HAEMATITE PNEUMOCONIOSIS IN CUMBERLAND MINERS*

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Iron oxide occurs as a dust in many industries apart from iron-ore mining. It is found in silver polishing, electric welding, and boiler scaling, as well as in the more obvious metal grinding, fettling, and blast furnace working where it may be mixed with varying amounts of silica.

Some still consider iron oxide a harmless dust (Baldi, 1952) while others think that, like coal, it can produce changes in the lung substance even in the absence of silica. Kettle (1932) observed that if he covered quartz with iron oxide little or no fibrosis occurred and animals inoculated with ferric oxide developed no fibrosis. In 1933 I found that if ferric oxide alone was injected into guinea-pigs and rabbits localized dust aggregates appeared but no progressive fibrosis. When the experiment was repeated with iron-ore containing 10% silica, even with the addition of dead tubercle bacilli, progressive massive fibrosis could not be produced, and as time passed the dust was transferred to the hilar glands.

Originally siderosis was considered to be a mixed dust pneumoconiosis, since the "knife grinder's lung" was the result of a combination of sandstone and ferric oxide. When the natural sandstones were replaced by carborundum and aloxan wheels it was thought that the problem was solved, but even the use of a silicon-free abrasive wheel and a silica-free parting powder did not prevent the disease, for in metal grinding and fettling there is still a considerable amount of silica attached to the casting in the moulding sand (McLaughlin, 1953).

A purer form of siderosis is seen in silver polishers where jewellers' rouge (pure ferric oxide) is used as an abrasive, and for long it has been considered harmless, though radiographs indicate a considerable retention of the dust in the lungs (McLaughlin and Harding, 1956). The abnormality of the radiological picture caused by the radio-opacity of the dust was not thought to be associated with any change in the

lung till Harding (1948) examined, at necropsy, the lungs of a silver finisher and found reticulation and focal emphysema. Siderosis is also found among electric arc welders who breathe in ferric oxide as a smoke, and lung opacities can be demonstrated in them by radiology. These opacities are associated with dyspnoea on exertion or chronic bronchitis, and biopsy shows a pneumonitis and fibrosis (Charr, 1956). It is in iron-ore mining that the greatest amount of ferric oxide dust is created and absorbed, and this was once the main industry in West Cumberland. In the beginning of the century the methods of mining were altered with the production of much more dust and the development of a disease that is now disappearing as the result of modern dust-suppressive methods.

Historical

The history of iron-ore mining in this country is long, for, like tin, it was mined before the Roman invasion (Fell, 1908), though, unlike tin, it did not attract the Mediterranean traders. Evidence of Norse and Roman workings exists in West Cumberland and the ore was smelted with charcoal made from the adjacent woods. Archaeological evidence reveals that the Romans used coal for smelting even in the time of Hadrian, but, generally speaking, coal was not used for smelting iron until the discovery of the coking process in the eighteenth century. The earliest written records of iron-ore mining in the north-west of England date from 1127 when Furness Abbey was founded. The monks are given the credit for having introduced iron-working into the area, following the dictate of King Edgar (950), who compelled every priest to learn a handicraft. A little later, in 1150, the monks of Holme Cultram Abbey, near Carlisle, were granted the right to mine and smelt iron-ore in Egremont, a village 10 miles south of Whitehaven. Thus these two abbeys (since the monks had a knowledge of geology as well as being iron-smiths) controlled the mining and

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smelting of iron-ore in the north. At one time one-sixth of the total income of Furness Abbey was derived from this source, since they had a virtual monopoly, owning all the iron deposits and bloomeries (which were primitive smiddies for smelting ore) and supplying the local needs for domestic and agricultural purposes.

The iron was smelted, using charcoal obtained from the local timber: soon, however, the available wood was consumed, and the ore, since it was less bulky than charcoal, was transported for smelting over the hills into the neighbouring valleys where trees existed to make charcoal. The names of the tracks over the hills by which the ore was carried from one valley to another in search of fuel still exist, such as Orsgarths between Coniston and Eskdale. There, in time, the supply of fuel became exhausted and, despite the practice of pollarding to maintain the trees for charcoal making, nevertheless great treeless areas existed. Thus travellers in the area wrote about the scarcity of trees, of which those existing were dwarfed and stunted. Such was the condition until the dissolution of the monasteries (1529–1536), when the property passed to the Crown and the monopoly was transferred to locally sponsored companies. These introduced German miners from the Carpathians, who settled in the area and whose names still exist in local families. This early attempt at nationalized mining did not survive.

The shortage of timber was the controlling factor and became so acute throughout the country that Queen Elizabeth passed a law forbidding the manufacture of charcoal for smelting ore. The forests of England were being depleted and she wished to conserve the oak trees as timber for her Navy because of the threat of the Armada (Fell, 1908). Thus between the fifteenth and eighteenth centuries, owing to shortage of fuel, the local smelting of ore periodically ceased.

