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INDUSTRIAL LUNG DISEASES OF IRON AND STEEL FOUNDRY WORKERS

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Industrial Lung Diseases of Iron and Steel Foundry Workers

An investigation of 3,059 workers in 19 foundries together with an analysis of the records of lung diseases in foundry workers in the files of the Factory Department and the Silicosis and Asbestosis Medical Board; pathological investigations of the lungs of 64 foundry workers and dust surveys in three foundries.

BY

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CHAPTER I

REVIEW OF LITERATURE

BY A. I. G. McLAUGHLIN

"That men are exposed to particular diseases from the occupations which they follow, is a fact well known; but to remedy this evil is a matter of some difficulty. Most people are under the necessity of following those employments to which they have been bred, whether they be favourable to health or not. For this reason, instead of inveighing in a general way, as some authors have done, against these occupations which are hurtful to health, we shall endeavour to point out the circumstances in each of them from which the danger chiefly arises, and to propose the most rational methods of preventing it." (William Buchan's "Domestic Medicine" – 2nd Edn. London 1782).

The making of metal articles by pouring the molten metal into sand moulds has been recognised since ancient times to be an occupation injurious to the health of foundrymen mainly on account of the dust which arises during the process. The history of foundry health risks is therefore largely that of the pneumoconioses or pulmonary dust diseases. Foundry work is hot as well as dusty and the workers are subjected to sudden changes of temperature; it also calls for the expenditure of much physical effort, though in modern foundry practice much of the excessive physical effort involved in the handling of heavy weights has been diminished by mechanical aids such as cranes and other lifting tackle. In addition to the pneumoconioses, it is said that foundrymen have a high incidence of pneumonia and bronchitis and also that they suffer unduly from rheumatism and hernia. In the present enquiry we have directed much of our attention to the pneumoconioses, though some reference will be made to the other health risks.

The early writers on occupational diseases dealt mainly with the risks of mining with incidental references to foundry work. Hippocrates (450 – 361 B.C.)(1) in his Epidemics described the illness of a metal digger (lead miner) who had constipation, a large spleen, shortness of breath and swelling of the left knee. The earliest recorded reference to the effects of dust is found in Pliny's Natural History (1st Cent. A.D.)(2). He mentioned the use of bladders worn as respirators by metal refiners to avoid inhalation of dust. Other ancient writers discussed dust phthisis which they confused with tuberculosis but not until we come to Paracelsus (1493 – 1541)(3) are there specific references to metal founders. He was a Swiss doctor and alchemist who had been a miner and metal founder between 1510 and 1520 at Schwaz in the Tyrol. About 1530 he wrote a book on industrial diseases, published in 1567 after his early death to which his work in the mines and foundries is said to have contributed. He described the chronic lung diseases of miners as "pulmonary consumption" and "asthma" and while he attributed these diseases mainly to the influence of astral bodies, he recognised that mineral emanations and the "climate" of mines might be the cause of the lesions. But he did not specifically refer to dust as a possible cause. Georgius Agricola (1494 – 1555)(4), a mining expert and also a student of industrial hygiene, in his *De Re Metallica*, published in 1557, also referred to the health risks of miners and foundrymen. He recognised that the inhalation of "corrosive" dust predisposed a worker to a disease characterised by exhaustion, coughing and "that difficulty of breathing which the Greeks call asthma" but he confused the condition with phthisis. He pointed out that in the Carpathian mountains there were women who had married seven

