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RADIOLOGY OF SOME RARER DUST DISEASES*

(Barytosis, Asbestosis and Siderosis)

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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, sand and asbestos, is a reactive state of the lung tissue comprised in the generic term 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 course 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 effect 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 peribronchovascular and hilar lymph nodes. Prolonged exposure to high concentrations leads to accumulation in the lymphatics surrounding the pulmonary vessels. These deposits are seen 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.

*Sprague
asbestos
1983, Fig. 9.*

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

Type of opacity	No pneumoconiosis	Suspect	Pneumoconiosis													
	0	Z	Linear opacities	Small opacities									Large opacities			
			1	p			m			n			A	B	C	
Qualitative features				1	2	3	1	2	3	1	2	3				
Quantitative features																
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
		m	Micro-nodular or military opacities. Greatest diameter between 1.5 and 3 mm
		n	Nodular opacities. Size between 3 and 10 mm
		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
Large opacities	2	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.	(di)	significant distortion of the intra-thoracic organs
(cv)	cavity.	(hi)	marked abnormalities of the hilar shadows
(em)	marked emphysema	(px)	pneumothorax.
(pl)	significant pleural abnormalities.		
(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.

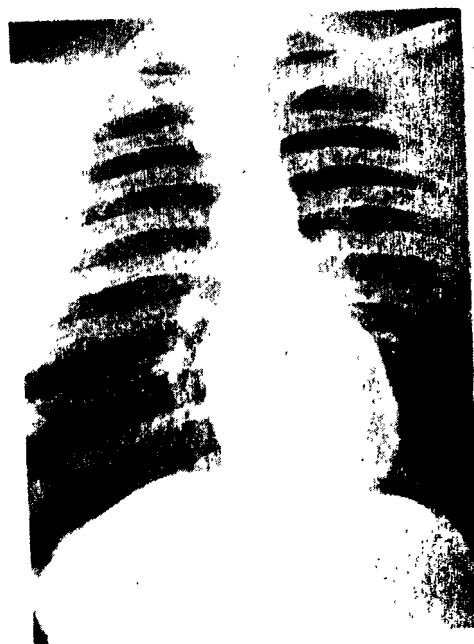


Fig. 1. Barytosis. G. W., aged 26 years. Morning cough; no dyspnoea, no sputum. Five years' exposure, early changes.

Fig. 2. Barytosis. dyspnoea. Third metrical change.

Fig. 3. Barytosis.

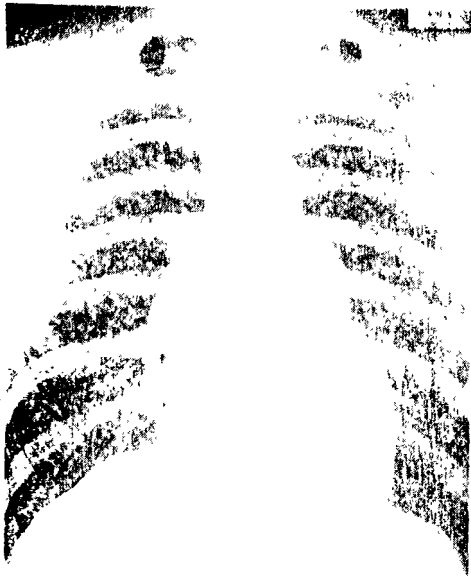


Fig. 2. Barytosis W.S., aged 33 years. Cough, no dyspnoea. Thirteen years' exposure, widespread symmetrical changes.



Fig. 4. Barytosis J.W., aged 38 years. No cough, no sputum, no dyspnoea. Fifteen years' exposure, mostly in the hilar.



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 From (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 Valdim (1939) make similar statements. The latter observers mention a diminution of the radiological abnormalities when the worker leaves the dusty environment.

The condition of barytosis does not seem to carry any increased tendency to tuberculosis; indeed, Fori (1935) actually wonders whether it might not even exert a protective action against that disease. The incidence of acute forms of lupercution, 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 the 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 Pincheri (1950 *a* & *b*) concludes that barytosis 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, is known, but reference to the condition has been made in the United States by Pendergrass (1942) and Pendergrass and Leopold (1947) who also regard it as a form of benign pneumoconiosis. Although most of the reports in the literature on the pneumoconiosis of baryum workers indicate that the condition is a benign one, it must not be forgotten that the workers may be exposed to silica 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 material (Fig. 6-11).