At the beginning of the seventeenth century, ore was transported by sea from Barrow and Whitehaven up the west coast of Scotland to Argyll, Inverness, and Sutherlandshire and smelted wherever forests existed. Thus there is a series of small clachans, called Furnace, scattered over the west coast. There is one on Loch Fyne, another on Loch Etive, one near Strontian in Ardnamurchan, another in Glen Morrison, and one on Loch Ewe, where it is known that bog iron was smelted in 1607 (Dixon, 1886). Later this was supplemented by haematite brought by sea from West Cumberland. This fact had been established by examining the residue among the workings, and iron-ore, identified as haematite and containing fossils, has been found. Fossil-bearing

haematite could only have come from the West Cumberland deposits.

The requirements for smelting iron are carbon, as a source of heat and to produce carbon monoxide, which reduces the ferric oxide to the metal, and limestone to act as a flux and to remove the impurities, sulphur and silica. Round about the middle of the eighteenth century the process of coking coal was discovered whereby gases and tar were removed and this solved, for the time being, the fuel problem of West Cumberland where abundant supplies of coal existed alongside iron-ore and limestone. Up to the beginning of the nineteenth century there was not much demand for iron, apart from agricultural and domestic purposes, but with the onset of the industrial revolution, the development of the railways, and the building of steel ships the demand for iron and steel grew apace and thus began the development of the iron-ore mines and the West Cumberland Iron and Steel Works. Fresh deposits of ore were sought for at greater and greater depths and numerous small pits were opened throughout the Cleator area. There was a shortage of labour and thus the actively developing industry attracted labour from overseas.

Iron-ore in the north-west of England is almost entirely in the form of ferric oxide or haematite, which is jewellers' rouge, and exists in two forms. In Lancashire and south-west Cumberland it is soft and claylike with a high moisture content and can be dug out with a pick and shovel. There is little blasting and little dust and, from these mines, very few cases of pulmonary siderosis have occurred. In the north-west Cumberland deposits the haematite is quite different; it is dry, crystalline and has an index of hardness similar to that of pure quartz, the hardest substance next to diamond. The haematite exists in large masses in the deep layers of the carboniferous limestone at depths approaching 1,500 feet and most of the ore lies in lodes associated with faults in the limestone through movement of the earth's crust several million years ago.

There are two theories for its formation. The first is that the ore was forced into the faults in a molten state from below, and the picture of a specimen of kidney ore appears to lend weight to this theory (Fig. 1). It is a mass of pure kidney ore found covered with irregular bubbles suggesting that it had boiled. The second theory is that water, acid in reaction from the decaying vegetation, and saturated with iron from the volcanic ash, percolated through faults in the overlying sedimentary rock, and dissolved out the calcium, replacing it with ferric oxide. Proof that this, metasomatic replacement, is

FIG. 1.—Kidney ore (pure ferric oxide).

FIG. 2.—Pure haematite containing an imprint of fossilized fern.

the correct solution is found in the fossils of marine animals and vegetation often seen in the limestone (Fig. 2). These are composed partly, and sometimes wholly, of haematite. Since the limestone contained silica which resisted the solution of the ferruginous water, and the volcanic roof and floor to the haematite deposits are silicious, miners, while developing the mine, are subject to a silica hazard, but those whose work takes them into contact with the ore alone still have the risk of a dust which contains 10% silica. While this figure is now recognized as dangerous, 50 years ago it was not considered to be high enough to constitute a risk and therefore no danger was visualized and no precautions taken.

Until the introduction of pneumatic drilling in 1913, no drills would stand up to the hardness of the crystalline haematite, and the ore was mined by the method known as "hammer and jumper." A miner hammered into the ore a long, rod-like chisel making a hole 3 ft. deep; into this explosives were placed and detonated to bring down the ore, which he loaded into a tub. A good man working under favourable conditions could make six such holes in a working day, but with the introduction of pneumatic drills he could make 28 holes 4 ft. deep, that is, more than five times the number in the same time. This resulted not in five but in at least 500 times the amount of dust, since the high-speed pneumatic drill pounded the ore, producing a voluminous cloud of dust of fine particle size which the compressed air blew back as a cloud into the man's face. Since the drilling of more holes resulted in more blasting and consequently more dust, the dust-content of the air was built up until the man worked in an atmosphere so thick that a light could not be distinguished at 6 ft. Since there was no gas problem in iron-ore mines blasting went on

almost continuously and the men returned to work immediately, the acrid fumes of the nitrous explosives contributing to the general unpleasantness.

With the experience of the South African mines before them and the known danger of silica, wet drills were introduced in 1925, but from the beginning they were unpopular as they sprayed water back over the miner, soaking him despite protective clothing. Although wet drilling was the official policy, in practice drills were used dry when the manager was not present.

The Hazards

Dust in the air falls at a rate dependent on the specific weight and particle size, and as haematite is three times the weight of quartz the dust produced by pneumatic drills and blasting settles at an inverse ratio to its dangerous qualities, the larger particles settling at once while the dangerous dust, $1\ \mu$ in size, hovers as an invisible but dangerous miasma. This explains why the early dust suppressive measures were ineffective, although the air looked clearer, since the heavier ferric oxide was washed or sedimented out while the toxic quartz particles remained.

