FILE NAME: Sprayed Asbestos (SPRA)

DATE: 1962

DOC#: SPRA016

DOCUMENT DESCRIPTION: Medical Journal Article - Radiology of Some Rarer Dust Diseases

Spraying asbestos 483, Fig. 9.

Scot. med. J., 1962, 7: 478

RADIOLOGY OF SOME RARER DUST DISEASES*

(Barytosis, Asbestosis and Siderosis)

C. K. Blum

Chest Clinics, Victoria Hospital, Group, Glasgow

UNLIKE gas and vapour, dust and fumes are composed of particles which are inhaled into the lungs. **Dusts** are particles or aggregates of particles of 1 to 150 microns in diameter. **Fumes** are of the size of 0.2 to 1 micron. **Smoke** is less than 0.3 micron in diameter.

Gases and **vapours** include simple asphyxiants, chemical asphyxiants, irritant gases, organo-metallic gases and anaesthetic vapour.

Fog and **mist** are essentially tiny liquid droplets condensed about solid particles, such as carbon, as a nucleus.

The defences against dust or fumes are: (1) Nasal defence with its mucous secretion, (2) ciliary action of nasal and bronchial epithelium; and (3) the phagocytic properties of cells. As the average size of dust particles is up to 5 microns, a great number of particles can get into the lung alveoli, which measure about 100 microns in diameter. The limiting factor of dust particles found in the lung is the diameter of lymphatic channels and not the alveoli. The amount of dust, the type of dust inhaled and the individual susceptibility differ considerably in different individuals.

Dust may be organic or inorganic. Inorganic dust may be: (1) Inert or non-proliferative dust; and (2) active or proliferative dust. Mixture of (1) and (2) results in a modified proliferative reaction, which is further modified by infection with resulting inflammation and emphysema. Inert dust does not produce a specific reaction in the lungs. Active dust is aggressive, producing reactive and proliferative changes in the lymphatic, vascular and parenchymatous pattern.

The result of prolonged inhalation of mineral dusts, such as those of granite, flint, [illegible] and asbestos, is a reactive state of the lung tissue comprised in the generic term of pneumoconiosis. It describes at times pulmonary fibroses, which result from the inhalation of noxious or innoxious dusts, which are different mixtures of substances in which different pathogenicity determines the outcome of resulting lung changes. Many of these lung changes are scarcely to be considered as lung diseases; they may exist without impairing physical efficiency; they have been termed benign pneumoconiosis (anthracosis, siderosis, chalicosis). On the other hand, dust containing a high concentration of silica produces more or less serious forms of pulmonary changes of considerable medical and economic importance (malignant pneumoconiosis). Admixtures of alumina, carbon, gypsum and hematite result in a temporary or permanent modification of the reaction to the silica content of the inhaled noxious material, retarding or inhibiting its deleterious effects on alveolar lung tissue. Thus silicates vary greatly in their pathogenicity; some are nearly completely inert or mildly noxious, while others may even be seen producing a retarding effect upon the action of free silica. As a rule the inhalation of organic dust produces no pneumoconiosis.

Inhaled particles of 10 microns or less are phagocytosed and carried through pulmonary lymphatics into the regional peribronchial and hilar lymph nodes. Prolonged exposure to high concentrations leads to accumulation in the lymphatics surrounding the pulmonary vessels. These deposits are seeen in the X-ray picture if the material is radio-opaque—as in siderosis, barytosis and anthracosis.

*Based on a lecture delivered at the meeting of the Scottish Radiological Society on 24th February 1962

Table 1. International classification of persistent radiological opacities in the lung fields provoked by the inhalation of mineral dust (Geneva Classification 1958*)

	No pneumoconiosis	Suspect	Pneumoconiosis											
Type of opacity			Linear opacities	Small opacities									Large opacities	
Qualitative features	0	/	1	p			m			n			A	B C
Quantitative features				1	2	3	1	2	3	1	2	3		
Additional symbols	(co)/(cp)	(cv)	(di)	(em)			(hi)			(pl)		(px)	(tb)	

*Including coal and carbon dusts.

