical examination. Ht. 5'6", Wt.opt. 11 sts., now 9 sts. 7 lbs.
Chest - expiration 34½"; inspiration 35¼".

Right lung. Resonance : dull over apex and root, slight heightening of pitch over rest of lung.
R.M. bronchial over right upper lobe with increased V.R. and V.F., weak vesicular over base.

Left lung. Resonance : dull over apex and root with definite heightening of pitch over rest of lung and more marked than on right side.
R.M. harsh vesicular with prolonged expiration over apex and root, very weak vesicular over remainder of lung.

Clinical diagnosis.
Chronic tuberculous infiltration of both apices, more extensive on right side; general industrial fibrosis of both lungs expecially left.

Other systems. No abnormality made out.

Sputum. Tubercle bacilli present 5:5 (first time 25-10-26).

Urine. S.G.1,020, acid; faint trace of albumen.

Blood pressure. Systolic 130, diastolic 70.

X-ray report. Tuberculous infiltration of the right upper lobe, general fibrosis both lungs, silicosis (stage 1).

Died 21-1-28. Progressive pulmonary tuberculosis and tuberculous laryngitis.

Remarks. This case ran the usual course of a case of sub-acute pulmonary tuberculosis with severe tuberculous laryngitis and tuberculous enteritis in the terminal stage.

Case No. X.

F.G.T. age 52 years.

Occupation. Table-blade grinder, sandstone (W) 17 years.

Social State. Married, alcoholic, half ounce tobacco per week.

Family History. No history of tuberculosis, one daughter is at present under supervision as a suspected case.

Previous History. Never off work on account of illness.

Present illness. Earliest symptom : persistentcough (December 1926).
Symptoms : cough, fatigue, and dyspnoea on the slightest exertion.

Physical examination. Ht. 5'10¼", Wt.opt. 10 stn. 11 lbs., now 10 stn. 7 lbs.
Chest - expiration 35½", inspiration 36".(barrel shaped).
Right lung. Resonance : marked dulness over right upper lobe with slight heightening of pitch over remainder of lung.
R.M. moderately high-pitched bronchial over right upper lobe, very feeble vesicular over base.
Left lung. Resonance : slight dulness apex and root with definite heightening of pitch over rest of lung especially towards base.
R.M. harsh vesicular over apex otherwise weak vesicular.

Clinical diagnosis.
Fibroid tuberculosis of the right upper lobe, with chronic infiltration of left apex; generalised silicosis.

Other systems. Arterio-sclerosis and pyorrhoea alveolaris.

Sputum. Tubercle bacilli present 20:1 (first time 26-5-27).

Urine. S.G.1,020, acid; no abnormal constitutents.

Blood pressure. Systolic 148, diastolic 90.

X-ray report. Tuberculous infiltration of the right upper lobe, acute spread of tuberculosis in left root zone, slight silicosis (stage 3).

Remarks. Case of tubercular silicosis. On discharge from sanatorium this man returned to his usual work; his son who lived in the same house worked with him.

Case No. XI.

F.S. age 37 years.

Occupation. Table-blade grinder, sandstone (W) 20 years.

Social State. Married, occasional beer, 30 cigarettes per day.

Family History. No history of tuberculosis.

Previous History. No history oftuberculosis.

Previous History. No illness.

Present illness. Earliest symptom : complete loss of energy since 1923. Symptoms : cough, occasional blood-streaked sputum, night sweats, fatigue, severe dyspnoea, loss of weight, and hoarseness.

Physical examination. Ht. 5' 2½", Wt.opt. 9 st., now 6 st. 13 lbs.
Chest - expiration 30½", inspiration 31½".

Right lung. Resonance : slight dulness over upper lobe with slight heightening of pitch and increased resistance at base.
R.M. bronchial over upper lobe, weak vesicular at base.

Left lung. Almost wooden dulness over upper lobe with some impairment at the base.
R.M. high-pitched bronchial over upper lobe with numerous mucous rales; very weak vesicular at base.

Clincial diagnosis.
Disseminated tuberculosis whole of right lung, with tuberculous infiltration of the left upper lobe, some fibrosis at both bases.

Other systems. Great loss of cardiac reserve (pulse 140 per min. regular, of poor volume and tension); tubercular laryngitis.

Sputum. Tubercle bacilli present 10:1 (first time 16-10-25).

Urine. S.G. 1,020, acid; no abnormal constitutents.

Blood pressure. Systolic 106, diastolic 70.

X-ray report. Extensive tuberculosis both lungs with moderately well marked silicosis (Stage 3).

Died 5-7-27.

Remarks. This patient ran the usual course of a case of progressive sub-acute pulmonary tuberculosis.

Case No. XII.

T.H.P. age 22 years.

Occupation. Magnet grinder, emery wheel (D) 1½ years.

Social State. Unmarried, occasional beer, 10 cigarettes per day.

Family History. Mother died pulmonary tuberculosis 1927, tubercle bacilli present in sputum.

Previous History. Bronchitis, scarlet fever.

Present illness. Earliest symptom : loss of appetite.
Symptoms : cough, frequent blood-stained sputum, night sweats, fatigue, severe dyspnoea on exertion, anorexia, loss of weight, hoarseness.

Physical examination. Ht. 5'8", Wt. opt. 8 st. 10 lbs., now 8 st. 6 lbs.
Chest - expiration 29½", inspiration 31½" (alar type).
Right lung. Resonance : slight dulness over whole lung.
R.M. puerile all over.
Left lung. Resonance : slight impairment all over lung.
R.M. weak bronchial with occasional consonating rales in supra-spinous fossa; elsewhere harsh vesicular.

Clinical diagnosis.
Disseminated tuberculosis of both lungs, with some reparative fibrosis.

Other systems. Loss of cardiac reserve.

Sputum. Tubercle bacilli present 10:1 (first time 3-4-26).

Urine. S.G. 1,014, acid; no abnormal constitutents.

Blood pressure. Systolic 122, diastolic 76.

X-ray report. Extensive tuberculosis both lungs; no evidence of silicosis.

Died 14-4-28.

Remarks. This patient ran the usual course of tuberculosis in a young adult, and was in the sanatorium for over one year before death.
N.B. Very poor physical development; mother died of pulmonary tuberculosis; short duration of occupation and not employed on sandstone.

Case No. XIII.

W.L. age 36 years.

Occupation. Crank grinder, emery wheel (W) 3 years.

Social State. Married, occasional beer, 10 cigarettes per day.

Family History. No history of tuberculosis.

Previous History. Recurrent colds.

Present illness. Earliest symptoms : loss of energy (April 1927).
Symptoms : cough, night sweats, fatigue.

Physical examination. Ht. 5'6½", Wt.opt. 9 st. 10 lbs., now 9 st. 4 lbs.
Chest - expiration 33", inspiration 34¼".

Right lung. Resonance : dulness over right upper lobe, with slight heightening of pitch and increased resistance at the base. R.M. vesicular with prolonged expiration over upper lobe; weak vesicular at the base.

Left lung. Resonance : dulness over root and apex, well marked behind. R.M. harsh vesicular over apex and very weak vesicular over remainder of lung.

Clinical diagnosis.
Chronic tuberculous infiltration of the right upper lobe, and left apex.

Other systems. Catarrhal deafness, pyrrhoea alveolaris.

Sputum. Tubercle bacilli present, isolated bacilli (first time 9-7-27).

Urine. S.G. 1,020, acid; no abnormal constitutents.

Blood pressure. Systolic 132, diastolic 78.

X-ray report. Definite tuberculous infiltration of the right upper lobe, disseminated tuberculosis left axillary region, no evidence of silicosis.

Remarks. This man is suffering from definite tuberculosis of the lungs, and is in the infectious state. The nature of the grinding on which he is employed is not likely to induce silicosis except after a very long period of employment. On discharge from Sanatorium he returned immediately to his former place of employment.

Case No. XIV.

G.H.C. age 22 years.

Occpuation. Agricultural implements grinder, emery wheel (B) 5 years.

Social State. Unmarried, occasional beer, 10 cigarettes per day.

Family History. No history of tuberculosis.

Previous History. Influenzal attack and recurrent pleurisy.

Present illness. Earliest symptom : pleurisy (May 1927).
Symptoms : cough, night sweats, fatigue, dyspnoea, loss of weight and recurrent loss of voice.

Physical examination. Ht. 5'8"; Wt.opt. unknown, now 8 stn. 6½ lbs.
Chest - expiration 30½", inspiration 32" (alar chest).

Right lung. Resonance : moderate dulness over upper lobe.
R.M. vesiculo-bronchial with consonating rales over upper lobe.

Left lung. Resonance : dulness over upper lobe.
R.M. soft vesiculo-bronchial with consonating rales over the upper lobe.

Clinical diagnosis.
Chronic tuberculous infiltration both upper lobes.

Other systems. Failing cardiac reserve. Myopia.

Sputum. Tubercle bacilli present 20:1 (first time 23-6-27).

Urine. S.G.1,017, alkaline; no abnormal constitutents.

Blood-pressure. Systolic 98, diastolic 70.

X-ray report. Disseminated tuberculosis of the left lungand right upper lobe; no evidence of silicosis.

Died 13-11-27 (at home).

Remarks. A case of primary pulmonary tuberculosis; the nature of his work and the short duration of employment exclude the possibility of silicosis.

Abbreviated notes on 23 additional cases.
Repeated examination of the sputum for tubercle bacilli was made in each case with negative result.
All these men are still alive, and the age and duration of occupation refer to the year 1927.

Case No. XV.

B.W. age 60 years.

Occupation. Pen and pocket knife grinder, sandstone (W) 23 years, emery stone 15 years.

Family History. Two brothers (grinders) died of pulmonary tuberculosis (tubercle bacilli in sputum).

Previous History. Rhuematic fever, acute pneumonia.

Earliest symptom. Intractable cough.

Clinical diagnosis. Fibroid tuberculosis of the right upper lobe, generalised industrial fibrosis. T.A.F. .005 (focal).

X-ray report. Fibroid collapse of the right lung, general fibrosis, silicosis (stage 1).

Remarks. Bad family history of pulmonary tuberculosis; started work in grinding hull at age of 10 years.

Case No. XVI.

W.C. age 39 years.

Occupation. File grinder, sandstone (W) 9 years.

Family History. Father and brother (file grinders) healthy.

Previous History. Rheumatic fever.

Earliest symptom. Blood-streaked sputum.

Clinical diagnosis. Old tuberculous lesion of the right apex (?); T.A.F. .005 (focal).

X-ray report. Old tuberculous lesion right apex, slight fibrosis, silicosis (stage 1).

Case No. XVII.

T.D. age 54 years.

Occupation. Table-blade grinder, sandstone (W) 36 years.

Family History. No history of pulmonary tuberculosis.

Previous History. Bronchitis.

Earliest symptom. Intractable cough.

Clinical diagnosis. Fibro-indurative collapse (tuberculous) of the left upper lobe; tuberculous infiltration of the right apex; definite silicosis. T.A.F. .001 (focal).

