

Pneumoconiosis of shale miners

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ABSTRACT Four patients are described in whom pneumoconiosis was diagnosed towards the end of a lifetime's work in shale mines. All developed complicated pneumoconiosis, diagnosed in two cases at necropsy, in one by lobectomy, and in one radiologically. Two of the patients were found at necropsy also to have peripheral squamous lung cancer. The clinical and histological features of the disease resembled the pneumoconioses of coalminers and kaolin workers and the lungs of three of the patients were shown to contain dust composed predominantly of kaolinite, mica, and silica. Shale miners' complicated pneumoconiosis has not previously been described. Although the British shale industry is now defunct, oil production from shale is expanding in other countries, notably the USA. It is suggested that control should be exercised over dust exposure levels in this industry and that epidemiological studies should be carried out to quantify the risks of both pneumoconiosis and bronchial carcinoma.

Among the first commercial sources of mineral oil in the world were the shale deposits of West Lothian in Scotland, where production started in 1851. The industry reached a peak of productivity in the early years of the present century but declined with the increasing availability of oil from other sources, eventually ceasing altogether in 1962.¹ The shale was produced from both open-cast and deep mines, though the latter were always the main source. The industry attracted medical attention because of the risk of skin and scrotal carcinoma in workers exposed to the oil, both in its extraction at the plants in Scotland^{2,3} and in its use, for example by mule-spinners in the Lancashire cotton industry.⁴ Mining the shale was, however, considered to be relatively harmless to health except for the risks of explosion and injury common to all work underground. There has not been thought to be a risk of pneumoconiosis, although a recent report from the USSR has described interstitial fibrosis in some shale workers.⁵

It is unlikely that the remaining British reserves of shale will be exploited commercially. However, the world's largest deposits of shale are to be found in the USA and future energy requirements are likely to require extraction of the oil from the reserves.⁶ Indeed, development of the Colorado Plateau reserves is proceeding to a plan which envisages the production

of 160 000 tons of shale per day by the end of the 1980s.⁷ Since shale consists largely of silicates such as kaolin, which are known to cause pneumoconiosis as in the china clay industry,⁸ it would be surprising if shale extraction were not associated with the same risk. It is therefore of interest to report for the first time a detailed description of pneumoconiosis of shale miners.

Case reports

PATIENT 1

Mr JR worked from 1927, when he left school, until 1959 as a shale miner in a drift mine. As was the practice in this industry he started work at the pit head unloading bogies. He then progressed through pony driving in the main tunnel to filling bogies with a hand shovel at the face. Finally, at the age of 32 years, he was promoted to faceworker, a job that involved drilling into the shale with a hand drill to place shots. Shale was mined by shattering the deposits with these charges, the broken rock then being shovelled into the bogies. No water was used to suppress dust and the mine was usually dry. Dust was generated in drilling and shot-firing and it was usual to wear gauze masks when performing these operations. Electric drills were introduced some time about 1950. The seam height was consistently about 8 ft and ventilation was always regarded as good.

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Development work, cutting from a spent seam to a new one, took place regularly. This was carried out by the same workforce and involved cutting through other, non oil-bearing shale known locally as blaes.

Mr JR left the industry in 1959 and became a general labourer and subsequently a process worker in a non-dusty operation at a petrochemical plant. He retired at the age of 69 years in 1980. He had smoked a pipe until 1979.

In 1961 he had a routine chest radiograph. This showed general diffuse nodularity, category 1/1 on the ILO scale,⁹ and a round 1 cm diameter lesion at the right apex. After appropriate tests and follow-up the lesion was considered to be healed tuberculosis, while the background nodularity was regarded as occupational in aetiology. As the patient was quite well, no further action was taken.