Definitions and comments

The object of the classification is to codify the radiological appearances of the pneumoconioses in a simple, easily reproducible way. It is intended to describe the radiographic appearances of the persistent opacities associated with pneumoconiosis, not to define pathological entities, nor to take into account the question of working capacity.

Where there is an appreciable difference in the appearance of the two lungs, the two appearances may be described separately, beginning with the right lung.

No pneumoconiosis	0	No radiographic evidence of pneumoconiosis	
Suspect opacities	Z	Increased lung markings	
Pneumoconiosis			
Linear opacities	L	Numerous linear or reticular opacities, the lung pattern being normal accentuated or obscured	
		The following types are defined according to the greatest diameter of the predominant opacities	The categorization depends on the extent and the profusion of the opacities
Small opacities	1	p Punctiform opacities. Size up to 1.5 mm	**Cat. 1.** A small number of opacities in an area equivalent to at least two anterior rib spaces and at the most not greater than one-third of the two lung fields.
		m Micro-nodular or miliary opacities Greatest diameter between 1.5 and 3 mm	**Cat. 2.** Opacities more numerous and diffuse than in Cat. 1 and distributed over most of the lung fields.
		n Nodular opacities Size between 3 and 10 mm	**Cat. 3.** Very numerous profuse opacities covering the whole or nearly the whole of the lung fields
Large opacities	2	A An opacity having a longest diameter of between 1 and 5 cm or several opacities each greater than 1 cm, the sum of whose longest diameters does not exceed 5 cm	
		B One or more opacities, larger or more numerous than those in Cat. A. whose combined area does not exceed one-third of one lung field	
		C One or more large opacities, whose combined area exceeds one-third of one lung field.	

Additional symbols.

(co)	abnormalities of the cardiac outline To be replaced by (cp): cor pulmonale, if this condition is strongly suspected.		
(cv)	cavity.	(di)	significant distortion of the intra-thoracic organs
(em)	marked emphysema	(hi)	marked abnormalities of the hilar shadows
(pl)	significant pleural abnormalities.	(px)	pneumothorax.
(tb)	opacities suggestive of active tuberculosis.		

1. The choice of order of the symbols is left to the convenience of the physician
2. The background of small opacities should be specified as far as possible

The merits of classification of dust diseases into different types are debatable. The radiological description is important, and should be as accurate as possible, taking into account the anatomical lung system, including hila, bronchi, alveolar structure, pulmonary vascular texture, especially arterial pattern, thoracic deformity, pleural involvement (such as in asbestosis) and cardiac contours, with special relation to right ventricular enlargement and features of *cor pulmonale*.

The Geneva classification of 1958, applicable to all dust diseases, has superseded all previous descriptive and clinical terms of dust inhalation diseases, and should be accepted as standard description. This is described in Table I. The radiological terms applied to dust diseases refer to the appearances: of linear or reticular opacities; punctiform opacities (1.5 mm. in size); micro-nodular or miliary opacities (1.5-3 mm. in size); nodular opacities (either single or multiple, having diameters of 1-5 cm.), which can be easily differentiated against the early appearance of suspected inhalation disease, in which only increased lung markings are found.

The uniform acceptance of the Geneva terminology (1958) would clearly obviate the radiological classification of reticulation or pinhead mottling, nodulation, coalescent nodulation, with development of massive shadowing and multiple fluffy shadowing, which has been hitherto applied to the classification of pneumoconiosis in coal miners, or supersede the classification for pure silicosis of Stages I, II and III. It would comprise the classification of Sampson (1955), Cochrane *et al.* (1951) and Cole (1946), which is embodied in the additional symbols, usefully applying clinical manifestations and features.

The essentials to the diagnosis of dust diseases are an exhaustive occupational history with regard to the exposure to dust, fumes, gases and vapours, and a good radiological picture of the lungs. The occupational history should cover the patient's whole life, and the length of employment in different jobs. The clinical findings are sometimes negligible.

Our material seen in the chest clinics of the Victoria Infirmary Group in the last few years covers a fair number of pure pneumoconioses of coal miners, cases of pure silicosis and silico-tuberculosis, of boiler scaler lungs, and of Kaplan's syndrome in coal miners. In addition, we have examined a considerable number of patients exposed to different types of dust and found seven cases of barytosis (referred by Dr A. T. Doig), four cases of asbestosis and thirteen proven cases of siderosis or sidero-silicosis, amongst 15,000 cases of non-tuberculous chest diseases.