X-ray report. Silicosis both lungs; extensive tuberculosis left lung and right apex. (stage 4).

Case No. XVIII.

E.T. age 57 years.

Occupation. Razor grinder, sandstone (W & D) 23 years.
emery stone (W) 10 years.

Family History. No history of pulmonary tuberculosis.

Previous History. Rheumatic fever - two attacks.

Earliest symptom. Intractable cough.

Clinical diagnosis. Old tuberculous infiltration of the left upper lobe, silicosis (?). T.A.F. .005 (general).

X-ray report. Early silicosis; old tuberculosis of the left upper lobe (stage 2).

Case No. XIX.

S.B. 18 years.

Occupation. Scraper grinder, sandstone (D) 8 months.

Family History. No history of pulmonary tuberculosis.

Previous History. Bronchitis since childhood.

Earliest symptom. Sore throat.

Clinical diagnosis. Old tuberculous infiltration of both apices; T.A.F. .01 (focal

X-ray report. Old root and possible apical tuberculosis; slight general fibrosis.

Remarks. Tuberculosis definitely present and apparently preceding his occupation of dry grinding. The fibrosis is probably non-industrial but associated with chronic bronchitis.

Case No. XX.

H.M. age 56 years.

Occupation. Razor grinder, sandstone (W & D) 30 years.
emery stone (W) 10 years.

Family History. No history of pulmonary tuberculosis.

Previous History. Inguinal hernia; pleurisy right side (1921).

Earliest Symptom. Loss of strength.

Clinical diagnosis. Fibro-indurative tuberculosis of the left upper lobe, and old infiltration right ap ex; early industrial fibrosis. T.A.F. .005 (focal).

X-ray report. Early silicosis both lungs; tuberculous infiltration of the right upper lobe and the left apex, compensatory emphysema both bases.

Case No. XXI.

A.E. age 37 years.

Occupation. Joiners'-tool grinder, sandstone (W) 14 years.

Family History. Father joiners'-tool grinder died pulmonary tuberculosis (tubercle bacilli in sputum)

Previous History. Acute pneumonia (1913); gassed (chlorine gas 1916).

Earliest symptom. Coughed up clot of blood (August 1926).

Clinical diagnosis. Chronic tuberculous infiltration both apices, especially left. T.A.F. .001 (intense local and general reactions;focal

X-ray report. Old tuberculous infiltration both apices; early fibrosis, silicosis (stage 1).

Remarks. Followed his fathers' occupation. Father was an infectious case. On one occasion had haemoptysis 2½ pints in all.

Case No. XXII.

T.S. age 47 years.

Occupation. Butcher-blade grinder, sandstone (W) 30 years.

Family History. No history of pulmonary tuberculosis.

Previous History. Major epilepsy.

Earliest symptom. Persistent cough.

Clinical diagnosis. Possible old tuberculous infiltration of the right apex; well marked silicosis. T.A.F. .001 (focal at right apex).

X-ray report. General silicosis both lungs; tuberculous infiltration right apex (?)(stage 3).

Case No. XXIII.

A.B. age 66 years.

Occupation. Saw grinder, sandstone (W) 30 years.

Family History. No history of pulmonary tuberculosis.

Previous History. Typhoid fever (1891); double pneumonia (1909).

Earliest symptom. Intractable cough.

Clinical diagnosis. Chronic tuberculous infiltration both upper lobes; well marked arterio-sclerosis. T.A.F. .01 (focal) right upper lobe.

X-ray report. Old tuberculous infiltration of the right upper lobe, and left apex; no silicosis.

Case No. XXIV.

G.S. age 43 years.

Occupation. Table blade grinder, sandstone (W) 31 years.

Family History. Father, fork grinder, died aged 57n(bronchitis), no history of pulmonary tuberculosis.

Previous History. Influenza, suspected duodenal ulcer (1925).

Earliest symptom. Abdominal pain, and indigestion.

Clinical diagnosis. Tuberculous infiltration of the left upper lobe and right apex, silicosis; thickened pleura at the right base. T.A.F. .001 (focal) right upper lobe.

X-ray report. Tuberculous infiltration of the left upper lobe, early silicosis thickened pleura right base; emphysema left base (stage 1).

Case No. XXV.

G.W. age 38 years.

Occupation. Table blade grinder, sandstone (W) 10 years.

Family History. No history of pulmonary tuberculosis.

Previous History. Dislocated semi-lunar cartilage.

Earliest symptom. Fatigue.

Clinical diagnosis. Definite tuberculous infiltration of the right apex, general fibrosis. T.A.F. .003 (focal) right apex.

X-ray report. Tuberculous infiltration both apices, expecially right; general fibrosis (stage 1).

Remarks. This man was awarded compensation for silicosis under Silicosis Order of 1927.

Case No. XXVI.

J.W.E. age 23 years.

Occupation. Edge tool grinder, sandstone (W) 7 years.

Family History. Father edge tool grinder (see skiagram No 7) - extensive silicosis; no history of pulmonary tuberculosis.

Previous History. Measles.

Earliest symptom. No complaint.

Clinical diagnosis. Lupus vulgaris face; chronic tuberculous infiltration both apices; slight genorall fibrosis. T.A.F. .001 (focal right apex; lupus patch became very inflamed)

X-ray report. Definite tuberculous infiltration both apices.

Remarks. A tuberculous condition present in this case previous to occupation of grinder1 (vide lupus vulgaris). The tuberculous lesion of lungs was diagnosed during a routine examination of the men at his place of employment.

Case No. XXVII.

W.H. age 47 years.

Occupation Table blade grinder, sandstone (W) 33 years.

Family History. No history of pulmonary tuberculosis.

Previous History. Gonorrhoea (1907).

Earliest Symptom. Sudden attack of breathlessness.

Clinical diagnosis. Tertiary syphilis (leukoplakia linguae, aortitis, and external strabismus left eye); general fibrosis of the lungs; no definite evidence of tuberculosis. T.A.F. .001 (slight focal at right apex); W.R. strongly positive.

X-ray report. General fibrosis of lungs; silicosis (stage 1). Broadening of shadow of mediastinum.

Case No. XXVIII.

R.D. age 29 years.

Occupation. Table blade grinder, sandstone (W) 11 years.
emery stone (W) 1 year.

Family History. No history of pulmonary tuberculosis.

Previous History. Constantly attending doctor; "Run down"; pleurisy (1925).

Earliest symptom. Haemoptysis - about 16 ozs. of blood.

Clinical diagnosis. Tuberculous infiltration of both apices, especially left; Early general fibrosis. T.A.F. .005 (local, general and focal reactions very severe).

X-ray report. Both lungs show fine discrete mottling throughout; disseminated tuberculosis (?); silicosis (?).

Remarks. The clinical findings in this case suggest tuberculosis father than silicosis. The period of employment in grinding is too short to produce discrete stage of silicosis.

Case No. XXIX.

W.W. age 39 years.

Occupation. Table blade grinder, sandstone (W) 16 years.

Family History. No history of tuberculosis.

Previous History. Occasional cold.

Earliest symptom. Pleurisy right side.

Clinical diagnosis. General fibrosis of lungs; thickened pleura at right base. T.A.F. .01 (slight general reaction).

X-ray report. Possible malignant infiltration from right root to base.

Died 13-6-29. Certificate : Lympho-sarcoma of right lung.

Remarks. Post-mortem was not allowed, and in the light of present knowledg of intra-thoracic neoplasm, the condition was probably a primary carcinoma of right lower bronchus. See skiagram No. 16.

Case No. XXX.

H.B. age 33 years.

Occupation Table blade grinder, sandstone (W) 12 years.

Family History. Father and brother, table blade grinders, healthy. No history oftuberculosis.

Previous History. Trench Fever; gassed (mustard gas).

Earliest symptom. Fatigue.

Clinical diagnosis. Tuberculous infiltration of the right apex; slight fibrosis. T.A.F. .005 (very severe general reaction focal at right apex).

X-ray report. Early general fibrosis (stage 1); definite tuberculous infiltration of the right apex.

Case No. XXXI.

H.D. age 51 years.

Occupation. Scissors grinder, sandstone (W) 29 years.

Family History. Brother died pulmonary tuberculosis 1927 (tubercle bacilli present in sputum).

Previous History. Rheumatic fever.

Earliest symptom. Intractable cough.

Clinical diagnosis. Tuberculous infiltration of the right upper lobe; general silicosis.

X-ray report. Silicosis both lungs (stage 4); tuberculous infiltration of the right upper lobe and left apex.

Remarks. This man was suffering from alcoholic hypertrophic cirrhosis of the liver. Whilst in sanatorium he developed delirium tremens and was transferred to a general hospital where he died of cardiac failure and cirrhosis of the liver.

Case No. XXXII.

W.M. age 56 years.

Occupation. Razor grinder, sandstone (W) 34 years.
(Has not worked since 1920)

Family History. No history of pulmonary tuberculosis.

Previous History. Influenza.

Earliest symptom. Pain between the shoulders (1926).

Clinical diagnosis. General fibrosis of both lungs, with emphysema left base. T.A.F. .001 (focal right apex).

X-ray report. General fibrosis of both lungs, silicosis (stage 1).

Remarks. Note long period of grinding followed by long period of unemployment. Discrete stage of silicosis not present.

Case No. XXXIII.

M.B. age 64 years.

Occupation. Jobbing grinder, sandstone (W) 49 years.

Family History. No history of pulmonary tuberculosis.

Previous History. Lumbago, alchholic neuritis.

Earliest symptom. Pleurisy (August 1927).

Clinical diagnosis. Tuberculous infiltration of the left upper lobe and right apex; general silicosis. T.A.F. not done; clinical signs suggest that sputum ought to contain tubercle bacilli.

X-ray report. Silicosis (stage 4) and tuberculosis both lungs.

Case No. XXXIV.

L.E.S. age 44 years.

Occupation. Engineers' tools grinder, emery stone (D) 8 years.

Family History. Two children under observation for tuberculosis.

Previous History. Tuberculosis was suspected nine years ago (previous to his becoming a grinder).

Earliest symptom. Fatigue.

Clinical diagnosis. Old tuberculous lesion both apices.

X-ray report. Old fibroid tuberculosis right apex; general fibrosis, silicosis (stage 1).

Remarks. Tuberculosis suspected previous to his occupation as a grinder.

Case No. XXXV.

T.A. age 19 years.

Occupation. Augur grinder, emery stone (D) 6 months.

Family History. No history of tuberculosis.

Previous History. Influenza 1925 - off work 15 weeks; always anaemic.

Earliest symptom. Swelling on right side of neck.

Clinical diagnosis. Caseating tuberculous gland right side of neck; early tuberculous infiltration both apices, especially right. T.A.F. .001 (focal right apex).

X-ray report. Tuberculous infiltration both apices; no evidence of silicosis.

Remarks. Definite tuberculosis present within six months' of work as grinder. Never employed on sandstone.

Case No. XXXVI.