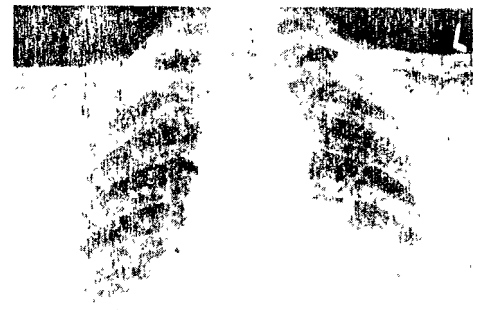


Fig. 6. Asbestosis. (A) 64 years' asbestos worker for 48 years. (B) Work each with severe bronchitis, increasing dyspnoea, morning cough, clubbing of fingers, cyanosed lips. Postero-anterior view, showing widespread lung and pleural involvement.

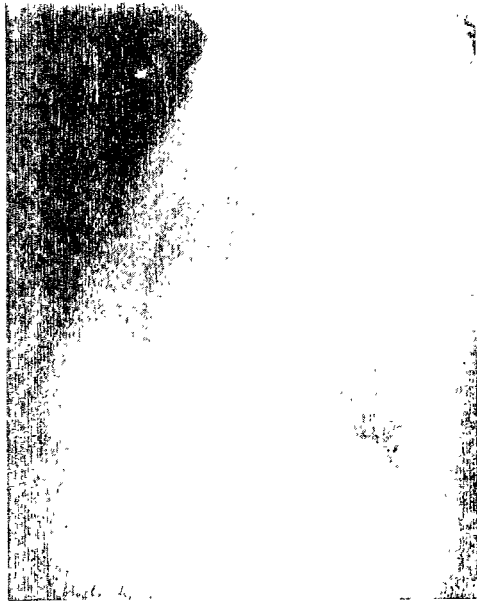


Fig. 7. A histological section of the tumor.

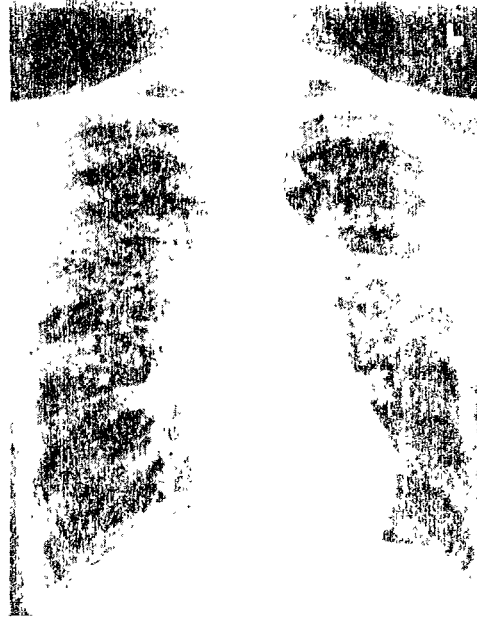


Fig. 9. A histological section of the tumor.

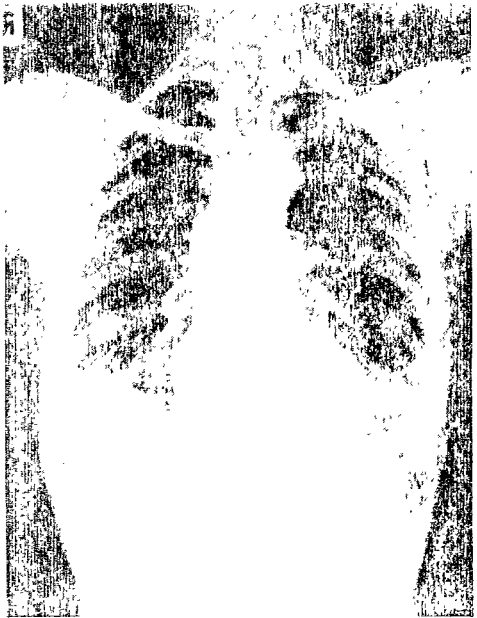


Fig. 10. A histological section of the tumor.



Fig. 11. A histological section of the tumor.

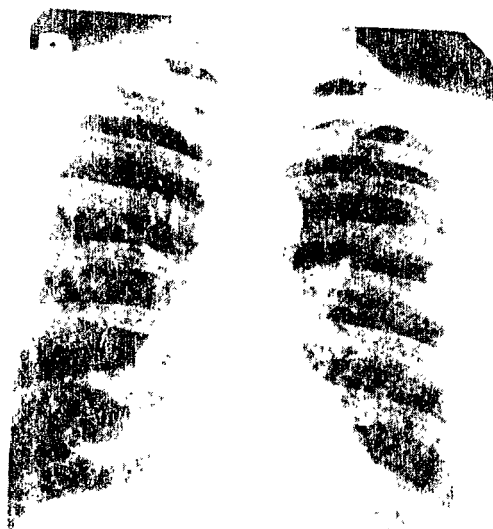


Fig. 11. Asbestosis (McDermott & Venn). A few weeks after removal of Central spinning mill dust. Note lung changes (left) and pleural thickening.