In the South African gold-mines counts of 400 dust particles (under $5\ \mu$ in size) per ml. of air are accepted as a maximum permissible standard (Skinner, Withers, Griffiths, and Williams, 1947) and over 1,500 is considered uncountable. In anthracite coal-mines in this country the standard is 660 particles, containing up to 10% quartz per ml. of air. At present in the haematite mines, with modern dust suppression, the recognized standard is 2,500 dust particles (under $5\ \mu$ with 10% silica) per ml. Under the old conditions with dry drills, counts at the face were frequently many million

TABLE I
MORTALITY FROM RESPIRATORY TUBERCULOSIS IN
MALES OF 20-65 YEARS

Period	England and Wales	Haematite Miners	
		Cumberland	Staffordshire and Yorkshire
1921-1923	100	107	56
1931-1933	100	170	55

(Craw, 1947a and b). In South Africa two types of silicosis are recognized: acute, which kills in two years, and a chronic type which takes 20 years to develop. In the former, the silica dust content of the air is high, and in the latter it is low. In general when the silica content of an industrial dust is high the mortality is highest among the young people, and when the silica content is low the mortality is highest among the older men.

Until 1913 iron-ore mining with the leisurely methods of hand drilling was considered a healthy occupation (Royal Commission on Metalliferous Mines and Quarries, 1912) and there was little evidence of bronchitis, pneumonia, or tuberculosis, but within 10 years of the introduction of pneumatic drills an increase in bronchitis was noticed with the subsequent development of shortness of breath on exertion among the older men. Several investigators (Collis, 1915, 1923; Collis and Goadby, 1930) published reports confirming this, but stating that while there was evidence of bronchitis and emphysema the mortality from bronchitis, pneumonia, and pulmonary tuberculosis was not outstanding. This is confirmed by the Registrar-General's (1927) Decennial Supplement for the period 1921-1923 when the death rate from respiratory tuberculosis among iron-ore miners between the ages of 20 to 65 years in Cumberland was only very slightly above the standard figure for England and Wales. It is of interest to compare this figure with the mortality among underground iron-ore miners in Staffordshire and Yorkshire whose death rate was only half the standard, though naturally the number of deaths in each case was small (Table I).

Cronin (1926) stated that on clinical examination of 100 drillers and underground workers in West Cumberland 64 were normal and only 30 showed emphysema, and the only complaints were dyspnoea on exertion and chronic irritation of the nose and throat when working in a dusty atmosphere. Tuberculosis was not found by him except in those miners who had worked in South Africa, for after the Boer War there was a shortage of skilled miners and many iron-ore miners from West Cumberland went to work in the gold fields. After varying times they

returned to Cumberland and some again went to work in the iron-mines. Cronin found that two-thirds of these had open tuberculosis and 17% of the underground workers in one mine had returned from working in the South African mines. Those men, known cases of open tuberculosis, were working in the ill-ventilated mines and were a source of infection to the other men, and may have been one of the means by which pulmonary tuberculosis was introduced into the industry.

Until 1930 phthisis was not recognized as a hazard in iron-ore mining, but between 1930 and 1933 several cases were diagnosed, and in 1933-34 12 out of 15 necropsies showed the cause of death to be active tuberculosis. Again, the Registrar-General's report (1938) confirms this (Table I), for the death rate in 1931-33 of the iron-ore miners of Cumberland was 70% higher than that of males at comparable age in the general population and three times that of the Staffordshire and Yorkshire underground miners.

Local statistics (Fig. 3) show that between 1921-51 the Cleator Moor district (population 8,000) had a death rate from pulmonary tuberculosis in males of twice that of England and Wales, while between 1948-54 the corresponding figure for the greater area of Ennerdale, containing Cleator and three other mining villages, was substantially higher than that of the remainder of the county and of England and Wales (Fig. 4).

Three factors may be responsible for the high death rate from pulmonary tuberculosis in a rural area with scattered villages surrounding small iron-mines: first, a local source of infection—this has

FIG. 3

Fig. 4

Fig. 5

been indicated; secondly, the genetic stock which in the area is different from the rest of Cumberland; and thirdly, the alteration of the industrial method with the introduction of an additional hazard. In the period immediately after the first world war there was general depression in West Cumberland with extensive unemployment, but this does not explain the high figure, for the depression involved the whole area and the other districts do not show the same high death rate from tuberculosis.