Barytosis (7 cases). This condition due to inhalation of barium dust, is generally classified as an inert dust disease (Figs. 1-5). Most of the reports of this condition come from Italy; they are confined to clinical and radiological aspects and do not include pathological findings. The affected men have been engaged in mining, sorting, grinding and bagging the ore *barytes* ($BaSO_4$) under conditions which exposed them to very high concentrations of dust. The most intense dust clouds were found at grinding operations and measures of suppression were inadequate or entirely lacking.

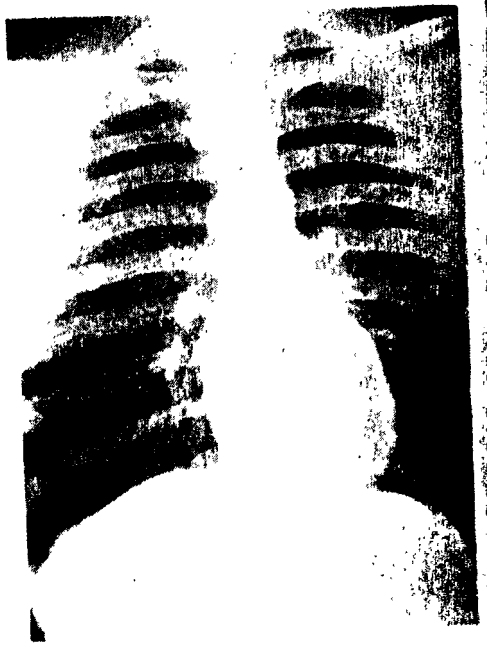


Fig. 1. Barytosis. G. W., aged 26 years. Morning cough; no dyspnoea, no sputum. Five years' exposure, early changes.

Fig. 2. Barytosi dyspnoea. Thirte metrical change

Fig. 3. Barytosi

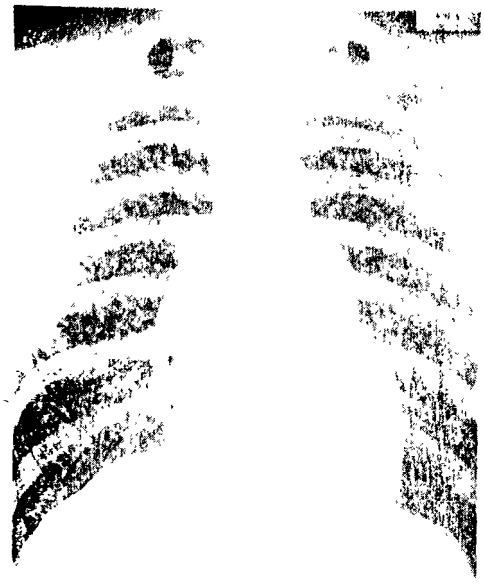


Fig. 2. Barytosis. W.S., aged 33 years. Cough: no dyspnoea. Thirteen years' exposure: widespread synnodular changes.

Fig. 4. Barytosis. J.W., aged 38 years. No cough, no sputum, no dyspnoea. Eighteen years exposure, mostly in the mill.

Fig. 3. Barytosis. W.S., lateral view.

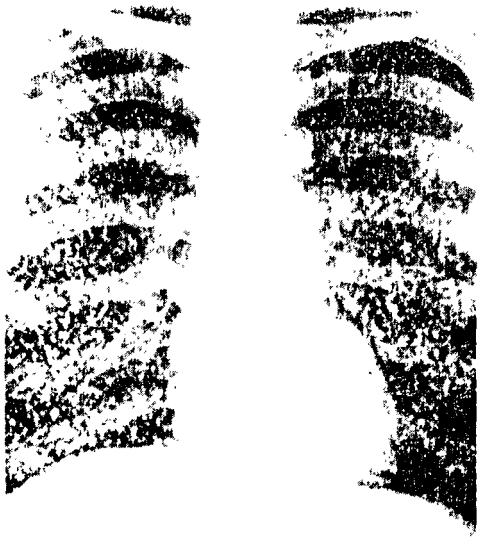


Fig. 5. Barytosis. A.S., aged 30 years. No cough, no sputum, no dyspnoea. Seven years' exposure: extensive "snow flake" appearance.