Siderosis (Iron dust). This condition is by no means a benign pneumoconiosis but is caused by minute mineral particles usually of iron or iron abrasives, ferrous oxides and boron, metal oxides and boron scales all can develop, in addition to chronic siderosis, silicotic changes in the lung due to an admixture of iron or dust with silica (Dojo (1950) has summarized knowledge about the presence of iron in the lung in a paper on non-silicotic iron disease).

Asbestosis

This is a collection of asbestoses applied to a group of fibrous silicates, all of which are valuable on account of their heat-insulating and insulating properties (asbestos is flame-incombustible). The raw material from Rhodes, Canada and Italy after identification and processing can be spun into yarn and woven into textiles. In these operations considerable amounts of dust are produced containing enormous small fibres which inhaled produce a severe fine fibrosis either by mechanical or chemical action.

Radiological changes in asbestosis (Himwitt (1961) are seen in the pleura and lungs

Pleural changes. These consist of unilateral or bilateral partial complete or complete obliteration of costo-phrenic sulci, pleural thickening, pleural adhesions or pleural fibrosis, pericardial adhesions or pleural fibrosis, pericardial and diaphragmatic outpouchings, well defined, producing pictures of the scalloped heart. The movements of the diaphragm are restricted. Calcification of pleura is seen usually bilaterally and often symmetrically in the mid and lower zones, often extending laterally (Silicotic plaques are seen symmetrically in the upper zones and are common). Calcified plaques are seen in cases of exposure to vermiculite dusts, in amphibole talc, calcium silicate or base metal smelting.

Lung changes. These may be grouped into categories:

Silicotic diffuse, non-nodular, non-calcified. Here there are no striated fibroclastic changes causing progressive reduction of transparency of the lung with hazy areas of consolidation.

Diffuse nodular, non-calcified. This is a difficult group to distinguish from silicosis, but the clinical history and H. S. stain are helpful.

Fibrotic honeycombing and H. S. stain negative.

Diffuse lung nodules, non-calcified, H. S. stain negative.

Focal nodular, non-calcified, H. S. stain negative.

Acute lung pattern. Less frequently well marked nodular lung patterns found due to a secreted silica exposure (impaired clearance and indistinguishable from silicosis).

Chronic nodular silicosis. These cases show a high percentage of silica and non-silicotic (Cox, Blum, Mine area).

Hemosiderosis

Hemosiderosis of the lung is caused by iron from iron oxides, iron scales, boiler scales, etc. from iron works and steel mills. The typical X-ray feature is enlargement with fine mottled opacities and enlargement of the heart and diaphragm (Figs. 12 & 13).

Pulmonary hemosiderosis of idiopathic type should be differentiated from iron poisoning and hemosiderosis. The radiological features are similar (Figs. 14 & 15). There are widespread small infiltrations producing a mottled ap-

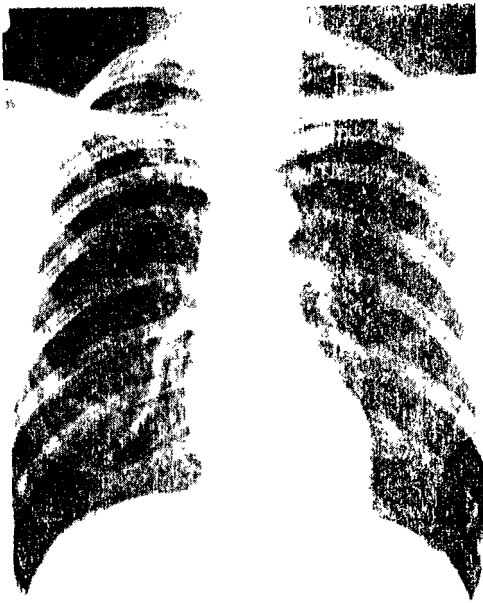


Fig. 12. Siderosis (I.B., aged 45 years). Electric welder. Early changes; exaggeration of pulmonary vasculature.