Before 1840 the population of Cleator Moor, the centre of the mining industry last century, was 750; by 1890 it had increased to 10,000 by the influx of labour attracted to the rapidly developing industry. This labour came largely from Ireland and there still exists a strong Irish element in the population as judged by names, temperament, and religion. In 1900 over 50% of the population of Cleator were adherent to the Roman Catholic Church (Caine, 1916) and the same holds good to-day. According to the Prothit Report the Irish are more prone to develop tuberculosis than the English, and, in addition to their lack of resistance, the social habits and standard of hygiene and housing encouraged the spread of disease among the families. As was shown by Heffernan (1930) the infectivity of silico-tuberculosis is of the same order as other open cases of pulmonary tuberculosis of a similar age group. The man with silico-tuberculosis usually is of an older age group, his family is grown up, and consequently fewer home contact cases occur.

The change of mining methods following mechanization is reflected by the increased output per man (Fig. 5). Before mechanization 4.6 tons were raised per man per week, but after mechanization three times that amount was raised, i.e., 14 tons per man per week. Thus in spite of a steadily decreasing labour force there was a relative rise in the total output and the amount of explosives used rose from 78 lb. per head annually in 1900 to 225 lb. per head in 1930 and 600 lb. in 1956 with a vast increase in the amount of dust.

Since the silica content of the dust is low, one would not expect to see abnormal results for 15 to 20 years, and so it was that the cases began to be recognized clinically and at necropsy from 1930 onwards—18 years after the introduction of compressed-air drills. As has been proved in coal-miners, dust alone does not produce progressive massive fibrosis without a concurrent or superadded chronic infection, for, without this, dust foci and emphysema do not progress.

The Pulmonary Lesion

In the siderosis of silver polishers, electric welders, and iron-ore miners the radiographic shadows largely consist of radio-opaque ferric oxide incorporated in the minimal amount of fibrous tissue within the bronchioles and lymphatics producing a reticular pattern. A somewhat similar reticular pattern is seen in coal-miners showing simple pneumoconiosis usually complicated by emphysema and associated clinically with dyspnoea and increased

residual air. On examining these lungs there are dust foci surrounded by focal emphysema and frequently peribronchial fibrosis. In contrast with the coal-miners' reticulation the siderosis associated with ferric oxide is not accompanied by respiratory embarrassment to the degree that the radiograph would suggest. It has been assumed that the reticular pattern associated with iron-oxide retention related to the various occupations is not associated with any structural changes and therefore could be discounted. This is not always the case and focal emphysema may develop late in life, often associated with cor pulmonale, and produce a degree of breathlessness on exertion greater than is usual for the age. There is considerable doubt about the association of focal emphysema and dyspnoea, and no reliable methods exist of correlating the degree of disability with the radiological appearance.

Progressive massive fibrosis of haematite miners is a modified form of infective pneumoconiosis, which, in 1934, when it was first described (Stewart and Faulds, 1934), was considered to be tuberculous. There is still much to support this, but on the other hand the view that the chronic infection is commonly, but not invariably, tuberculous is probably nearer the truth. Harding and Massie (1951), in describing the pneumoconiosis of boiler scalers, are "unconvinced by the argument that such lesions are invariably and necessarily due to added tubercle." Cochrane and Miall (1956) hold a similar view.

There is another possible explanation that is worth considering. Brock (1954) argues that pus from the nasopharynx trickles down the trachea during sleep, and runs into the upper lobe bronchus on whichever side the patient lies sleeping; when he sleeps on his back it flows into the apical segment of the lower lobe. For this reason the upper lobe is the site of election for pulmonary abscesses.

All the miners I have seen remark that drillers quickly get their noses choked with dust and suffer from irritation of the throat and nose and become, of necessity, temporary mouth breathers. After coming off the shift, they cough and spit large quantities of rusty-coloured sputum. This fact was commented upon by Cronin (1926) and also by Lawson, Jackson, and Gardner (1931) in America. Thus during the day the miners breathe in dust containing silica and during the night drain into the upper lobes an infected purulent secretion containing dust and organisms. Could not this be the source of the chronic infection, for it would account for the location of the fibrosis in the upper lobe more commonly than in the lower? I am aware that these are sites where chronic tuberculosis

is also found, but the same physical explanation might account for the location of both lesions.

Causes of Death

In analysing the causes of death among the 240 iron-ore miners on whom post-mortem examinations have been performed during the last 25 years two features stand out as significant, the high incidence of tuberculosis as a cause of death in spite of modern methods of treatment, and the incidence of carcinoma of the lung. To ascertain if there was an association between the degree of fibrosis and the amount of silica present, a chemical analysis was carried out on the lungs of 163 of the iron-ore miners and the results are grouped into four categories. The first group shows no progressive massive fibrosis though there were varying amounts of emphysema, and since the cause of death was not related to pneumoconiosis this group acted as a control. The second group shows progressive massive fibrosis in varying degrees, but no histological evidence of active tuberculosis. The third group shows fibrosis complicated by active tuberculosis, and the fourth group died of cancer of the lung. In the four groups as shown in Table II, the