The first case was described by Dott (1938) in a man who had been employed for 27 years in a *barytes* mill, but, in addition to marked X-ray changes with nodular shadows of the size of a pea, this man complained of weakness, cough, marked dyspnoea on exertion and occasional attacks of angina pectoris. The clinical and radiological picture was, therefore, very suggestive of silicosis, although the dyspnoea could have been cardiac in origin.

Subsequent writers, however, make special mention of the trivial nature of the clinical picture, in spite of well-marked radiological changes. Arrigoni (1933) says that this type of pneumoconiosis is characterized by a marked contrast between the benign subjective and objective clinical symptoms (or even their entire absence), and the gravity of the radiological picture. Preti and Tallini (1938) and Spedini and Valdini (1939) make similar statements. The latter observers mention a diminution of the radiological abnormalities when the worker leaves the dusty environment.

The condition of baritosis does not seem to convey any increased tendency to tuberculosis. Indeed, Dott (1938) actually wonders whether it might not even exert a protective action against that disease. The incidence of acute forms of lung infection such as pneumonia, however, seems to be high. Camba (1951) also found rather a high incidence of pleuro-broncho-pulmonary affections, active or healed, on X-ray examination of fourteen Sardinian miners. He did not regard these as being related to occupation.

Experimental studies. Arrigoni (1933) succeeded in producing nodulation in X-ray films of guinea-pigs and rabbits subjected to the inhalation and intratracheal injection of *barytes* dust. Histologically, he speaks of this lesion as a 'dust granulosis' rather than a pneumoconiotic nodule, although he declines to express an opinion on the ultimate and possible fibrous transformation of the granuloma. The intense opacity of the nodules in X-rays is commented on by various writers, and Pinchert (1950 *a* & *b*) concludes that baritosis is a pneumoconiosis of a benign character which can be readily distinguished from silicosis in that (1) the radiographic changes may appear after only a few months' exposure, (2) the condition does not cause death, (3) there is no frequent association with tuberculosis, and (4) the X-ray shadows diminish on removal of the subject from exposure to dust.

Incidence and relation to silicosis. No case has been described in Great Britain so far as is known, but reference to the condition has been made in the United States by Pendergrass (1942) and Pendergrass and Leopold (1945) who also regard it as a form of benign pneumoconiosis. Although most of the reports in the literature on the pneumoconiosis of barium workers indicate that the condition is a benign one it must not be forgotten that the workers may be exposed to silica dust derived from the ore. In some areas grinding is performed between millstones which are themselves highly siliceous and which wear down rapidly in use, thus contributing to the silica content of the dust (Doig, 1956).

Asbestosis (4 cases). This has been observed in asbestos workers, asbestos being a hydrated magnesium silicate, used in the manufacture of asbestos cloth and materials (Figs. 6–11).