husbands "all of whom this dreadful disease had brought to an early grave". He also referred to the use of respirators by the miners. Collis (1919) sums up these observations as indicating that asthma follows dust inhalation, that only certain "corrosive" dusts cause consumption and that female contacts do not contract the disease. Nicola le Pors (1527 – 1633)(6) of Pisa was the first to describe a case of phthisis due to continued exposure to the atmosphere of a furnace. Van Helmont (1577 – 1644)(7) described metal miners' disease due to dust as a kind of asthma, midway between the "dry" and "damp" varieties of the condition. He found that the disease also affected makers and stampers of coins and maintained that in this case the metallic vapours set up a vaso-constrictive action in the lungs. Martin Pansa,(8) about this time also published a work on diseases of miners and metal founders but added little to the clinical picture or to the aetiology. Leonardus Ursinus(9) wrote in 1652 on the peripneumonia of miners and metal founders. He put the lung lesions into two groups, those arising from various metal fumes and those affected by the humours. The principal symptoms of miners' disease were cough, dyspnoea and asthma and the main contributory factors were dust inhalation, fatigue, strain and trauma.

Samuel Stockhausen,(10) a doctor at the Goslar mines with over 10 years experience refers in a book (1649) to metal disease and miners' phthisis. He visited foundries and went down mines to supervise workers' health and he even exposed himself to the inhalation of what were regarded as toxic fumes. Amongst the factors responsible for miners' phthisis he mentioned meteorological and atmospheric conditions, metal fumes and especially dust, which at Goslar was so abundant that it covered the miners' bodies and forced them to wear "bits of material" over the nose and mouth. They were affected by a sense of oppression in the chest, asthma, persistent irritating cough with marked hoarseness of the voice. The condition tended to degenerate into consumption and death. Usually these symptoms were accompanied by others due to metal poisoning. Diemerbroeck,(11) also in 1649, was the first to carry out an autopsy on a case of what we now know as silicosis. Ramazzini,(12) who wrote the first text book on occupational diseases in 1700, quotes Diemerbroeck's description of examinations of diseased stonecutters "in whose lungs he found such heaps of sand, that in running the knife through the pulmonary vesicles he thought he was cutting some sandy body". Erythopilus(13) in 1675 was the first writer after Diemerbroeck to emphasise the importance of dust as the cause of pulmonary disease and referred to workers engaged in pouring and refining metal – "idem artificibus, qui circa metalla caedenda et purificanda versantur, contingere constat. Quemadmodum, autem hic aer quibusdam vicinus a vaporibus et fumis inficitur, ita alic rum locorum totus aer infectus est, ut dein in plurimos agat"*. William Buchan(14) in his book Domestic Medicine (1782) wrote that "chymists, founders, forgers, glassmakers and several other artists are hurt by the unwholesome air which they are obliged to breathe. This air is not only loaded with noxious exhalations arising from metals and minerals, but is so charged with phlogiston as to be rendered unfit for expanding the lungs sufficiently, and answering the other important purpose of respiration. Hence proceed asthmas, coughs and consumption of the lungs, so incident to persons who follow these employments". Incidentally he goes on to say that "it may seem romantic to recommend gardening to manufacturers in great towns: but observation proves that the plan is very practicable. In the town of Sheffield, in Yorkshire, where iron-manufacture is carried on, there is hardly a

* It is agreed that the same thing happens to workmen engaged in cutting and refining metals. Not only is the air adjacent [to these processes] contaminated by certain exhalations and fumes but in some places the whole air is polluted in such a way that very many persons are affected.

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The 19th century is notable for the development of the idea that the inhalation of excessive quantities of any kind of dust affects the lungs but that some dusts are more harmful than others. A good account of this period is to be found in E. L. Collis's[15] classical Milroy Lectures to the Royal College of Physicians in 1915. He quoted, for instance, the statement of Thackrah[16] of Leeds that "dust of every kind irritates, but not in equal degree". Thackrah noted that bricklayers and limeworkers were long-lived, that sandstone masons and stonedressers were short-lived and he also drew attention to the prevalence of phthisis amongst the metal grinders of Sheffield. He quoted Knight's opinion that fork-grinding ought to be confined to criminals