TABLE II
RESULTS OF CHEMICAL ANALYSIS OF LUNGS IN 163
HAEMATITE MINERS

Findings in 163 Haematite Miners	Fibrosis	No. in Group	Average Age at Death	Years in Mine	Ash (% of Dry Weight)	Silica Content (% of Ash)	Fe ₂ O ₃ Content (% of Ash)
Normal lung (Cumberland)	0	—	—	—	1.0	1.0	0.1
No fibrosis and no tubercle	0	19	62	26	11.0	5.5	33.0
Fibrosis only	+	58	60	32	15.0	8.3	39.0
Fibrosis plus tuberculosis	++	69	57	28	13.0	8.9	38.0
Carcinoma of lung	+	17	61	31	13.0	9.2	39.0

amount of iron and silica present seems to bear a relationship to the degree of fibrosis, for the longer the men have worked in the mine the higher is the ash and silica content. Twelve per cent. had no fibrosis and yet had spent 26 years in the mines and had an ash content of 11 times normal, and since the ash is high the silica is also five times the maximum normal amount. The average age of those dying of pulmonary tuberculosis was lower than the other groups, while those dying of cancer had worked longer and lived longer, several in the group being over 70 years and one over 80 years.

The Cardiff school (Gough, 1940) maintains that tuberculosis is the commonest infective agent, yet in only 50% can they recover tubercle bacilli at necropsy. Zaidi, Harrison, King, and Mitchison (1955), in

a recent experiment, have produced the nearest approach to massive fibrosis by injecting into sensitized animals dust plus tubercle bacilli. The lesions, however, regressed spontaneously after three months and they could not recover tubercle bacilli from more than 50%, thus resembling the condition in the Welsh miners.

In the last two and a half years necropsies have been carried out upon 58 iron-ore miners of whom 30% have died of pulmonary tuberculosis in spite of active treatment by modern methods. In many of the cases it cannot be decided whether the tuberculous infection is a terminal event in an already damaged lung 25 years after being exposed to the intense dusting of dry drills, or has been present undiagnosed for all these years, producing the massive fibrosis and eventual necrosis and cavitation. I favour the former view in most cases, believing that the dust predisposes to infection and development of a progressive tuberculous disease.

The influence of silica on a tuberculous infection is seen in three cases which failed to respond to treatment and died of extensive pulmonary tuberculosis with lungs pigmented with iron but showing no naked-eye or microscopic evidence of fibrosis (Table III). Chemical analysis revealed an increase

TABLE III
FOUR TUBERCULOUS MINERS WITH NO FIBROSIS

Weight Both Lungs (g.)	Dry Weight (g.)	Percentage of Ash	Total Ash (g.)	Percentage of Silica	Total Silica (g.)	Increase
3,275	468	5	23	3.6	0.8	× 2
3,785	697	7	49	4.0	1.9	× 5
1,925	308	16	49	10.0	4.9	× 12
Normal						
1,000	200	4	8	4.0	< 0.4	King

both in dry weight and total ash. If the silica is expressed as a percentage it does not seem spectacular, but when expressed as total silica a very considerable increase is demonstrated, which must have been a contributory factor in the progress of the disease, and possibly offers an explanation for the failure to respond to treatment.

The chemical agents are ferric oxide and a small amount of silica, and it has always been considered that the silica was the important irritant and that the iron merely modified it, but caution must be exercised, for Harding has shown that focal emphysema can be produced in silver polishers using pure jewellers' rouge, and from the Lorraine district in France comes a report (Dechoux, 1954) that the iron-ore miners there working in haematite are also showing radiological changes of massive

fibrosis similar to those seen in West Cumberland. The ore is alleged to contain a very low silica fraction, and from the chemical analysis of the lungs in the few necropsies done the silica content is also low, yet the post-mortem findings are similar to, and the clinical symptoms of dyspnoea and bronchitis identical with, those of the West Cumberland miners.

Incidence of Carcinoma

The other disease with an increased incidence found among iron-ore miners is cancer of the lung. This disease has increased in the male population in the United Kingdom during the last 50 years by nearly 100 times, being responsible for death in 0.06% of the population in 1955 while in 1905 the figure was only 0.0005%. In view of this, an increase in the incidence in any particular trade has to be accepted with caution, but considering the small number of men now employed in the iron-ore industry (about 1,000) the number of deaths from this disease seems quite out of proportion. No similar increase has been found in coal-, tin-, or lead-miners in this country, though an increased incidence has been observed in asbestos and chrome workers. In general it is believed that there is probably a delay of 20 years between the exposure to the agent responsible and the subsequent development of the malignancy. All the known carcinogenic agents have been considered: radioactive elements, arsenic, nickel, tar products from the combustion of coal, and the only factor that cannot be excluded is the chronic irritation of silica plus iron in the presence of infection, producing a slow progressive fibrosis.

The striking feature in the lung tumours found in iron-ore miners is the location of the primary tumour to the sites where the dense fibrosis is commonly seen. Schmorl, in 1925, first suggested a connexion between scars and tumour formation in the lung, and Raeburn (1951) and Raeburn and Spencer (1953) established a connexion between scars and tumours in several of their cases where the tumour arose in a peripheral lung field. Walter and Pryce (1955) examined 207 lungs removed in the surgical treatment of cancer and were able to locate the primary lesion in 59%; in 50% of these this primary lesion was in a peripheral field, yet it is still commonly believed that all lung tumours arise at the hilum. In iron-ore miners the massive fibrosis is usually located in the apex of the upper lobe in a peripheral field just under the pleura and from there it may spread to involve the whole lobe. It also arises around the interlobular fissure and the upper part of the lower lobe and later involves the hilum.