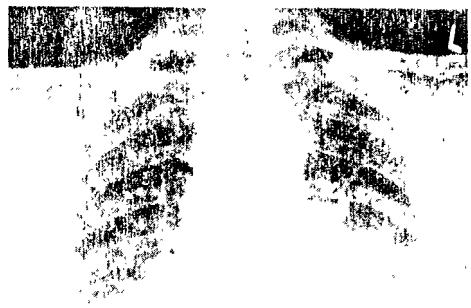
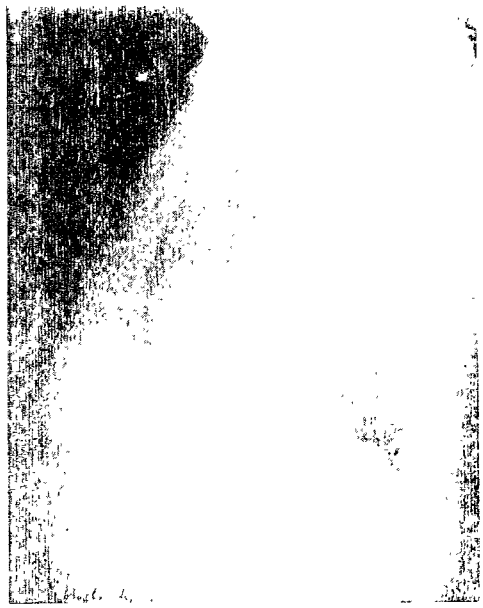
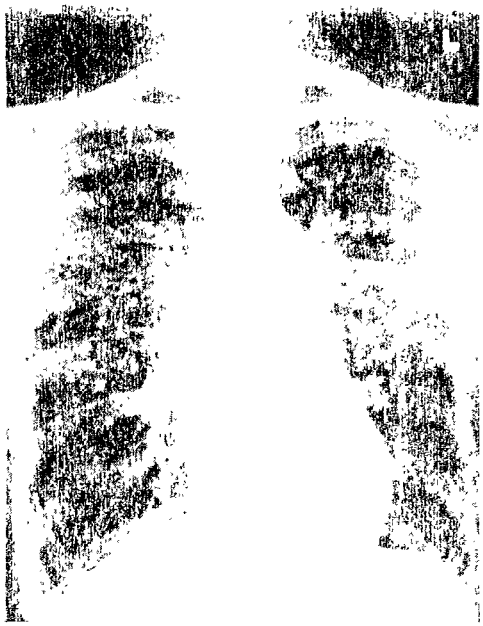
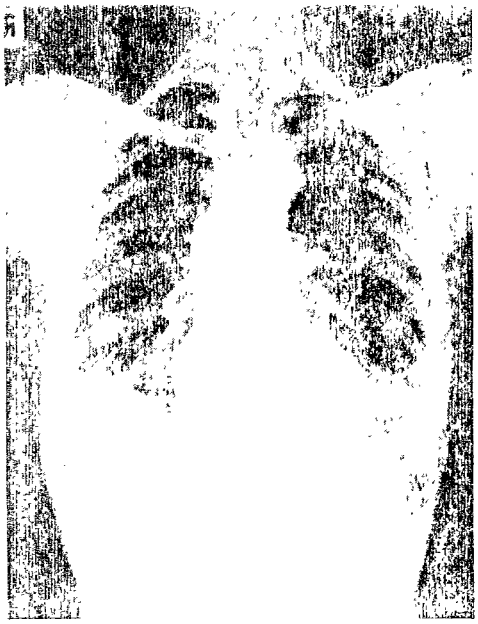


Fig. 6. Asbestosis. A.B., aged 61 years, asbestos worker for 18 years. Chronic bronchitis, increasing dyspnoea, moderate clubbing of fingers, examined 1955.
Postero-anterior view, showing widespread lung and pleural involvement.







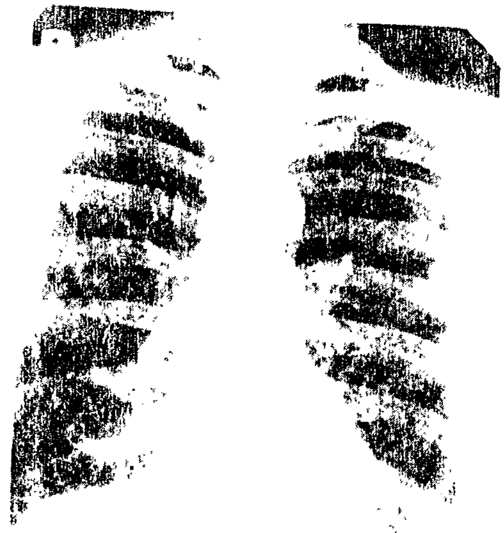


Fig. [illegible] Asbestosis. [illegible] McD [illegible] aged [illegible] years. [illegible] worked [illegible] years [illegible]. Cervical spine [illegible] [illegible]. Fine [illegible] [illegible] no pleural [illegible].

Siderosis (F[illegible] cases). This condition [illegible] [illegible] benign pneumoconiosis but [illegible] [illegible] [illegible] mine [illegible] [illegible] [illegible] as abrasive [illegible] grinders and [illegible] metal grinders and [illegible] welders all can develop, in addition to [illegible] siderosis, silicotic changes in the lungs due to an admixture of iron or dust with silica. Doig (1960) has summarized knowledge about the [illegible] in the lungs in a paper on non-silicotic lung disease.