Early in 1980 he was investigated for suspected cholecystitis. In the course of this a chest radiograph revealed an increase in size of the lesion at his right apex which now measured 5 × 2 cm (fig 1). Spirometry at this time was within normal limits. Bronchoscopy and sputum cytology were negative, but as neoplasm could not be excluded it was felt advisable to carry out thoracotomy. Right upper lobectomy was performed by Mr P Walbaum in April 1980 for what

appeared to be a benign lesion. The postoperative course was complicated by acute severe asthma, requiring a period of assisted ventilation and high dose corticosteroids, but eventual recovery was satisfactory. When the patient was last reviewed (August 1980) respiratory function tests showed moderate airways obstruction consistent with his late-onset asthma. At this stage he submitted a claim for disablement benefit to the Department of Health and Social Security and was examined by a Pneumoconiosis Medical Board who agreed with the diagnosis and advised the Local Insurance Officer regarding disablement assessment.

Pathology

Examination of the right upper lobe revealed a firm palpable lesion deep within the apical segment. On sagittal slicing the lesion was heavily pigmented, 4.5 cm in greatest diameter with a central cavity filled with black fluid. Adjacent to this lesion were several small, irregular, pigmented nodules.

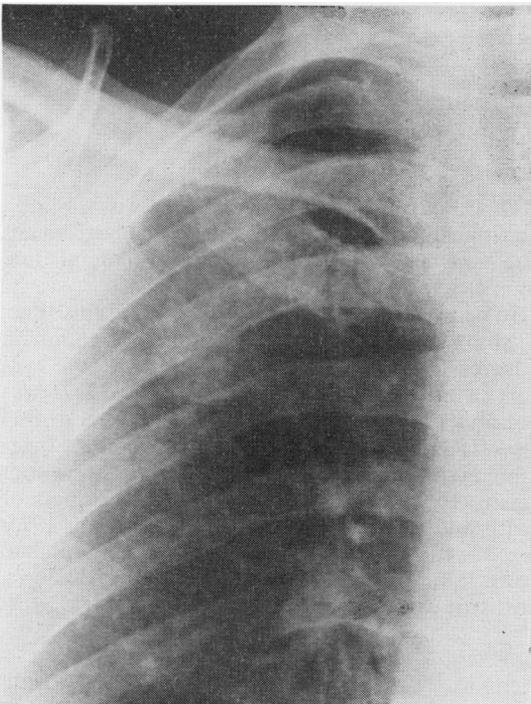


Fig 1 Patient 1. Postero-anterior radiograph of upper right lung showing ovoid lesion.

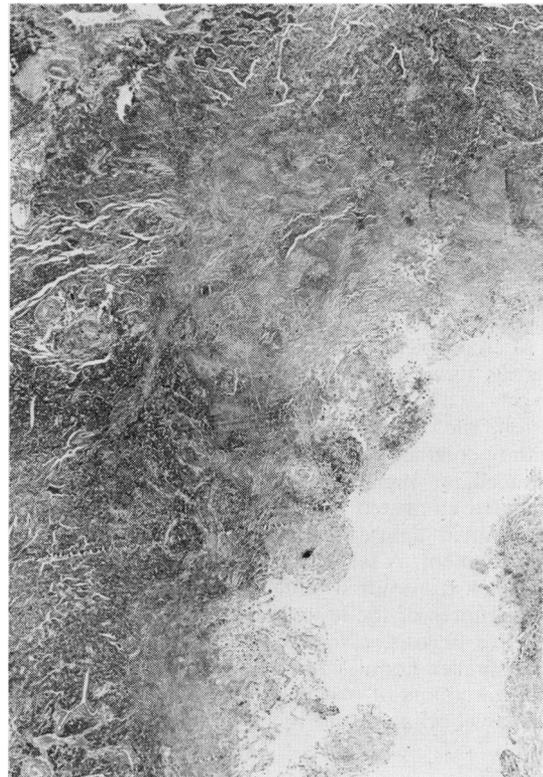


Fig 2 Patient 1. The wall of the cavitating lesion, with the cavity to the right. The wall consists of irregularly distributed bands of collagen with patchy pigmentation. Adjacent to the cavity the tissue is necrotic. H and E, original magnification × 15.