G.G. age 21 years.

Occupation. Edge tool grinder, sandstone (W) 7 years.

Family History. Mother alive, pulmonary tuberculosis (tubercle bacilli in sputum); maternal uncle died pulmonary tuberculosis; father and brother, grinders, healthy.

Previous History. No illness.

Earliest symptom. Fatigue.

Clinical diagnosis. Early tuberculous infiltration both apices; slight general fibrosis. T.A.F. .001 (focal both apices).

Remarks. (1) Definite case of tuberculosis.
(2) Mother andinfectious case.
(3) Whole family engaged in grinding.

Case No. XXXVII.

D.M. age 32 years.

Occupation. Engineers' tools grinder, emery stone (W) 10 years.

Family History. No history of tuberculosis.

Previous History. Gun shot wound of chest.

Earliest symptom. Fatigue.

Clinical diagnosis. Chronic tuberculous infiltration both apices.
T.A.F. .005 (severe general reaction focal at right apex).

X-ray report. Tuberculous infiltration right apex, no evidence of silicosis.

Remarks. Definite tuberculosis; whole period of occupation on emery stone.

SUMMARY.

Diseases of the lungs due to the inhalation of dust and characterised by cough, increasing breathlessness and wasting or phthisis have been recorded since the 5th century B.C. At first many kinds of dust were believed to be equally injurious in their effects on the lungs, but in the light of present knowledge, the sole offending agent is recognised to be free crystalline silica in a finely divided state. This when inhaled into the lungs gives rise to fibrosis (silicosis), the degree of which varies with the intensity and duration of exposure to the dust. Furthermore, this type of fibrosis seems peculiarly liable to complication by pulmonary tuberculosis. The mode of action of the silica is not wholly understood, but at present the majority of workers in this field adhere to the hypothesis of Gye and Kettle, who assert that the silica forms a sol which acts as a direct cell poison. Thereby an area of coagulation necrosis results, in which the tubercle bacilli are protected from phagocytosis and so can multiply rapidly with impunity.

That tuberculosis complicating silicosis is of an unusually rapid and fatal type is the recorded opinion of a great many writers on the subject. Metal grinders in the Sheffield cutlery industry suffer from silicosis, the silica being derived from the sandstone or gritstone wheels on which they work. Tuberculosis also is exceedingly common among them.

With a view to throwing some light on the problem of silicosis and tubercular silicosis, these conditions have been investigated among the Sheffield metal-grinders. Data have been assembled concerning 310 fatal cases of tuberculosis in metal grinders, and, as controls, 1,361

fatal cases among males engaged in other occupations in Sheffield. A further series of living cases - 71 grinders and 409 controls - have also been analysed.

The following aspects of the "Silica Dust" problem have been examined and analysed :

a. Mortality rate of pulmonary tuberculosis among metal-grinders at the present day.

b. Review of mortality rate of pulmonary tuberculosis among metal-grinders for the last 40 years (1886 - 1925).

c. Mean age at death.

d. Average "Fatality" period.

e. Average "Morbidity" period.

In Table 15 is set forth a summary of the main facts established.

Table 15.

	Group	No. of cases	Mean age at death	Average "Fatality" period	Average "Morbidity" period
	All Grinders	310	46 yrs.	110 weeks	135 weeks
	Controls	1,361	39 "	97 "	126 "
	Grinders - age 25 years and over	294	47 "	113 "	138 "
	Controls - age 25 years and over	1,133	42 "	105 "	134 "
(A)	Tubercular-silicosis (clinical cases)	44	50 "	106 "	131 "
(B)	Tubercular-silicosis (X-ray cases)	36	52 "	83 "	115 "
	(A) plus (B)	80	51 "	96 "	123 "

CONCLUSIONS.

1. The Mortality rate of pulmonary tuberculosis among metal-grinders is still greatly in excess of that in the general population.

2. No marked improvement in Mortality rate of pulmonary tuberculosis in metal-grinders has occurred in the last 40 years.

3. Pulmonary tuberculosis in metal-grinders is divisible into two main groups :
   (a) Natural group
   (b) Industrial group.

4. The "industrial group" comprises the cases of true tubercular silicosis.

5. Pulmonary tuberculosis in metal-grinders, industrial group, is a disease of late middle life and belongs to Brownlee's middle age type.

6. The course of pulmonary tuberculosis in metal-grinders, as judged by average "Fatality" and "Morbidity" periods, is not more rapid than is the case in the average individual.

APPLICATION.

I have suggested, that pulmonary tuberculosis still exacts an excessive toll of lives in the Sheffield metal-grinding industry, and that no appreciable improvement has occurred since 1886. I have put forward the hypothesis of two groups of cases, natural and industrial. In addition, I have shewn that the duration of the infectious stage ("Fatality" period) is not of shorter duration than in the average individual.

I believe that pulmonary tuberculosis is due to the tubercle bacillus and that it is spread by infection, as a result of repeated close contact with infectious cases or infectious material.

If these contentions are substantially true, then if possible,

we must concentrate, not on the silica dust but on the tubercle bacillus.

The natural group must be eliminated from the industry by careful initial and periodical medical examination of the workers, assisted in every case by skilled X-ray examination. No infectious case must be allowed to remain in or return to the industry as is permitted at present. Was this the gospel of a prophet crying in the wilderness ? -

In conclusion I would urge the great importance of making strict regulations against uncontrolled spitting in workshops, and elsewhere, and of insisting on regular medical examination of the workers so that suspicious cases and, at any rate, patients with tubercle bacilli in their sputum should at once be removed from the workshop, or from such working conditions in which they are likely to be a source of infection to their fellows, for I believe that unless something of this nature is done, all the elaborate precautions to minimise the amount of dust are only touching the fringe of the whole question. (8)

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ADDENDUM.

In the British Medical Journal of Spetember 14th, 1929, two articles on **Silicosis** are printed.

1. "The Present Position of Silicosis in Industry in Britain"

by

E. L. Middleton, M.D.,D.P.H.,
H.M. Medical Inspector of Factories.

2. "Some Notes on the Biophysics of Silica and the Etiology of Silicosis"

by

Patrick Heffernan, M.D.,
Tuberculosis Officer, Derbyshire County Council.

A leading article by Sir T. M. Legge, in the same issue, is devoted to a review of the points raised in these papers. It is interesting to note in this "leader" the statement that -

> "The problem of Silicosis in South Africa is now different from ours; the stages we have to traverse have been passed..... The excessive use of water is disadvantageous, moreover, facilitating the entry of the tubercle bacillus, and this constitutes the main problem in that country to-day."

This is essentially the thesis which I have tried to sustain in connection with the Sheffield metal-grinders. To-day tuberculosis rather than silicosis is the main problem of the grinding industry.

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A SERIES OF ILLUSTRATIVE SKIAGRAMS.

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1. Normal lungs.
2. Acute miliary tuberculosis of lungs.
3. Healed miliary tuberculosis of lungs.
4. Simple silicosis (Stage 3) in metal-grinder.
5. Simple silicosis (Stage 3) in stonemason.
6. Tubercular silicosis in South African gold-miner.

Natural group.

7. Early tuberculosis in metal-grinder.
8. Extensive tuberculosis in metal-grinder.
9. Extensive tuberculosis in metal-grinder.

Industrial group.

10. Tubercular silicosis (Stage 1) in metal-grinder.
11. Do. (Stage 1) do.
12. Do. (Stage 2) do.
13. Do. (Stage 3) do.
14. Do. (Stage 4) do.
15. Do. (Stage 4) do.
16. Primary malignant growth of right lung in metal grinder.