The term "pneumonokoniosis" (later shortened to pneumoconiosis) was first coined by the German pathologist Zenker[17] about 1860, he also described a variety of pneumoconiosis due to the inhalation of iron oxide dust and called it siderosis. Many names were later invented, to describe different varieties of pneumoconiosis such as melanosis, anthracosis (coal miners' lung), chalicosis (stoneworkers' lung), schistosis (slateworkers' lung). A recent one is byssinosis, used to describe the lung affections of cotton workers. The more striking names for the pulmonary lesions are the local or trade names such as 'grinders' rot," 'miners' phthisis," 'potters' rot or asthma'

Progress in the study of pulmonary diseases was dependent on advances in pathological knowledge and technique and particularly on the discovery of the microscope and X-rays. The most noticeable naked-eye feature of the dusted lung is pigmentation and it is recorded that there was controversy over the way in which the pigment reached the lungs. Pearson[18] in 1813 showed that it came from the dust inhaled into the lungs but the French and German pathologists believed that it was secreted from the body. The term "silicosis" is said to have been first used by Visconti[19] in 1870 to describe the pathological condition of the lungs resulting from the inhalation of silica. But in spite of this it was long before the nature of silicosis was distinguished from that caused by the tubercle bacillus. Indeed it was not until Clark, Hadley and Chaplin[20] in 1874 published their monograph on Fibroid Disease of the Lungs that the fibrosis associated with chronic tuberculosis was clearly differentiated from other causes of fibrosis of the lung. Even they make no reference to silicotic nodulation but they include a short paragraph on dust phthisis. They quote the work of Peacock and Greenhow who give "interesting cases of lung disease induced by the inhalation of irritating dusts. Many of these were undoubtedly tubercular, having been set going by the irritating dust, but some on microscopic examination appeared to present all the characters of chronic pneumonia caused by irritation. Greenhow relied on a cool skin, quiet pulse, and a wheezy asthmatic cough to distinguish this disease from tubercular phthisis. Peacock held that if a man had an hereditary predisposition to tubercle, his occupation followed in an atmosphere of dust, would favour the onset of tubercle, whereas if there were no such predisposition, he would be more likely to develop only chronic pneumonia"

During the present century researches into the dust diseases by many investigators in Great Britain, South Africa, Australia, the United States and Europe have established the fact that the most dangerous of the industrial dusts is free or uncombined silica (SiO_2) and that it sets up a nodular fibrosis or silicosis of the lungs. Combined silica or silicates are less dangerous, one exception being asbestos which is a complex silicate and produces a different type of fibrosis of the lung called asbestosis. More recent work on the radio-opaque dusts has shown that a worker who inhales such dusts (e.g. iron oxides, barium salts or finely divided metallic dusts) may have X-ray appearances of the lungs simu-

History of illness. Began to have pains in chest and dyspnoea February 1936 but continued at work until May when panel doctor referred him to hospital where he became an inpatient for some time. Tubercle bacilli were found in the sputum towards the end of his illness. Died 7.4.40. In receipt of compensation.

Gross morbid anatomy. (Abstract of autopsy report) Fingers clubbed. Pleura: adhesions at both bases. Lungs: Massive silicosis involving all lobes: emphysematous bullae both apices; old dry cavity in right lower lobe.

Histological sections. Sections of lungs showed massive conglomerate nodulation of silicosis; no evidence of tuberculosis.

Comment. Massive silicosis with bullous emphysema, and dry cavity of indolent tuberculosis.

(57) Male. Age 35 years.

Occupational history. Employed as a sandblaster of iron castings with a firm of vitreous enamellers from the age of 21 to 27. During this time he also occasionally mixed enamel and drew it out from the furnace. He stated that he never took any precautions.

History of illness. Began to complain of pain in chest in 1931: had attack of pneumonia in 1932: cough commenced in 1933; attack of haemoptysis in 1934. Admitted to hospital 6.9.34 with signs of subacute nephritis and advanced silicosis. No T.B. were found in sputum (5 tests). The possibility of malignant disease of lung was also discussed but he continued to improve and was discharged 29.11.34. Re-admitted cyanosed and moribund 29.8.35 and died on the following day.