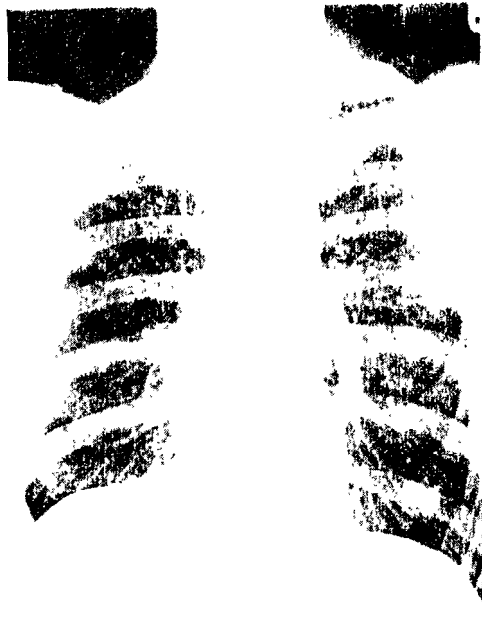


Fig. 14. Idiopathic pulmonary haemosiderosis (I.R., aged 68 years). Typical miliary pinhead nodulation.

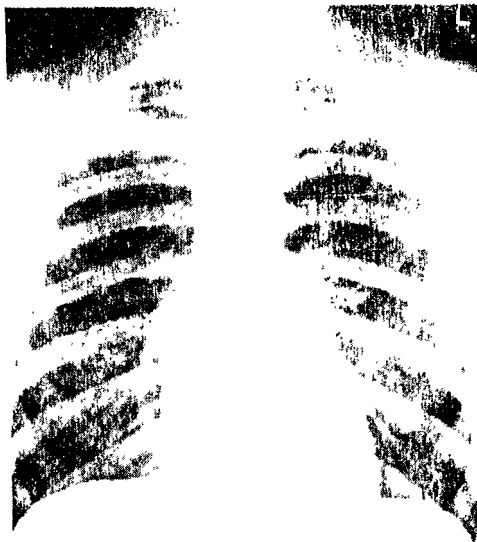


Fig. 13. Siderosilicosis (I.H., aged 36 years). Electric welder (in foundries) for 20 years. Well developed siderosis with nodulation. Patient exposed to some silica dust raised by other workmen.

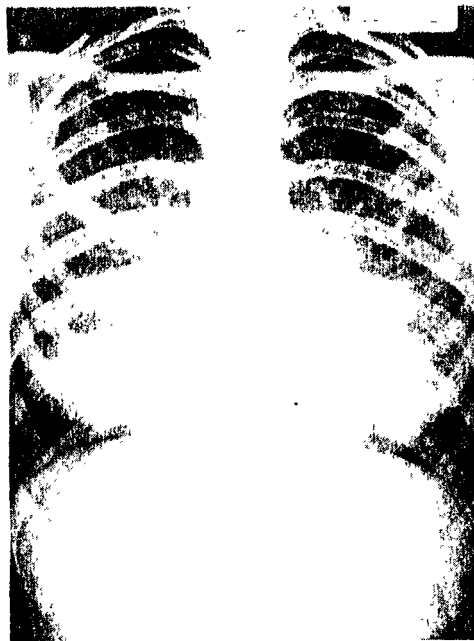


Fig. 15. Idiopathic pulmonary haemosiderosis (M.D., aged 20 years). Acute haemolytic phase.

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Interesting facts about pneumoconiosis statistics are contained in the Digest published by the Ministry of Pore (1960). The statistics are no longer restricted to the mining and quarrying industry but also give details of industrial disease in other industries. The Pneumoconiosis Field Research, recommended by the National Coal Board, has contributed to the elucidation of the relationship between pneumoconiosis and other respiratory condition. The important fact the survey plays in the reactive changes to inhaled particles is still under discussion. A working hypothesis has been forwarded to explain the difference between compensation diseases and the large completely compensable group, as expressed by a low and high exposure rate respectively. The role of mesothelioma disease and the role of tuberculosis infection in the development of progressive fibrosis is still not wholly understood. The part of the miner's life, the dust infection and the miner's work and the development of compensable fibrosis and progressive pneumoconiosis has been discussed.

Dr. A. L. D. Cochrane, Director of the Department of Health, Victoria, Australia, has been elected President of the International Association of Agricultural and Veterinary Hygienists. Dr. Cochrane has been Director of the Department of Health, Victoria, Australia, since 1964. He is a member of the Executive Committee of the World Health Organization and the International Union of Pure and Applied Chemistry. He is also a member of the International Association of Agricultural and Veterinary Hygienists. Dr. Cochrane has been a member of the Executive Committee of the World Health Organization since 1964. He is also a member of the International Union of Pure and Applied Chemistry. He is also a member of the International Association of Agricultural and Veterinary Hygienists. Dr. Cochrane has been a member of the Executive Committee of the World Health Organization since 1964. He is also a member of the International Union of Pure and Applied Chemistry. He is also a member of the International Association of Agricultural and Veterinary Hygienists.

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