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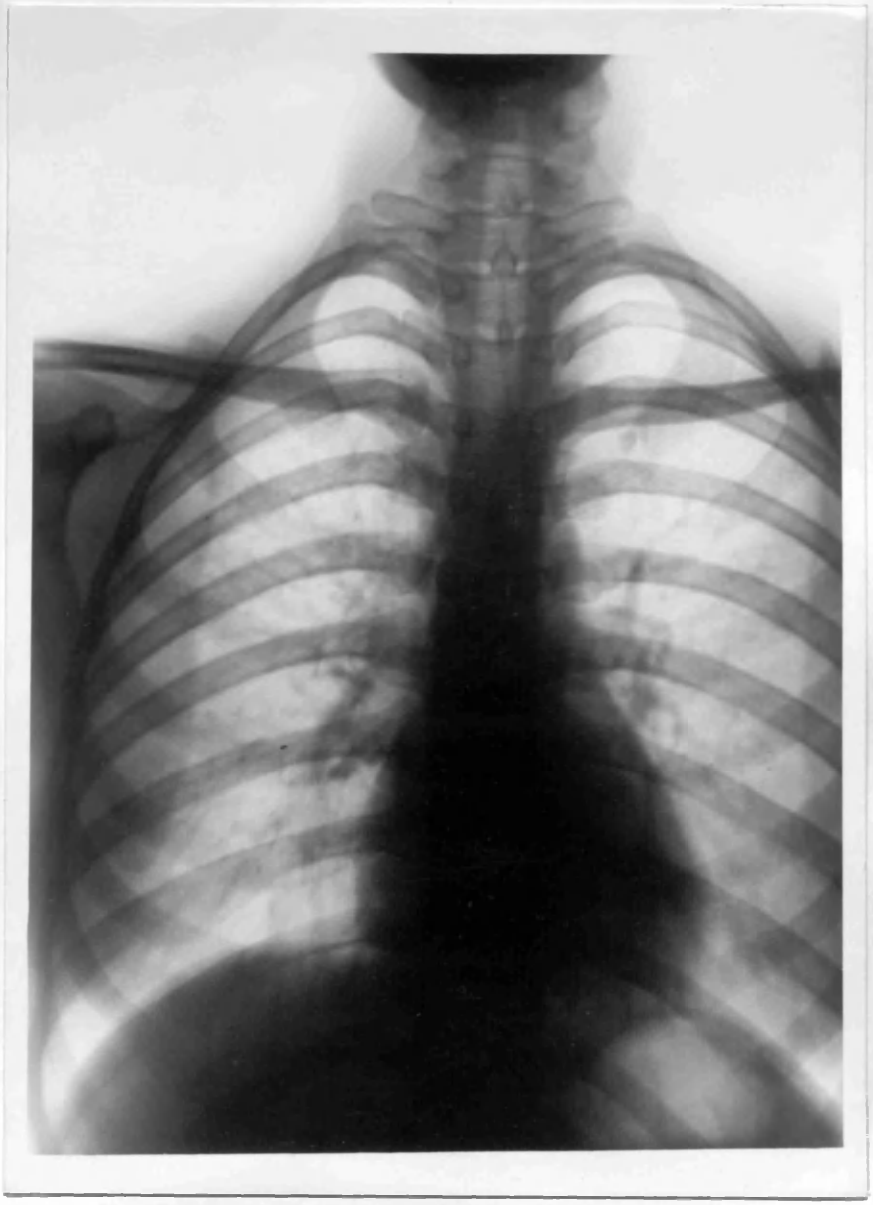
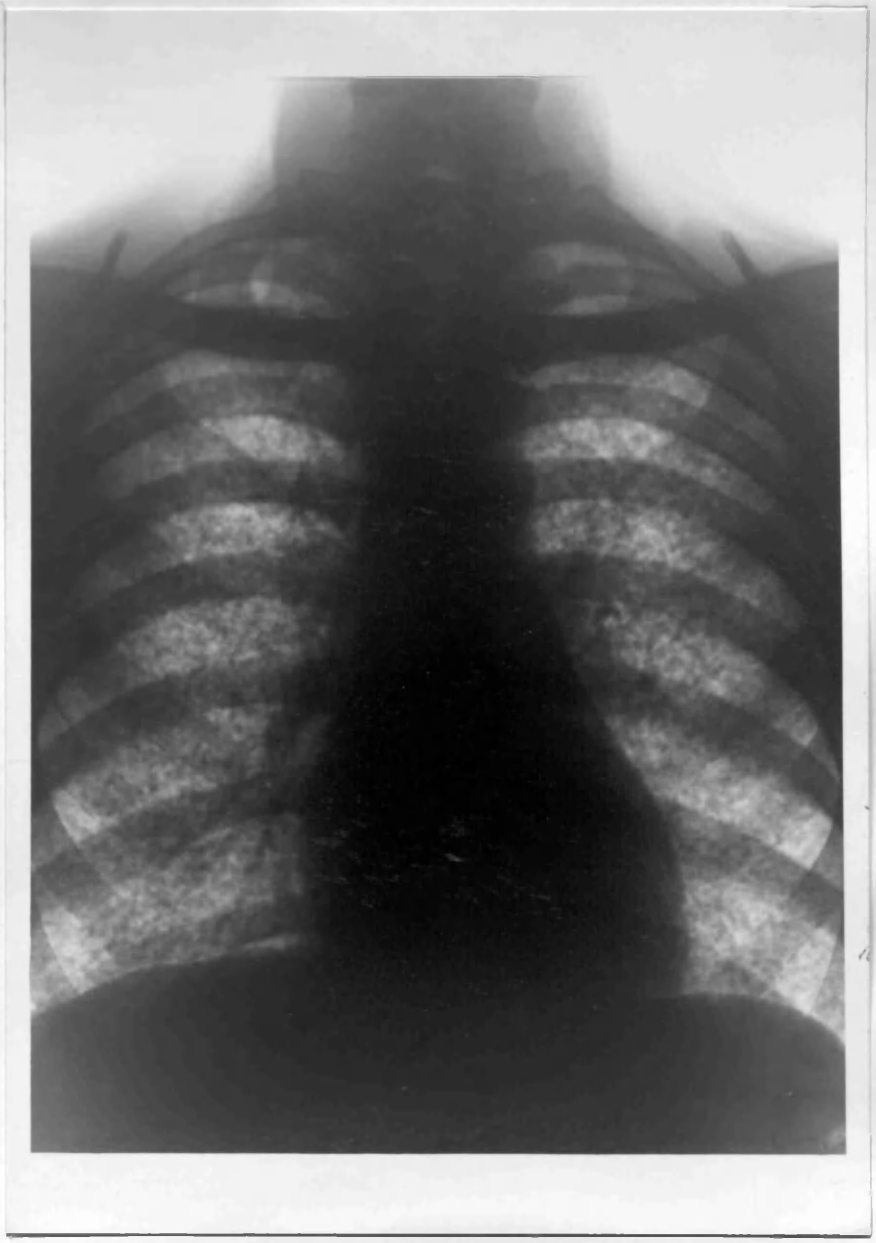
No. 1.



M.E. age 24 years Nurse.

Normal lungs.

No. 2.

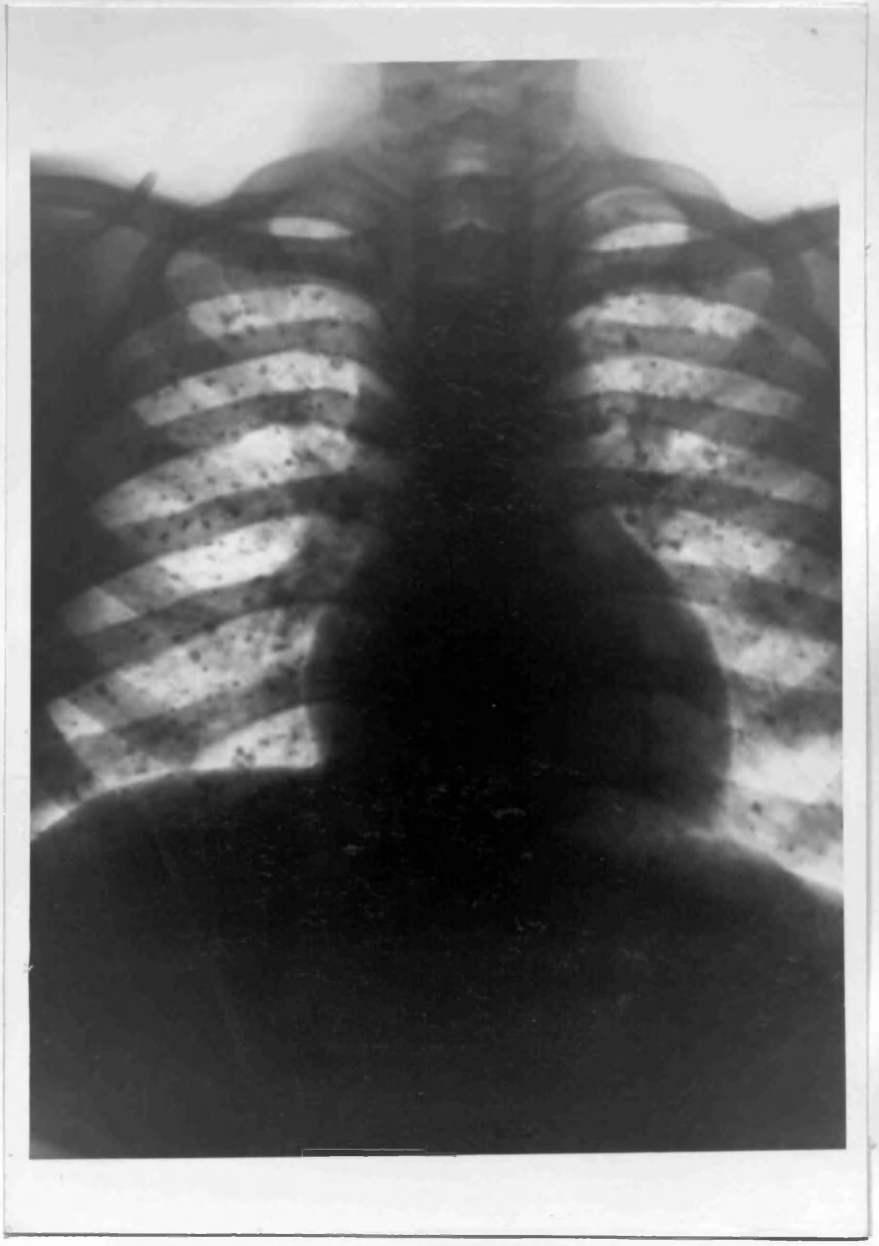


J.E. age 19 years. Acute Miliary Tuberculosis;

confirmed at autopsy.

Remarks : Note the extreme fineness of the mottling and its uniform distribution throughout both lungs.

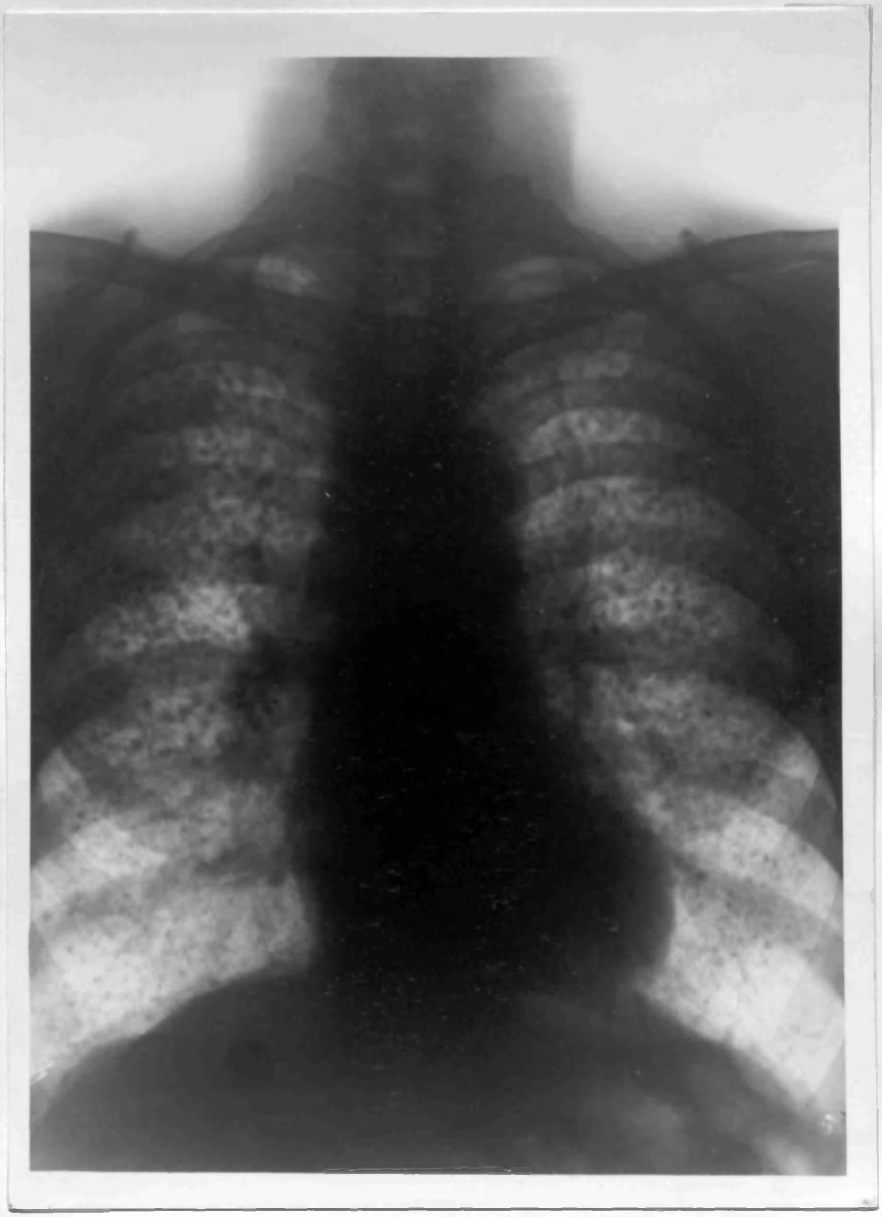
No. 3.



W.F. age 6 years. Healed miliary tuberculosis of lungs.