Asbestosis

This [illegible] collective [illegible] applied to a group of fibrous [illegible] which are valuable on account of their [illegible] and insulating properties (asbestos [illegible] inconsumable). The raw material from Rhodesia, Canada and Italy [illegible] and processing can be spun into yarn [illegible] into textiles. In these operations [illegible] amounts of dust are produced containing [illegible] small fibres which [illegible] produce [illegible] fine fibrosis [illegible] mechanical or chemical action.

Radiological changes in asbestosis (Hun[illegible] 1961) [illegible] the pleurae and lungs.

Pleural changes. Th[illegible] or bilateral parietal [illegible] obliteration of costo-phrenic [illegible] pericardial adhesions or pleural fibrosis [illegible] cardiac and diaphragmatic outlines [illegible] defined producing [illegible] of the [illegible] heart. The movements of the [illegible] restricted. Calcification of pleurae [illegible] usually bilaterally and often [illegible] the mid and lower zones [illegible] [illegible]. (Silicotic [illegible] plurally in the upper zones and [illegible] common.) Calcified [illegible] cases of exposure to [illegible] dusts [illegible] [illegible] talc, calcium [illegible], mica or [illegible] (insulation).

Lung changes. The[illegible] may be grouped [illegible] categories.

[illegible] diffuse, [illegible] Here there are [illegible] striated [illegible] [illegible] causing [illegible] translucency of the lung with blurring of [illegible] [illegible].

[illegible] [illegible] diff[illegible] honeycomb [illegible] and [illegible].

[illegible]

[illegible] honeycomb [illegible]

[illegible] pattern. Less frequently well marked nodular lung pattern is found [illegible] associated silica exposure (mining) [illegible] indistinguishable from silicosis.

[illegible] [illegible] high percentage of silica and iron in the [illegible] Bin[illegible] Mine area.

Haemosiderosis

Haemosiderosis of lungs [illegible] [illegible] iron turners, grinders, [illegible] fitters, solder[illegible] welders [illegible] iron workers and [illegible] foundry. The typical X-ray features [illegible] with fine pinhead opacities and [illegible] dense and sharp (Figs. 12 & 1[illegible]).

Pulmonary haemosiderosis of [illegible] should be differentiated from [illegible] haemosiderosis. The radiological features [illegible] (Figs. 14 & 15) [illegible] small infiltrations producing a mottled ap-

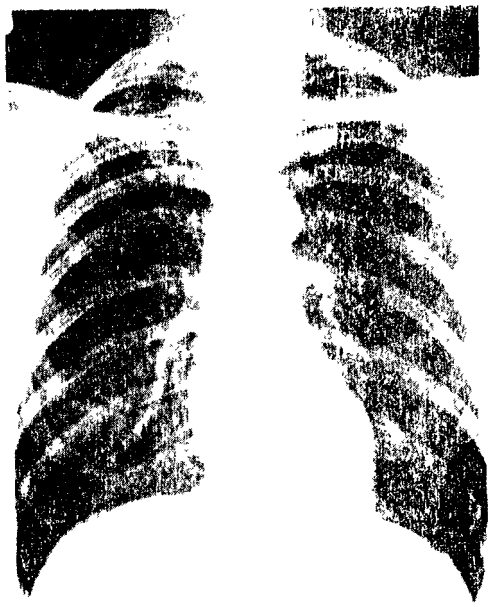


Fig. 12. Siderosis. I B., aged 45 years. Electric welder. Early changes; exaggeration of pulmonary vascular markings