X-ray examination. Film taken in 1934 showed advanced silicosis with suspicion of pulmonary neoplasm as well.

Gross morbid anatomy. (Abstract of autopsy report) Pleura: old adhesions at both bases, with extensive subacute pleurisy. Lungs: very extensive nodular silicosis throughout both lungs, most marked in the peripheral portions of the lower lobes; terminal acute congestion of upper lobes. Chronic nephritis with small peritoneal effusion.

Histological sections. Pleura was enormously thickened with fibrin which here and there contained collections of leucocytes; the fibrin was beginning to organize, with formation of collagenous fibres and new capillaries. In deep layers of pleura silicotic nodules were present.

Lungs showed many conglomerate nodules of silicosis with intervening lung tissue containing areas of focal emphysema, atelectasis or early broncho-pneumonia. Mediastinal glands exhibited small areas of caseation with typical giant cells.

Comment. Very extensive nodular silicosis of somewhat unusual distribution (i.e. most marked in peripheral portions of lower lobes) with minimal tuberculosis of mediastinal glands and terminal nephritis and broncho-pneumonia.

(58) Male. Age 55 years.

Occupational history. Had been employed at a small arms factory for 38 years, including about 20 years as sandblaster. Prior to this had had odd jobs, the nature of which is not known. He had worn a helmet-mask, overalls and rubber gloves whilst at work.

History of illness. Had been examined every 3 months whilst sandblasting; had been off work for a fortnight in 1941 through illness; apart from this had made no complaints of ill health until he broke down in April 1942 with extreme fatigue and obstinate cough, and silicosis was diagnosed. Died 9.4.43.

X-ray examination. Report stated: Long standing silicosis with signs at right upper lobe of ?tuberculosis ? collapse.

Gross morbid anatomy. (Abstract of autopsy report). Pleura: old adhesions at left apex, recent pleurisy at base; Lungs: Massive silicosis right and left upper lobes with nodules in lower lobes; subpleural nodules also: emphysema of focal type; fibrocaseous tuberculosis in both upper lobes and right lower, with cavities in left upper lobe; area of terminal pneumonia in left lower lobe.

Histological sections. Microscopical examination of lungs confirmed the presence of tuberculosis; conglomerate nodules of silicosis some of which had caseous centres; numerous asbestosis bodies were found also but the histological picture was that of typical silicosis with tuberculosis: hilum gland contained conglomerate nodules of silicosis almost entirely replacing normal lymphoid tissue. Aorta showed extensive collagenous thickening of media and round cell infiltration of vasa vasorum.

Comment. Massive silicosis with emphysema accompanied by tuberculosis and syphilis of aorta. Terminal pneumonia. Although typical asbestosis bodies were found in sections no history of exposure to asbestos could be obtained.

(59) Male. Age 42 years.

Occupational history. Sandblaster for 25 years at Foundry N.

History of illness. Three abdominal operations for gastric and duodenal ulcers. No details of chest illness until admission to hospital October 1945 with septic pneumonia from which he died.

Gross morbid anatomy. (Abstract of autopsy report). Trachea and main bronchi filled with muco-pus. Pleura: Dense adhesions lower part of left and apical portion of right sac. Lungs: much engorged: both showed extensive fibro-caseous tuberculosis involving all lobes, with large cavity in left lower lobe. This cavity had ragged walls and thrombosed vessels crossing it; it had become secondarily infected with foul smelling pus. Both lungs also contained numerous silicotic nodules. Small pericardial effusion. Extensive tuberculous meningitis.

Histological sections. The meninges and pus from the basal lung cavity contained tubercle bacilli and streptococci. Discrete and conglomerate nodules of silicosis were present in the lung tissue.