Remarks : This case was discovered by accident when child was being X-rayed for Pott's disease of lumbar spine. Note the extreme density of nodules and absence of fibrosis in remaining parts of lung fields. Patient is still alive and well, now aged 16 years.

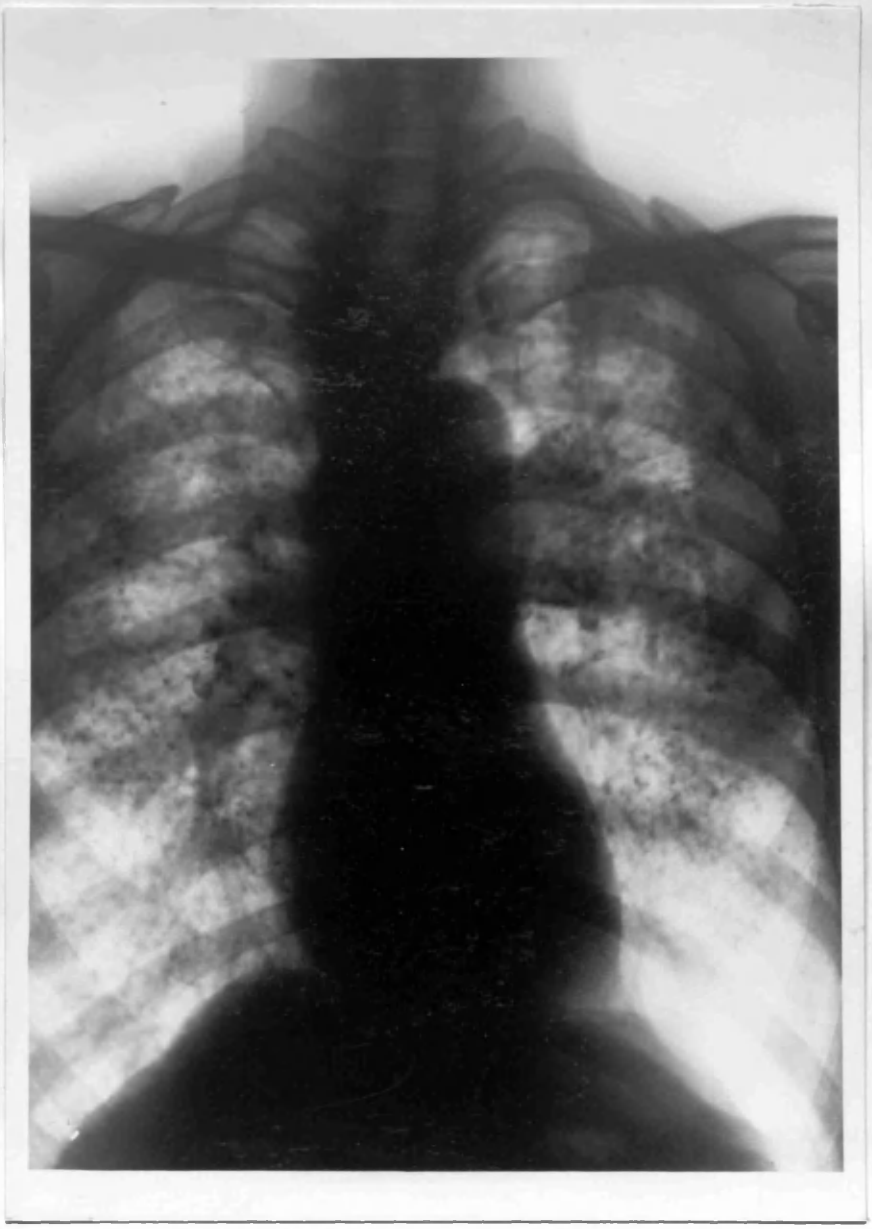
No. 4.



W.E. age 44 years. Edge tool grinder - sandstone (W) 30 years.

Silicosis (stage 3).

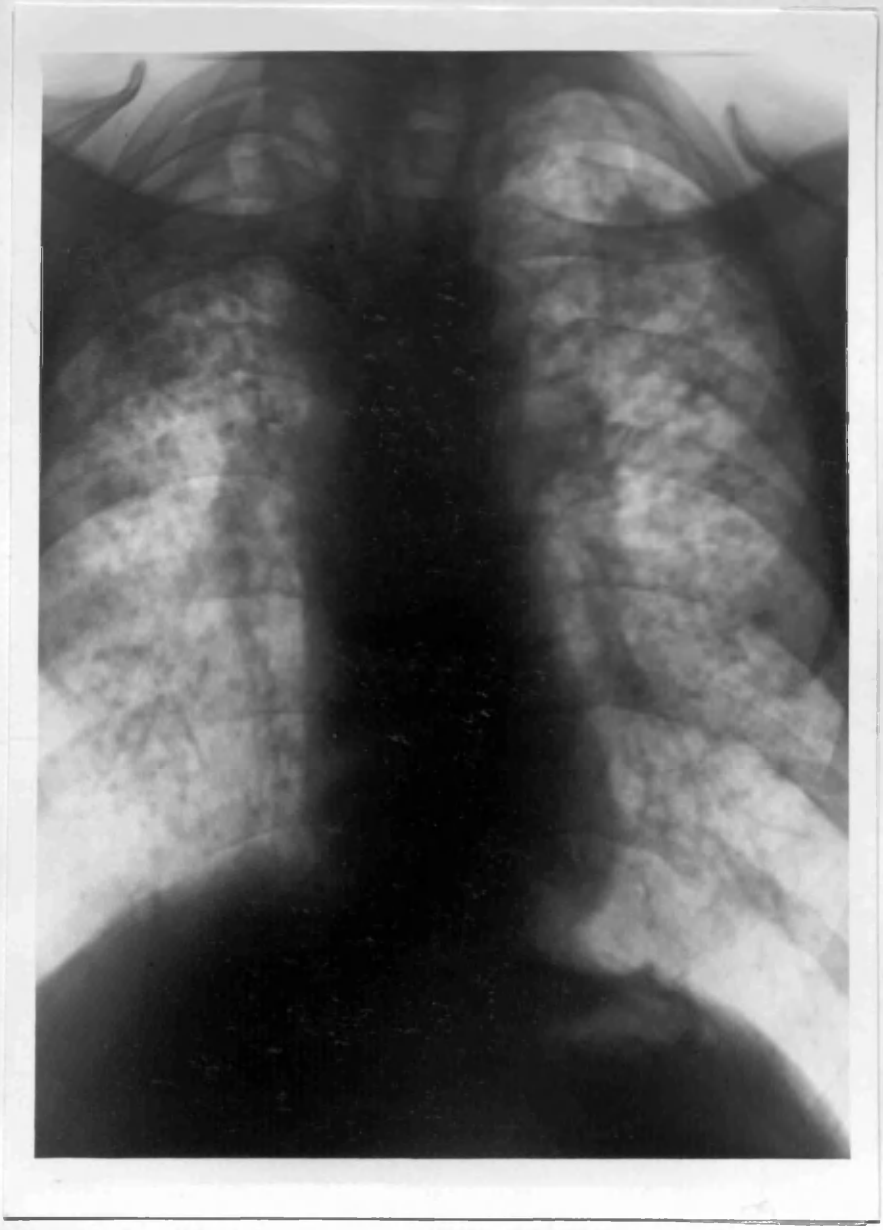
No. 5.



I.E. age 65 years. Stonemason 45 years.

Silicosis (stage 3)

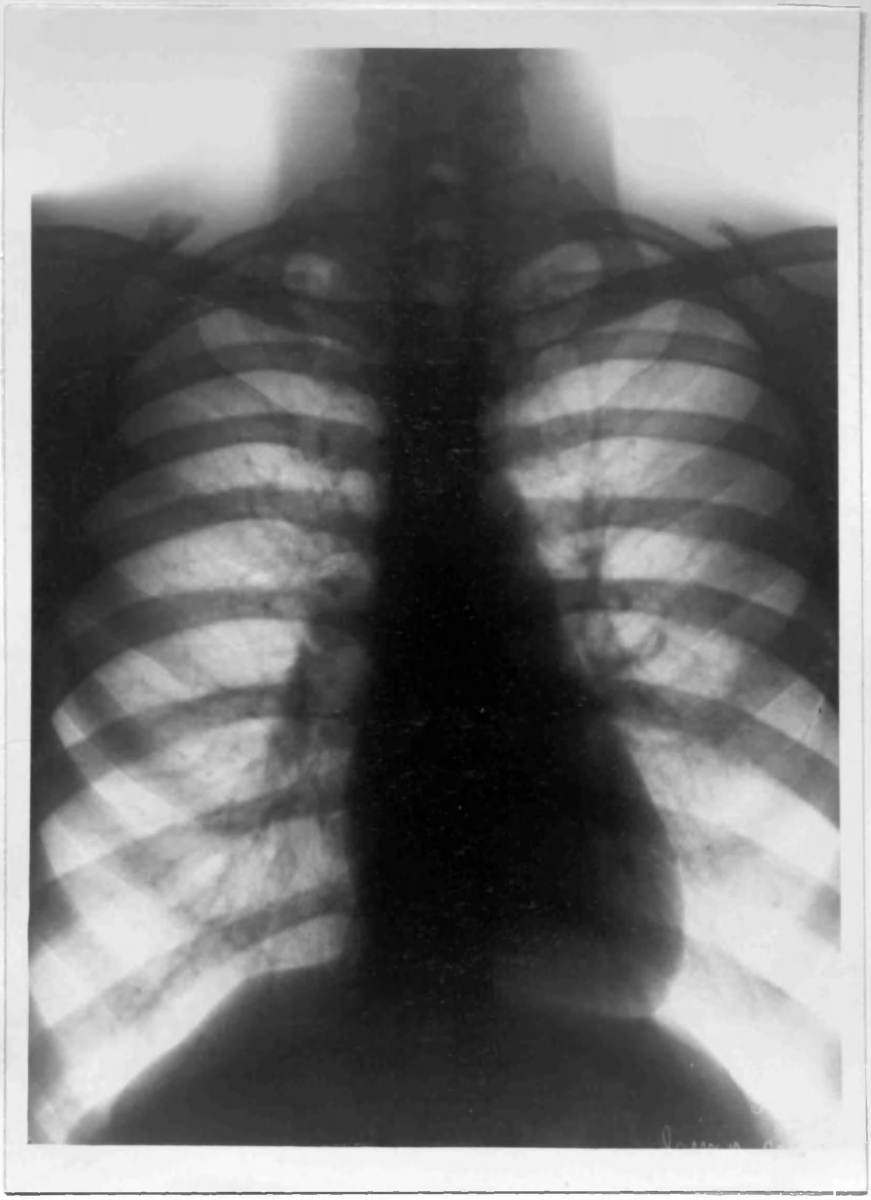
No. 6.



J.G.H. age 50 years. Gold miner (South Africa) 15 years.

Tubercular silicosis.

No. 7.