FIG. 6.—Early stage of progressive massive fibrosis involving the apex of the upper lobe (P.M. 5418).

FIG. 7.—Progressive massive fibrosis involving the upper lobe and upper part of lower lobe (P.M. 5016).

FIG. 8.—Advanced stage of progressive massive fibrosis (P.M. 2939).

FIG. 9.—Advanced silico-sidero-tuberculosis (P.M. 5063).

Figs. 6 to 9 show sections of whole lung of varying degrees of fibrosis which apparently is not directly proportional to the length of time spent underground nor connected directly with the amount of silica present, but the interest lies in the location of the fibrosis; for it is in these sites that primary lung tumours are found in haematite miners, and not in the main bronchus where it usually manifests itself clinically and where it presents itself most obviously at necropsy. In Fig. 10 there is delineated the sites of the primary tumours in the last 22

FIG. 10.—Location of primary tumours in haematite miners.

cases; this shows their equal distribution between the two lungs; seven arose in the right upper lobe, one in the upper part of the right middle lobe just under the pleura, and one in a similar position in the right lower lobe. Two were found arising at the hilum on the right side. On the left side three were found in the upper lobe, three in a peripheral field in the lower lobe and five at the hilum.

Figs. 11 and 12 are photographs of lungs from haematite miners and show malignant tumours arising in close association with a sidero-silicotic mass. In Fig. 11 (P.M. 6388) the tumour arose immediately under the pleura in the upper part of the lower lobe, while in Fig. 12 (P.M. 6643) the tumour arose in the upper lobe close to a sidero-silicotic mass and extended from there to the main bronchus where it could easily have been mistaken for a primary hilar tumour.

In progressive massive fibrosis of haematite miners and asbestos workers the fibrous mass extends by enclosing gradually the adjacent alveoli which usually show focal emphysema. Cells lining the alveoli and bronchioles, and dust-laden macrophages lying within their lumen, become incorporated into the fibrous tissue mass and are compressed and altered in appearance, so that eventually

FIG. 11.—Lower lobe of lung, showing carcinoma arising in a sidero-silicotic mass just under the pleura (P.M. 6388).

FIG. 12.—Carcinoma arising in the upper lobe in a sidero-silicotic mass and extending towards the hilum (P.M. 6643).

it is impossible to distinguish cells of epithelial origin from fibroblasts.

At the periphery of the fibrous mass, dust-laden cells can be seen lying in the isolated and distorted alveoli and bronchioles. These cells, after the collapse of the alveoli and bronchioles, become surrounded by fibrous tissue and eventually are destroyed, leaving the enclosed dust incorporated in the fibrous tissue. Thus in the centre of the mass there exists the dust, imbedded in collagenous fibrous tissue with little blood supply, in the intermediate zone dust and indistinguishable cells, and in the periphery recognizable macrophages, alveolar and bronchial cells laden with dust particles (Figs. 13 and 14).

It is an established fact (*British Medical Journal*, 1956, 1, 906) that dust is an aetiological factor in the production of chronic bronchitis with hyperplasia of the mucus-secreting glands, and one wonders if certain dusts may not be an important factor in the production of lung cancer, for carcinoma of the lung is now recognized as an industrial disease in asbestos workers, being present

in 17 to 18% of cases of asbestosis (Kennaway and Kennaway, 1947; Merewether, 1949; Gloyne, 1951; Doll, 1955).

In haematite miners I believe that lung cancer arises from the damaged epithelial cells at the edge of a fibrous mass; for the tumour arises in these sites where fibrosis is most common and from there spreads to the hilum and not in the reverse direction.

The hypothesis that tumours arise in a bronchus and invade a peripheral avascular fibrous mass against the natural lymph flow is not supported by our histological investigations.

With the exception of asbestos workers, moulders and foundrymen (McLaughlin, 1956; McLaughlin and Harding, 1956) and chromate workers (Bidstrup and Case, 1956), no similar increase in lung cancer has been noticed in any other silicious trade, and if silica alone was the causal agent it would have been reported in South Africa. Among iron-ore miners, moulders, and foundrymen there is an exposure to the physical combination of silica and iron acting as an irritant and leading slowly to fibrosis, while both asbestos

FIG. 13.—Low-power view of an area of progressive massive fibrosis surrounded by distorted collapsed alveoli.

FIG. 14.—High-power view of lower right-hand corner of Fig. 13, showing the incorporation of dust-laden macrophages, bronchiolar and alveolar cells into the fibrous mass.

and chromate contain a varying amount of iron in the chemical composition.

