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THE PRACTICE OF INDUSTRIAL MEDICINE

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with a chapter on

The Hazards of Coal Mining

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With 8 Diagrams.

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taken at present as indicating anything more than previous inhalation of asbestos dust. . . .

Disablement produced by the Asbestos Fibrosis. Regarding the amount of disablement produced by the development of pulmonary fibrosis in asbestos workers, for a number of years this is surprisingly slight, even more so than is generally the case in silicosis. This is partly due to the character of the disease, and partly to the nature of the work, which in the majority of these processes does not involve much physical exertion. The affected person may, and often does, continue at work, with occasional intermissions, latterly, due to exacerbations of bronchitis, until the condition is advanced although he suffers increasing inconveniences from shortness of breath, on exertion. Sometimes a terminal broncho-pneumonia, or other acute infection, commences while still at work, and there is no long period of invalidism.

There is no doubt but that fibrosis of the type produced by asbestos can of itself lead to complete disablement and to a fatal termination, and this in the absence of a superadded tuberculous infection."

Age and sex have no effect on the incidence of asbestosis, which is influenced by length of employment and the type of work (i.e., dustiness). Though deaths have been recorded after only ten months' exposure to asbestos dust, Merewether found no case of asbestosis with less than five years' exposure. Spinners showed the lowest incidence (dust counts confirmed that this was the least dusty occupation) and though nothing less than the complete elimination of asbestos dust can be accepted as an ideal objective, as a practical standard, an atmosphere showing a dust contamination of something less than a spinning room might be regarded as a reasonably safe level. The National Institute of Health (the Industrial Hygiene Division), U.S.A. has suggested that the maximum permissible concentration is five million particles per cubic foot of air, but that proper control should reduce this to 2·5 million particles per cubic foot (approximately eighty-seven particles per cc.).

The Prevention of Silicosis and Asbestosis. (1) The substitution of less dangerous materials, for example, the use of carborundum type wheels instead of sandstone wheels and alumina instead of flint for embedding pottery (see pages 93 and 122), the substitution of hydro-blasting (i.e., a jet of water and sand) for sandblasting in cleaning metal castings.

(2) Suppression of dust at the source, for example, the prohibition of racing or dressing sandstone wheels except under special exhaust ventilation, the wetting of coal or rock when drilling in mines or quarries (Warner, 1943), the enclosure of asbestos carding machines, a high standard of local exhaust ventilation for all dusty processes, and the use of vacuum methods of cleaning instead of hand cleaning.

(3) Segregation of dusty processes so that such processes are carried on mechanically in a room which workers do not enter.

(4) Protection of workmen, for example sandblasters' helmet, but this method should never be regarded as a substitute for other measures, first because it is not external to the worker (see page 57) and secondly, respirators and masks neither fit sufficiently well nor are sufficiently efficient to afford full protection.

(5) Medical examinations (*a*) pre-employment, (*b*) periodic to detect early signs of pulmonary damage and especially to discover tuberculosis.

Compensation Schemes for Silicosis and Asbestosis.[3] The Silicosis and Asbestosis (Medical Arrangements) Act, 1930, and the Silicosis and Asbestosis (Medical Arrangements) Scheme, 1931, and other regulations, established a Medical Board (with headquarters in Sheffield), which acting in panels of not less than two is required on request to examine a workman, to issue (or refuse) a certificate that a workman is totally disabled from silicosis or asbestosis or suffering from either disease to such a degree to make it dangerous for him to continue at work in the industry or process. In the event of a certificate being granted the workman is entitled to compensation either from the employer or from a general compensation fund. The Board is required to certify after a *post mortem* examination, which should be performed by or in the presence of a member of the Board, whether deaths of workmen engaged in scheduled occupations are due to silicosis or asbestosis.

All new workmen entering scheduled employment,[4] unless over twenty-one years having been employed for three years in a process included in the schedule or in a process not included in the schedule but subject to a compensation scheme, or under twenty-one years of age having been previously examined and passed, must be examined by a member of the board within two months. All workmen employed in schedule process must be examined periodically at prescribed intervals. In both cases the Board has compulsory powers of suspension if the workman is found to be suffering from silicosis or asbestosis to a dangerous degree or from tuberculosis. Applications for medical examinations for the purpose of certification should be made to the

[3] From April 1st, 1945, administration was transferred from the Home Office to the Ministry of National Insurance.

[4] The most important process being grinding of metal by means of sandstone, and work incidental thereto, and racing of grindstone (sandstone) wheels; sandstone quarrying; mining, drilling and blasting of silica rock, and when incidental to mining other minerals; grinding and crushing of silica rock, flint, or any material containing not less than 80% of silica and manipulation (including manufacture of bricks) of same; any operation underground in tin, haematite and slate mining, getting and manipulation of granite; sandblasting; certain process in steel and pottery manufacture, including crushing of silica and flint, freeing, castings of silica (except when done on foundry floor), manufacture of china and earthenware, polishing and grinding glossed ware and tile slabbing; breaking, crushing, disintegrating, opening and grinding of asbestos, and the manufacture of asbestos textiles, insulation slabs, mattresses (and the repair of the same), cleaning machinery, chambers and flues, etc., manufacture of silica flour and artificial stone.

Sanders (1944) has given a similar description of a fine pseudo-nodulation (easily mistaken for military tuberculosis) unaccompanied by fibrotic reaction and not disposing to tubercle, in electric arc welders who have worked in confined spaces for long periods of time which he ascribed to iron deposits in the lung. He suggested the term "siderosis of welding" and emphasised that the condition was not progressive if the welder ceased to work in confined and ill-ventilated spaces.

According to the composition and covering of the electrodes electric arc welders may be exposed to fume or dust of zinc, manganese, silica and asbestos (welding rods are of two types (*a*) uncovered, (*b*) covered either with chemical flux or asbestos).

Metal fume fever (see page 144) may result from welding of galvanised metal, especially if this is done in confined spaces.

Nitrous fumes, carbon monoxide and carbon dioxide, and in the case of oxy-acetylene welding, acetylene and impurities may be present in the fumes from welding, and it is said, with what justification is doubtful, that welders are especially liable to suffer from bronchitis. Krasno and others (1943) have suggested that effects due to fume can be avoided by supplying fresh air inside the usual type of welder's hood. In confined spaces the supply of fresh air to the welder is essential; otherwise headache, giddiness, nausea, vomiting and symptoms due to nitrous fumes, such as pharyngitis and bronchitis will occur.

Bridge (1934) has reported a number of cases of acute poisoning from nitrous fumes in oxy-acetylene welders.

Mercury. Mercury poisoning may be due to (1) mercury vapour or air-borne particles, (2) contact of mercury fulminate with the skin, giving rise to a dermatitis, and (3) organic mercurial salts.

Mercury vapour and air-borne particles. Mercury is most often absorbed through the lungs in the form of vapour. The refining of mercury, the recovery of gold and silver, water gilding, the making of thermometers and scientific instruments, all involving the heating of mercury and in the absence of good local ventilation, mercury vapour may be present in the air in sufficient quantities to give rise to poisoning. In the manufacture of surgical dressings, and in hatter's furriers' workshops (mostly of the backroom type) where felt and furs are brushed or "carrotted" with mercury nitrate solution, there may be sufficient dust of textile fibres coated with mercury to give rise to poisoning. The classical symptoms of mercury poisoning, often therapeutically induced in the treatment of syphilis before the introduction of arsphenamines and bismuth compounds, are salivation, loss-