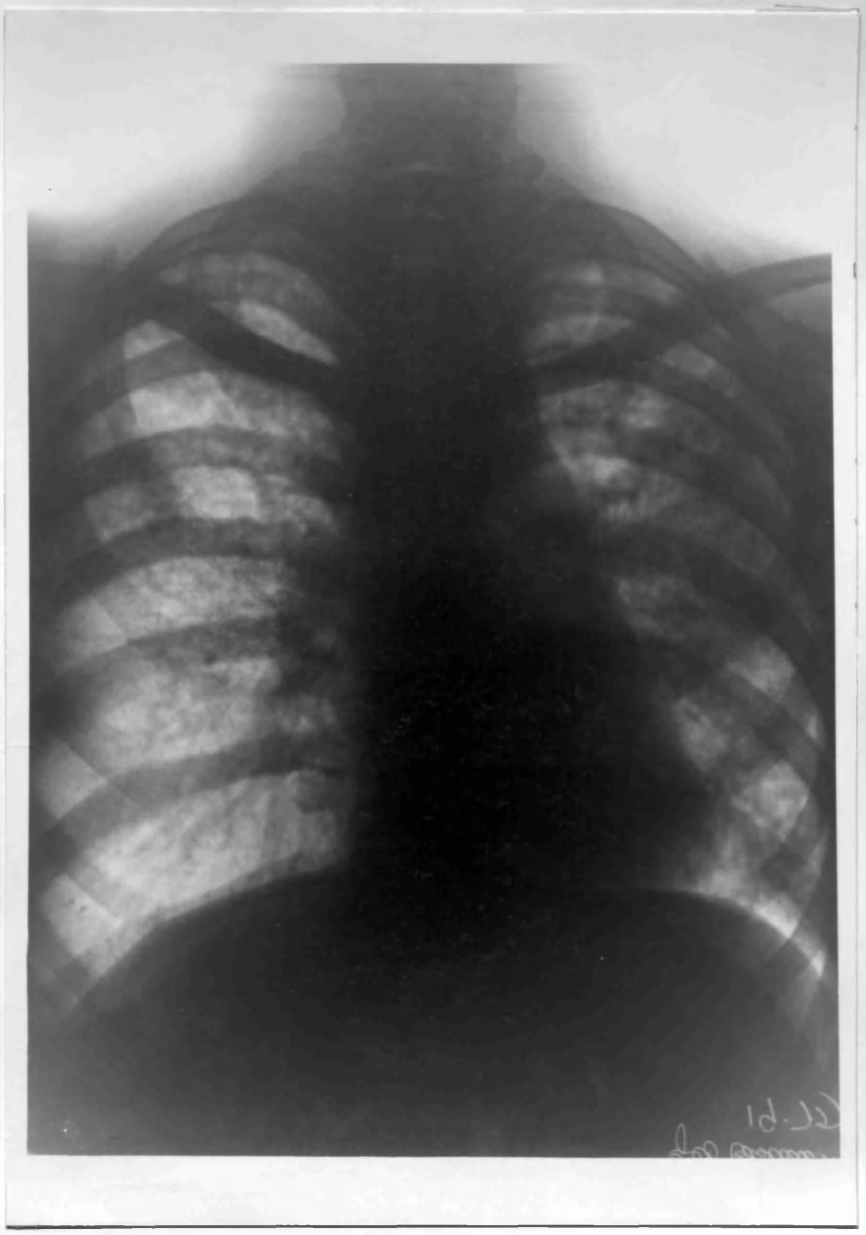
Case XXVI.

J.W.E. age 23 years. Edge tool grinder, sandstone (W) 7 years.

No evidence of silicosis.

Remarks : Definite tuberculous infiltration of left apex.

No. 8.



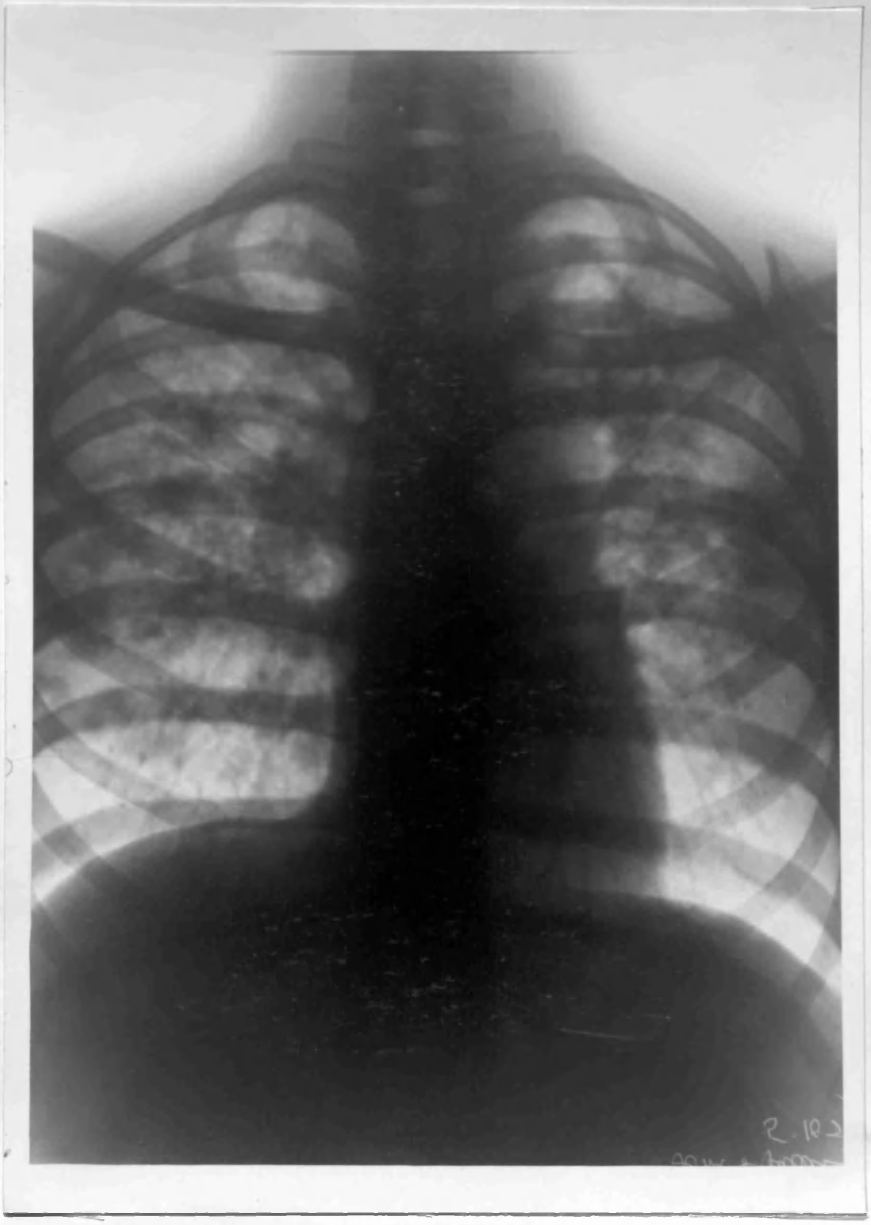
Case No. XIV.

G.C. age 22 years. Agricultural implements grinder; emery wheel (D) 5 years.

No evidence of silicosis.

Remarks : Extensive tuberculosis of left lung and right upper lobe.

No. 9.



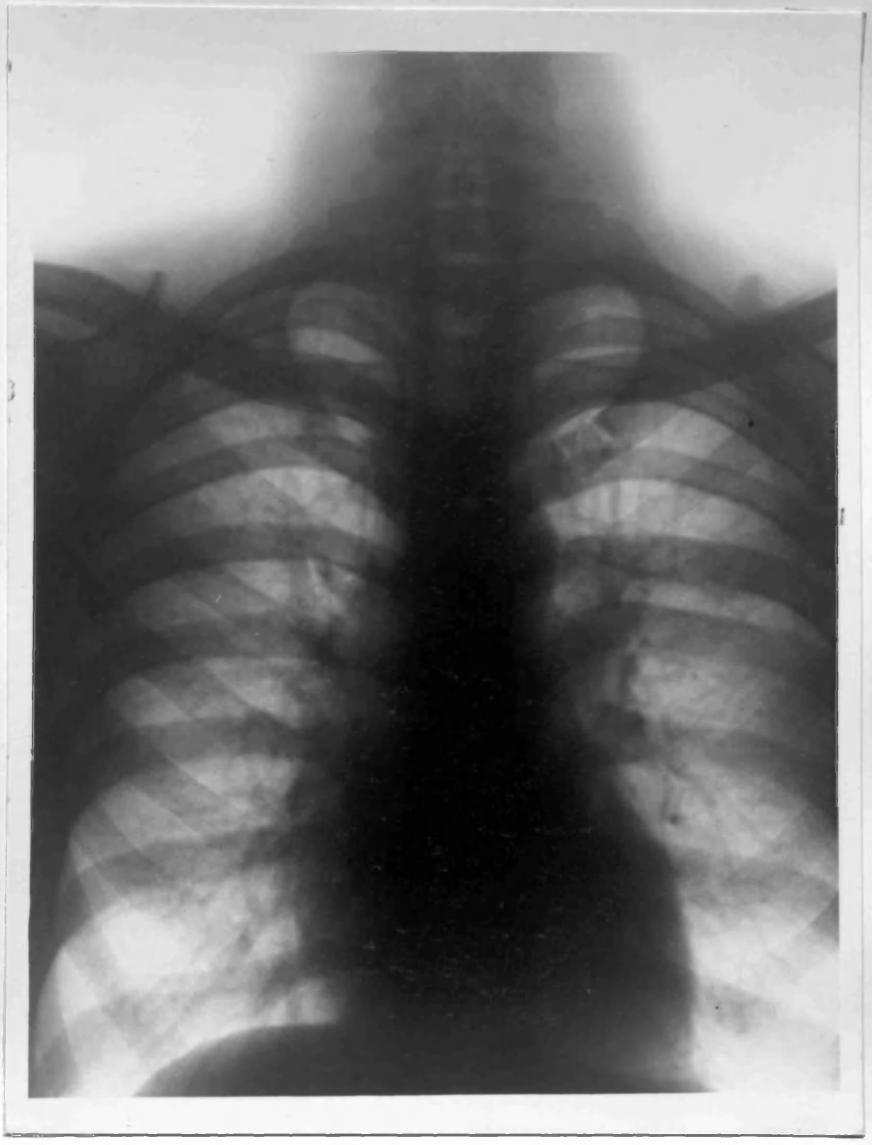
Case No. XII.

T.H.P. age 22 years. Magnet grinder; emery wheel (D) 1½ years.

No evidence of silicosis.

Remarks : Acute tuberculous infiltration of both lungs.

No. 10.