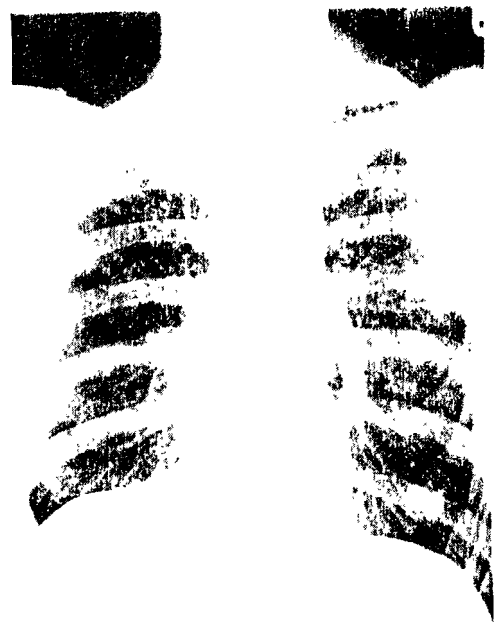


Fig. 14. Idiopathic pulmonary haemosiderosis. I R., aged 68 years. Typical miliary pinhead nodulation

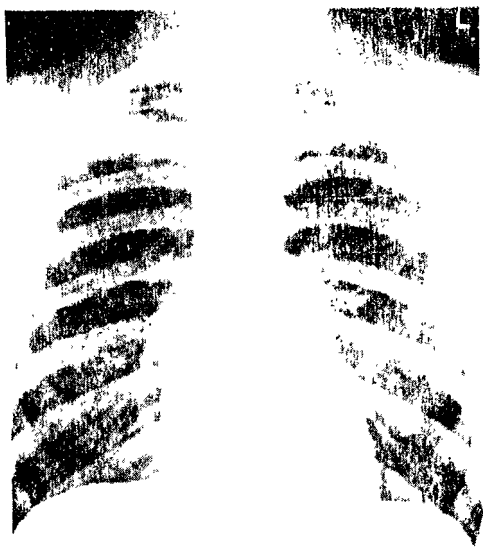


Fig. 13. Siderosilicosis. I. H., aged 36 years. Electric welder (in foundries) for 20 years. Well developed siderosis with nodulation. Patient exposed to some silica dust raised by other workmen

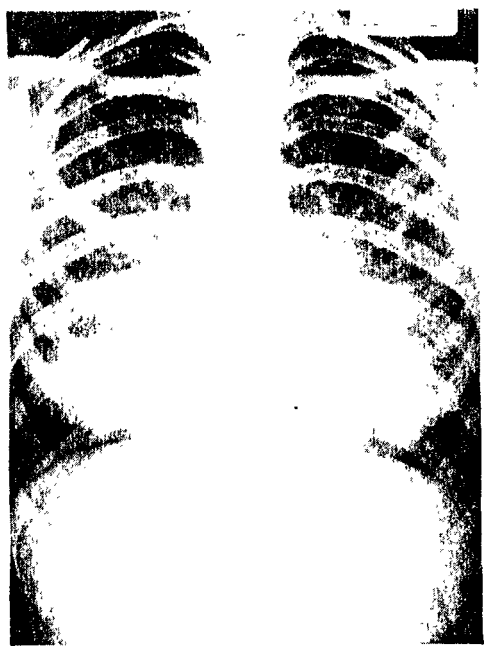


Fig. 15. Idiopathic pulmonary haemosiderosis. M. D., aged 20 years. Acute haemolytic phase

pearance which is distributed throughout both lungs. In the ... is not clearly defined ... alveoli ... filled with fibrin and blood ... walls of alveoli ... partly fibrotic and the elastic fibrillation is reduced.

The clinical features comprise anaemia of hypochromic type, low serum iron values, increased haemolysis, urobilinuria, high reticulocyte count and normal fragility of erythrocytes.

A rare condition, pulmonary alveolar microlithiasis, should be considered in differential diagnosis. The radiological features are similar to those of ... sclerosis occurring in young people. There is extensive involvement of both lungs; the ... lesions are partly discrete ... with subpleural ... of bases. Histologically ... concentric lamellated ... situated in the alveoli, the alveolar walls usually being normal. Familial occurrence has often been found and the X-ray picture (Fig. 16) belongs to a man of 25 years ... 1943 who is still ... today ... brother presents the same X-ray ...

Fig. 16. ... brother discovered ...

DISCUSSION

... publication ... The International Symposium organized by the British Occupational Hygiene Society (Davies 1964) and the Proceedings of the Pneumoconiosis Conference in Johannesburg (Orenstein 1960), have greatly added to our understanding of the pathogenesis of many pulmonary diseases and of the control and prevention of dust diseases.