Comment. Nodular silicosis accompanied by pulmonary tuberculosis with dissemination to meninges, and a terminal streptococcal infection of a tuberculous pulmonary cavity.

(60) Male. Age 31 years.

Occupational history. Sandblaster 1927–1933 followed by 1½ years as zinc sprayer. Further details not known.

History of illness. No details known. Examined by Silicosis Board 29.9.36 and certificate of total disablement on account of silicosis accompanied by tuberculosis (sputum contained T.B.) issued.

Gross morbid anatomy. (Abstract of autopsy report). Tracheitis. Pleura: right lung adherent to chest wall and left lung at apex behind. Lungs: coarse nodular fibrosis of silico-tuberculosis on both sides, more on right; cavities at both apices; terminal oedema and congestive heart failure.

Histological sections. Microscopical examination of lung confirmed presence of silicosis and tuberculosis.

Comment. Nodular silicosis accompanied by pulmonary tuberculosis with cavity formation. Death from congestive heart failure.

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Nodulation (*X-ray*). A pattern seen in X-ray films of the chest, consisting of numerous rounded shadows or nodules usually about 5 mm. in diameter.

Oil Sand. Sand bonded with oil and used for making cores.

Open Hearth Furnace. A steel making furnace with an open hearth and regenerators to preheat air and gas for combustion. May be acid or basic.

Owen's Jet Dust Sampler. An instrument for taking quick, nearly instantaneous samples of the dust in a dust cloud. The dusty air is first drawn through a chamber where the air is made moist, and then at high speed through a narrow jet. This causes the moisture in the cloud to condense, and coat each dust particle with water. The wet particles are caught on a microscope cover glass and then examined under the high power microscope to enable dust concentrations and size distributions to be determined. The instrument does not catch all the dust particles.

Parting Sand. Sand without a bonding agent used to place between two layers of bonded sand so that they will not adhere and can be separated at a later stage.

— *powder.* A powder used in the same way as parting sand and also sprinkled on patterns to prevent adhesion of the sand.

Pattern. A wooden or metal replica of a casting, and round which sand is rammed to impart to it the desired shape.

Pneumoconiosis. This is a shortened form of the original term *pneumonokoniosis* introduced by Zenker in 1866, and really means "dust in the lungs." It is usually considered to be a general term embracing all forms of fibrosis or scarring of the lungs caused by dust, such as silicosis and asbestosis. Pneumoconiosis, as used by pathologists, denotes a fibrosis of the lungs made up of reticulin and collagen (q.v.).

Pouring. Introducing molten metal into a mould. See Teeming and Casting.

Refractory. High melting point material in which molten metal can be contained (e.g. silica sand, ganister, sillimanite, zircon, etc.).

Reticulin Fibre. (Path.) This is a fine connective tissue fibre staining black with silver impregnation methods. It may develop into collagen (q.v.).

Reticulation (*X-ray*). A term which means a "network," used to describe a pattern of shadows seen in X-ray films of the chest. See also *dust reticulation*, a term used in pathology.

Risers. Columns of molten metal rising from a casting which provide a reservoir of molten metal to feed into the casting while it contracts on cooling. The term also refers to the openings through the sand mould made to contain the columns of metal.

Rumbler. A cylindrical rotating box in which light castings are cleaned by friction of the castings against each other when the box is rotated.

Sand. High silica refractory used for making moulds.

Backing ———. Inferior quality sand used for the back of the mould and which does not come into contact with the molten metal.

Dry ———. The sand composing a mould which has been dried before introducing the molten metal.

Green ———. Sand containing about 5 per cent moisture. A mould which has not been dried before introducing the molten metal.

——— *blasting.* The cleaning of castings by impingement of a stream of sand under pressure. See Blasting.

——— *slinger.* A machine used to propel a stream of sand around a pattern to form a mould.

Shotblasting. The cleaning of castings by propelling a stream of metal shot against them. (See Blasting).