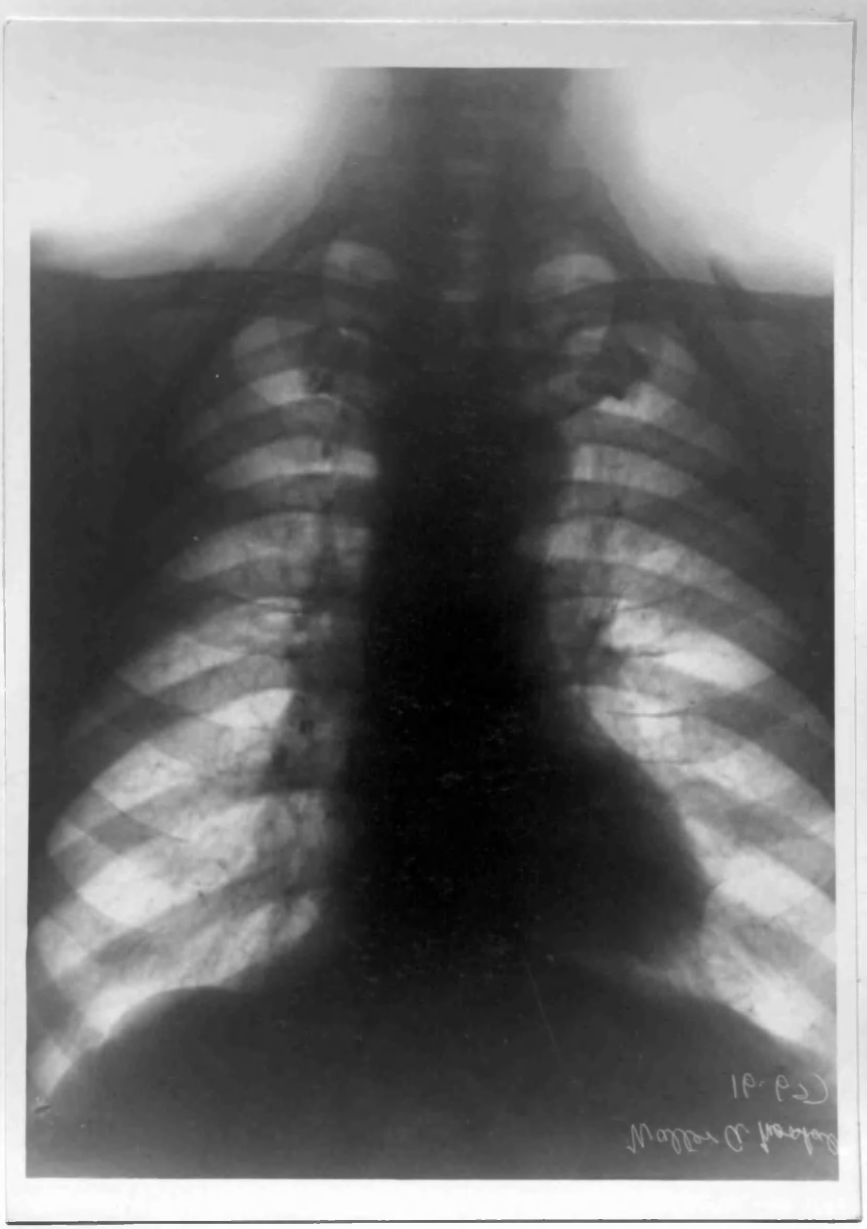


Case No. XXV.

G.H.T. age 33 years. Table blade grinder, sandstone (W) 10 years.

Silicosis (stage 1)

No. 11.



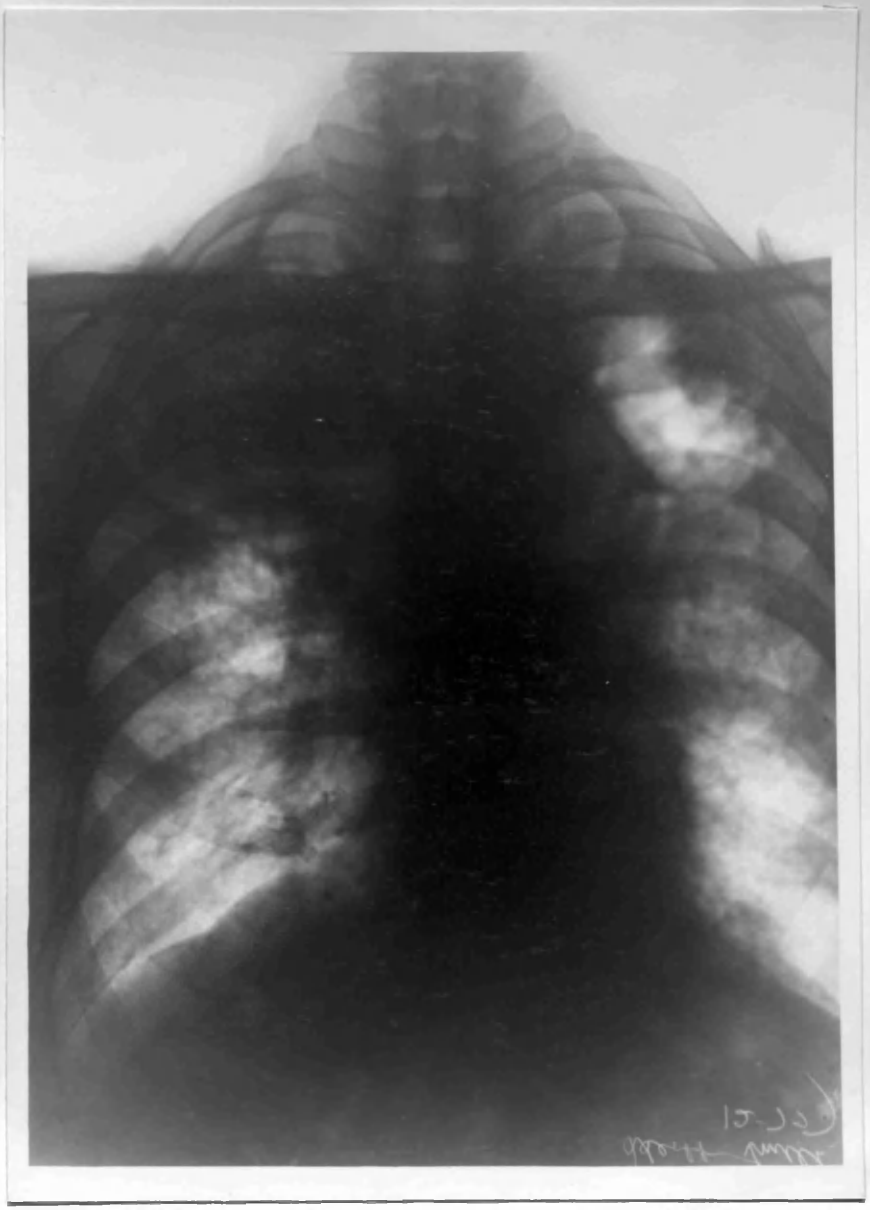
Case No. XXXIII.

W.M. age 56 years. Razor grinder, sandstone (W) 34 years.

Silicosis (stage 1).

Remarks : Note long period of exposure to "silica risk".

No. 12.



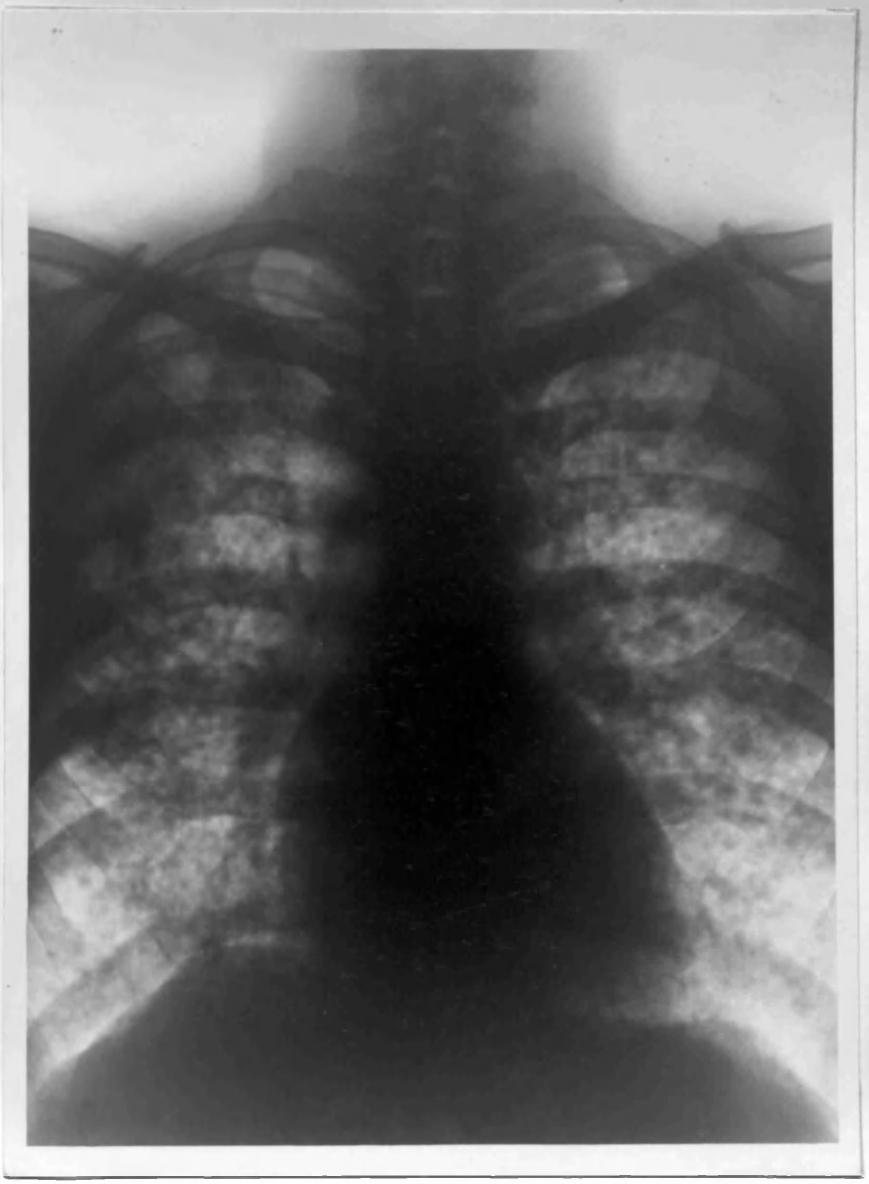
Case No. V.

H.S. age 59 years. Scissors grinder, sandstone (W) 44 years.

Silicosis both bases (stage 2).

Remarks : Note dense confluent shadows in subapical regions on both sides with extensive cavitation on left side. This suggests tuberculosis rather than confluent silicosis.

No. 13.