Interesting facts about pneumoconiosis statistics are contained in the Digest published by the Ministry of Power 1960. The statistics are no longer restricted to the mining and quarrying industry but also give details of ... disease in other industries. The Pneumoconiosis Field Research ... by the National Coal Board has contributed to the foundation of ... and ... condition. The important part ... the reactive changes to inhaled particles ... dissertation ... inhalation diseases ... tuberculous infection ... fibrosis ... development of ... and progressive pneumoconiosis has been discussed.

... only a few of the problems which are still unsolved today. The ... pneumoconiosis ... pleural ... It would appear ... still very scanty ... natural history of inhalation disease.

ACKNOWLEDGEMENTS ... D. A. F. D... for permission ... for allowing ... history of ... Dr. ... for their ... photographs ... Victoria Hospital Group for the photographs ... for ... help.

REFERENCES

ARRIGONI, A. (1933) La pneumoconiosi da barite *Med. d. Lavoro*, **24**, 461

CAMBA, R. (1951) Contributo alla conoscenza dell'azione delle polveri di barite sull'uomo *Med. d. Lavoro*, **42**, 221

COCHRANE, A. L., DAVIES, I., FLETCHER, C. M. (1951). Entente radiologique; step towards international agreement on classification of radiographs in pneumoconiosis. *Brit. J. industr. Med.*, **8**, 244

COLL, L. G. (1946) Pneumoconiosis. In *Clinical radiology*, edited by G. U. Pillmore. Vol. I. Philadelphia: Davis

DAVIES, C. N. ed (1961) *Inhaled particles and vapours*. Proceedings of an International Symposium organised by the British Occupational Hygiene Society. Oxford: Pergamon Press

DOIG, A. T. (1950) Iron in the lungs. *Arch. belges Méd. soc.*, **8**, 442

DOIG, A. T. (1956). Dust diseases, excluding fibrotic pneumoconiosis. In *Industrial medicine and hygiene*, Vol. III, edited by E. R. A. Merewether. p. 123. London: Butterworth & Co.

FEGI, L. (1938). Pneumoconiosis. In *Occupation and health*. Geneva: International Labour Office

FIORI, (1938). Pneumoconiosis. In *Occupation and health*. Geneva: International Labour Office

HICKS, D. FAY, J. W. J. ASHFORD, J. R. RAE, S. (1961). *The relation between pneumoconiosis and environmental conditions*. London: National Coal Board.

HURWITZ, M. (1961) Roentgenologic aspects of asbestosis. *Amer. J. Roentgenol.*, **85**, 256

MINISTRY OF POWER (1960) *Digest of pneumoconiosis statistics*. London: H.M. Stationery Office

ORENSTEIN, A. J. ed (1960) *Proceedings of the pneumoconiosis conference* held at the University of Witwatersrand, Johannesburg, 1959. London: Churchill

PANCHERI, G. (1950*a*) Su alcune forme di pneumoconiosi particolarmente studiate in Italia (tuo-pneumoconiosi e baritosi) *Med. d. Lavoro*, **41**, 73

PANCHERI, G. (1950*b*) L'aspirazione delle polveri in miniera con l'apparecchio Borsari. *Rass. Med. industr.*, **19**, 217

PENDERGRASS, E. P. (1942) Some considerations concerning the roentgen diagnosis of pneumoconiosis and silicosis. *Amer. J. Roentgenol.* **48**, 571

PENDERGRASS, E. P., LEOPOLD, S. S. (1945). Benign pneumonoconiosis. *J. Amer. med. Ass.*, **127**, 701

PRETI, L., PATRINI, P. C. (1939) *Report of 8th International Congress on Industrial Accidents and Occupational Diseases*. Leipzig: Thieme

SAMPSON, Cited by SHIPMAN, S. J. (1955) Diseases of the lungs. In *Oxford medicine*, Vol. 2, p. 116. Oxford: University Press

SPEDINI, I., VALDINI, P. I. (1939) Contributo alla studio della pneumoconiosi da barite. *Radiol. med. (Torino)*, **26**, 1