The original figures published on lung tumours in iron-ore miners (Faulds and Stewart, 1956) covered the period 1932 to 1953, and since then an additional 58 necropsies have been performed making a total of 238. In the last 58, seven (12%) primary lung tumours have been found, and, if the figures from 1949 to 1956 are taken, 20 (14%) lung cancers have been found in a total of 148 necropsies (Table IV).

TABLE IV

INCIDENCE OF CARCINOMA OF THE LUNG IN HAEMATITE MINERS COMPARED WITH CONTROL GROUP

Period	No. of Necropsies on Haematite Miners	Carcinoma of Lung		No. of Necropsies on Males > 30 Years not Haematite Miners	Carcinoma of Lung	
		Total	%		Total	%
1930-1948	90	4	4	2,221	41	2
1949-1953	90	13	14		33	5
1954-1956	58	7	12		78	2.7
Total..	238	24	10	2,882		

The average age of this group is 61 years and the average time spent in the mines is 30 years. All these men, therefore, had worked during the period when conditions were bad and those who did not die of silico-tuberculosis at a younger age survived

to the period when cancer is common. The fact that the cancer has originated in the lung and in the areas where fibrosis is most commonly found suggests an association between the two.

Lymphadenitis

In addition to pulmonary tuberculosis and lung cancer, there is another lesion occasionally seen in iron-ore miners which may give rise to difficulty in diagnosis during life and even at necropsy. This is lymphadenitis of hilar glands, with ulceration into a bronchus and an associated sclerosing bronchitis. Frequently this lesion is considered, radiologically, to be malignant, and at bronchoscopy ulceration, with stenosis, is found. Histologically it is seen to be chronic inflammation containing iron oxide and silica crystals, and is caused by ulceration of a hilar lymph node into a bronchus. The following two cases illustrate this lesion.

P.M. 2828.—An iron-ore miner, age 59 years, had worked 40 years underground and suffered from dyspnoea on exertion. After a pyrexial cold he got up one morning to return to work, and had a massive haemoptysis and died. Necropsy showed no evidence of tuberculosis or tumour, but the main bronchus was thickened and stenosed, and into it a silicotic lymph node had ulcerated. At the same time the lymph node had ulcerated into the pulmonary artery and established a

FIGS. 15 AND 16.—Probe passing from fibrosed ulcerated bronchus into the pulmonary artery (P.M. 2828).

sinus between this and the bronchus. To the naked eye the specimen displays a densely fibrosed upper lobe and a thickened bronchus resembling tumour. Figs. 15 and 16 show the probe passing through from the pulmonary artery to the bronchus. Histological examination revealed no tumour, but the lymph node lying between the artery and the bronchus showed a sidero-silicotic fibrosis with subacute inflammation.

Fig. 17 is from a similar case (24435/56), a young man of 27 years who had been an iron-ore miner for seven years and had a marked stenosis of the bronchus, with partial collapse of the lower lobe. He suffered from chronic bronchitis and repeated attacks of pneumonia. A biopsy of the bronchus showed a chronic ulcer with no evidence of tumour. A pneumonectomy was carried out and the specimen showed a bronchus reduced to a narrow slit, surrounded by dense fibrous tissue which was ossified and attached to the prevertebral tissue. It was also attached to the right auricle, and extended across to the bronchus on the other side. Both the clinical condition and the specimen suggest tumour, but on histological examination the lesion is one of dense fibrous tissue and chronic inflammation, with areas of ossification and small foci of bone marrow; there is no evidence of neoplasia. The condition may have originated as a lymphadenitis with ulceration into the bronchus as in the previous case.

Present Position

The effect of the dust-suppressive measures has been seen in the general health of the miners during

FIG. 17.—Calcifying fibrous mass surrounding and compressing the bronchus.

the last 20 years (Craw, 1947a and b). In 1935 30% of the miners who had spent 10 to 20 years underground had abnormal chest radiographs and 1.5% had progressive massive fibrosis. Now 20 years after the introduction of dust suppressors only 1% have abnormal radiographs (Craw, personal report) due to the active co-operation of both sides of the industry in reducing the amount of dust.

Experimental dusting of animals may be a useful means of confirming a finding in pneumoconiosis if the results are positive, but great caution must be observed in deciding that a dust is harmless on conclusions drawn from a negative animal experiment.

All dusts are dangerous if present in excessive amount, and since the effect of an industrial dust may not be evident for 25 years the only way to increase our knowledge is to keep accurate records: (a) in industry, of the type of work done by the workmen; and (b) in hospitals, of the duration of the various occupations followed by all patients, especially those who have retired. These should be correlated with the findings at necropsy in all cases of pulmonary fibrosis.

Summary

The development of sidero-silicosis in haematite miners can be traced to the alteration in the methods of mining in the beginning of this century, which resulted in a great increase in output of ore per man but was associated with a great increase in dust production. A complicating factor was the large percentage of Irish in the industry, and the introduction of tuberculosis into the mining communities.