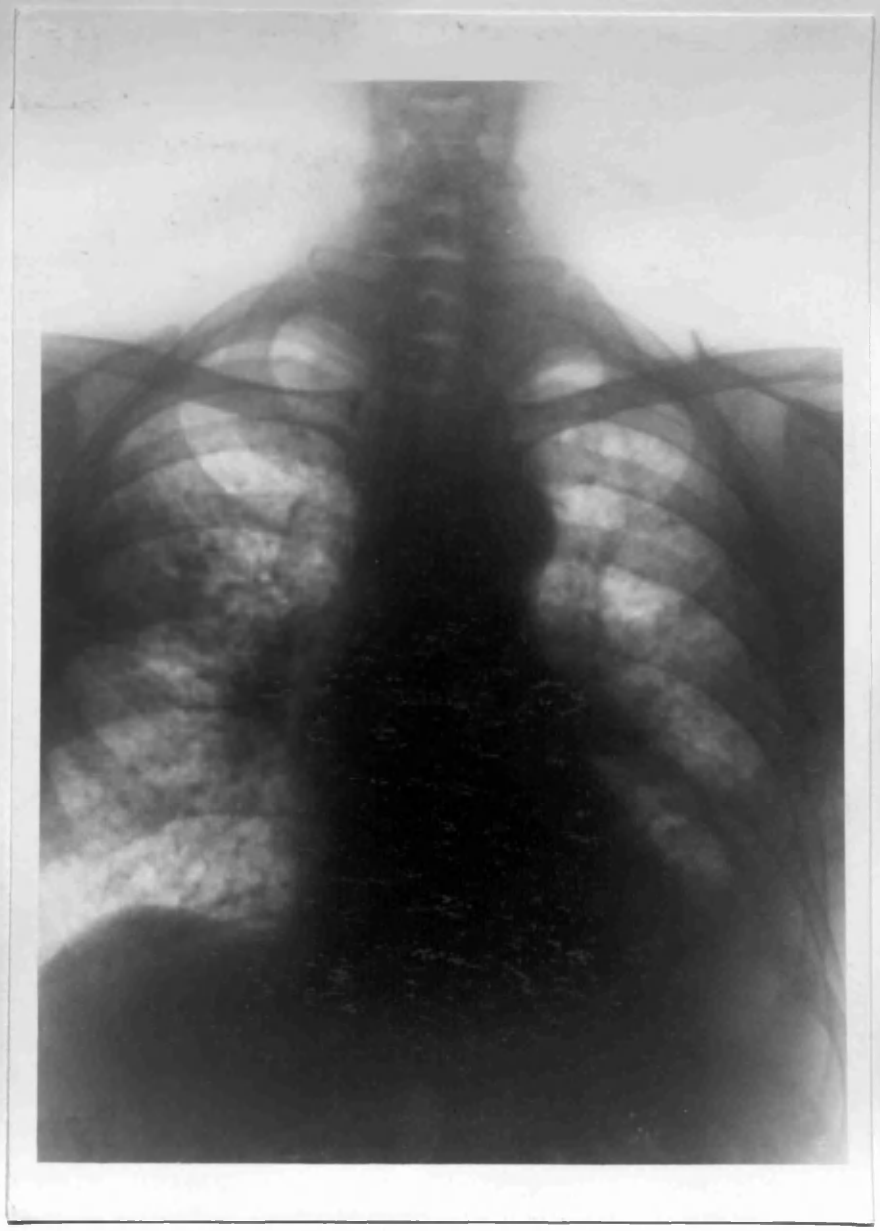


Case XXII.

T.S. age 47 years. Butcher blade grinder, sandstone (W) 30 years.

Silicosis (stage 3) progressing towards stage 4.

No. 14.



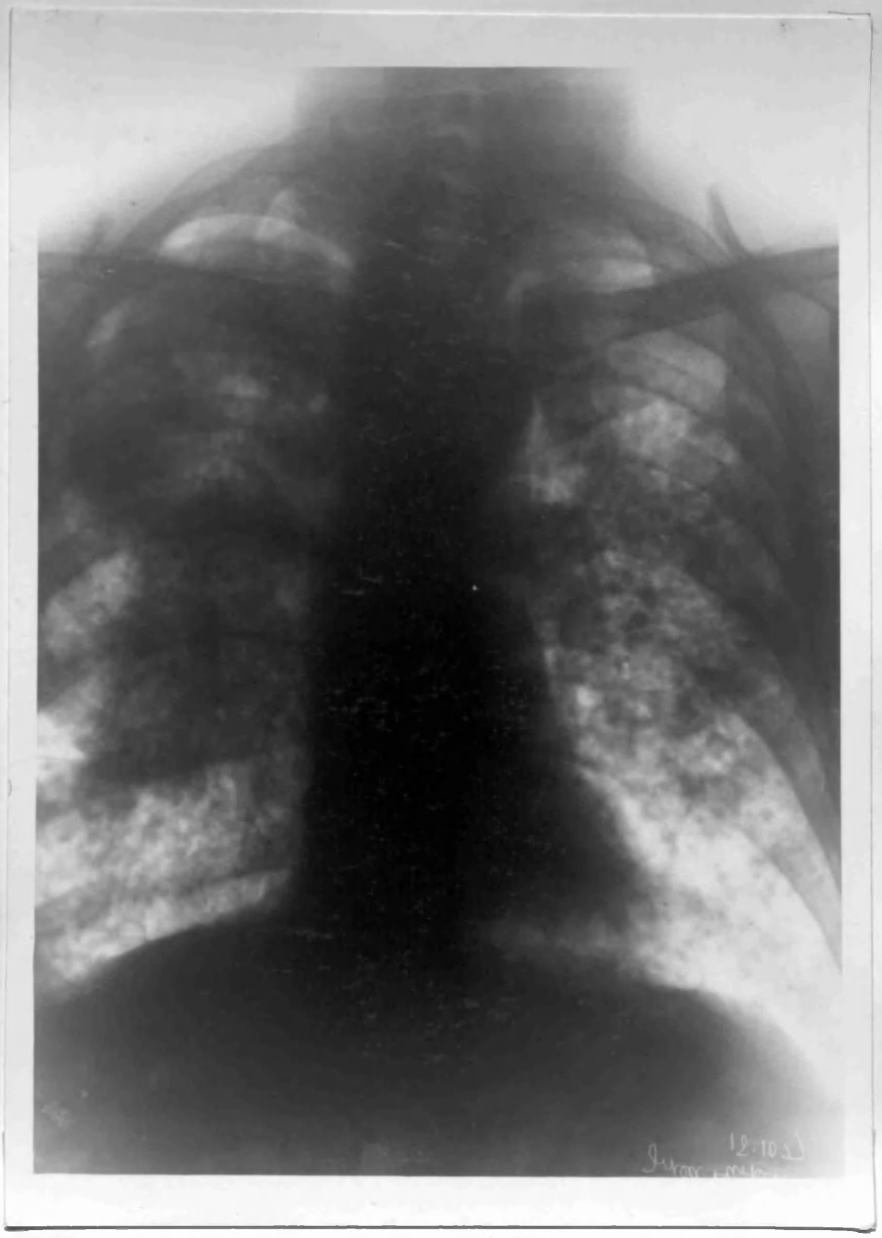
Case No. III.

P.M. age 55 years. Sheep-shear grinder, sandstone (W) 22 years.

Silicosis (stage 4).

Remarks : Note thickened pleura at left base.
Shadow in right axillary region probably denotes acute tuberculous infiltration.

No. 15.

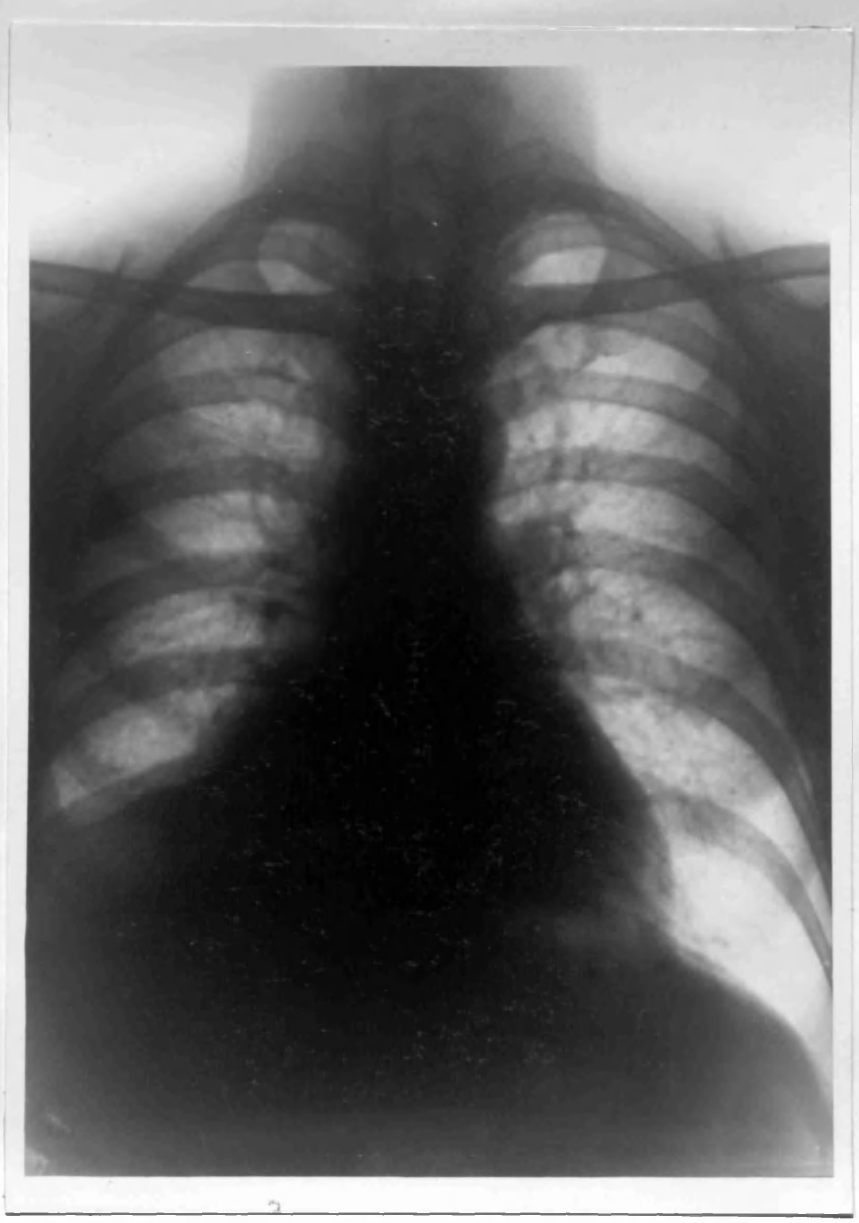


Case No.

F.P. age 52 years. Jobbing grinder, sandstone (W) 38 years.

Silicosis (stage 4).

No. 16.



Case No. XXIX.

W.W. age 39 years. Table blade grinder, sandstone (W) 16 years.

Silicosis (stage 1) ?

Remarks : Primary malignant growth of right lung - probably bronchial carcinoma.