The incidence of lung tumours among haematite miners is greater than expectation. It is suggested that this may be explained by the chronic irritation of ferric oxide and silica, since the location of the tumours corresponds with the peripheral areas of fibrosis.

The prevention of unnecessary dust is essential in all industrial processes, since the effects may not be manifest for 25 years. Unless accurate records are kept it will be impossible to trace the responsibility for dust.

REFERENCES

- Baldi, S. (1952). *Riv. Infert. Mal. proff.*, **39**, 136.
- Bidstrup, P. L., and Case, R. A. M. (1956). *Brit. J. industr. Med.*, **3**, 260.
- Brock, R. C. (1954). *The Anatomy of the Bronchial Tree*, 2nd ed. Oxford University Press, London.
- Caine, Caesar (1916). *Cleator and Cleator Moor: Past and Present*. Wilson, Kendal.
- Charr, R. (1956). *Ann. intern. Med.*, **44**, 806.
- Cochrane, A. L., and Miall, W. E. (1956). *Brit. med. J.*, **1**, 1193.
- Collis, E. L. (1919). *Industrial Pneumoconioses, with Special Reference to Dust-Phthisis*. Milroy Lectures (1915). H.M.S.O., London.
- (1923). *Proc. roy. Soc. Med. (Sect. of Epidem. and State Med.)*, **xvi**, 85.
- and Goadby, K. W. (1930). *J. industr. Hyg.*, **12**, 266.

- Craw, J. (1947a). *Brit. J. industr. Med.*, **4**, 30.
- (1947b). (Proc. of Conf.) *Silicosis, Pneumokoniosis and Dust Suppression in Mines*, p. 68. London Instn. of Mining Engineers and Instn. of Mining and Metallurgy.
- Cronin, A. J. (1926). *J. industr. Hyg.*, **8**, 291.
- Dechoux, J. (1954). *Les Pneumoconioses des Mineurs de Fer du Bassin de Lorraine*. Nancy.
- Dixon, J. H. (1886). *Gairloch in North-West Ross-shire: its Records, Traditions, Inhabitants, and Natural History*. Edinburgh.
- Doll, R. (1955). *Brit. J. industr. Med.*, **12**, 81.
- Faulds, J. S., and Stewart, M. J. (1956). *J. Path. Bact.*, **72**, 353.
- Fell, Alfred (1908). *The Early Iron Industry of Furness*. Kitchin, Ulverston.
- Gloyne, S. R. (1951). *Lancet*, **1**, 810.
- Gough, J. (1940). *J. Path. Bact.*, **51**, 277.
- Harding, H. E. (1948). *Brit. J. industr. Med.*, **5**, 70.
- and Massie, A. P. (1951). *Ibid.*, **8**, 256.
- Heffernan, P. (1930). *J. industr. Hyg.*, **12**, 236.
- Kennaway, E. L., and Kennaway, N. M. (1947). *Brit. J. Cancer*, **1**, 260.
- Kettle, E. H. (1932). *J. Path. Bact.*, **35**, 395.
- Kilpatrick, G. S., Heppleston, A. G., and Fletcher, C. M. (1954). *Thorax*, **9**, 260.
- Lawson, G. B., Jackson, W. P., and Gardner, J. E. (1931). *J. Amer. med. Ass.*, **96**, 1129.
- McLaughlin, A. I. G. (1953). *Lancet*, **2**, 49.
- (1956). In *Conditions in Iron Foundries* (First Report of the Joint Standing Committee). [Ministry of Labour and National Service.] H.M.S.O., London.
- and Harding, H. E. (1956). *Arch. industr. Hlth*, **14**, 350.
- Merewether, E. R. A. (1949). Annual Report of the Chief Inspector of Factories for 1947, p. 79.
- Raeburn, C. (1951). *Lancet*, **2**, 474.
- and Spencer, H. (1953). *Thorax*, **8**, 1.
- Registrar-General (1927). Decennial Supplement, England and Wales, 1921, Pt. 2, Table 16, p. lxxi.
- (1938). *Ibid.*, 1931, Pt. 2a, Tables 4a and 4d, pp. 224 to 309.
- Royal Commission on Metalliferous Mines and Quarries (1912). Minutes of Evidence, Vol. 1, p. 7, Question 153 [1910]. Cd. 6390.
- Schmorl, A. (1925). *Munch. med. Wschr.* **72** 757
- Skinner, D. G., Withers, A. G., Griffiths, J. H., and Williams, F. T. (1947). (Proc. of Conf.) *Silicosis, Pneumokoniosis and Dust Suppression in Mines*. London Instn. of Mining Engineers and Instn. of Mining and Metallurgy, p. 156.
- Stewart, M. J., and Faulds, J. S. (1934). *J. Path. Bact.*, **30**, 233.
- Walter, J. B., and Pryce, D. M. (1955). *Thorax*, **10**, 117.
- Zaidi, S. H., Harrison, C. V., King, E. J., and Mitchison, D. A. (1955). *Brit. J. exp. Path.*, **36**, 539.