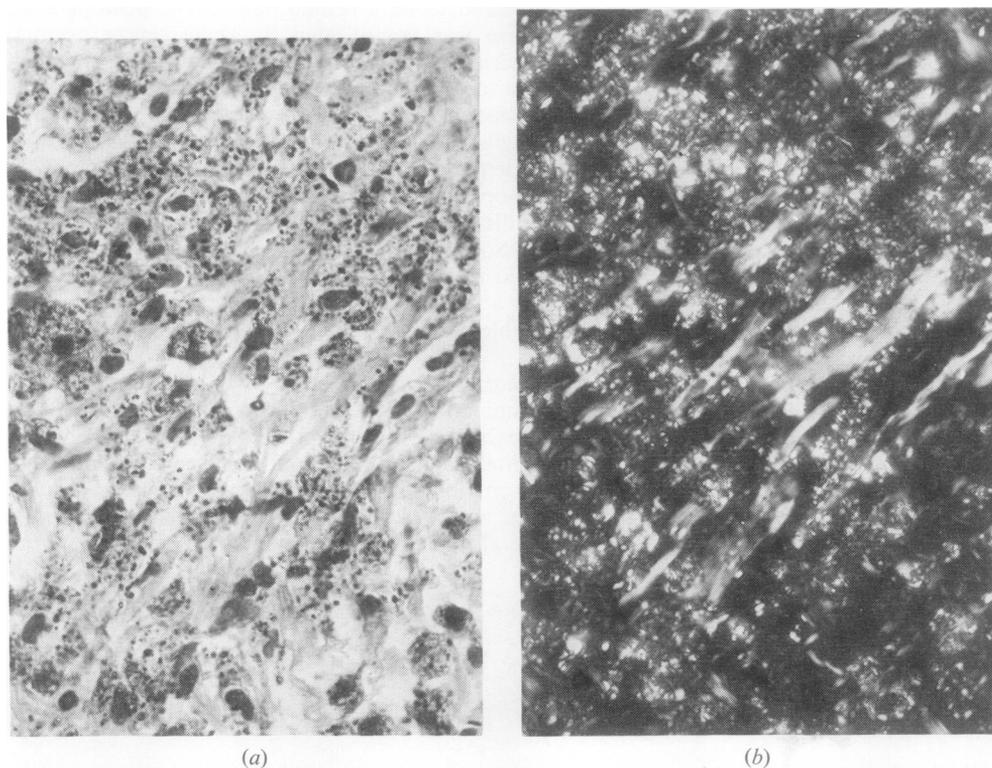


Fig 3 Patient 1. The particulate matter is mostly within macrophages though less than half appears black by transmitted light. Using polarised light the remaining brownish pigment polarises. Two views of the same microscope field by (a) transmitted and (b) polarised light. Original magnification $\times 675$.

Microscopically the large lesion consisted of fibrous tissue with irregularly distributed pigment, largely within macrophages. The margins of the lesion showed a prominent cellular component, mainly pigment-filled macrophages (fig 2). The intracellular pigment was variable in shape, and included elongated particles, brown in colour with transmitted light rather than the black typical of coal pigment. A large proportion of the particulate material transmitted polarised light (fig 3). The appearances of the lesion were those of progressive massive fibrosis, associated with silica or silicates. The smaller nodules resembled coal macules, with small amounts of irregular fibrosis but without the typical whorling of silicotic nodules.

PATIENT 2

Mr WK worked as a shale miner from 1903 until his retiral aged 65 in 1956 apart from a period as a prisoner of war in 1914–18. In 1958, after two years of undue shortness of breath, he applied for disablement benefit and was examined by a Pneumoconiosis

Medical Board who diagnosed that he was suffering from pneumoconiosis and he received disablement benefit. Chest radiograph showed bilateral fine reticulo-nodular shadowing.

He had smoked 10 cigarettes daily until this time, but stopped subsequently. He continued to complain of increasing exertional dyspnoea and by 1963 was short of breath at rest. At this time his FEV¹ was 1.4 l and FVC 2 l (predicted values 3.0 l and 4.6 l respectively) suggesting a restrictive defect. Finger clubbing and crackles on auscultation were not present. He became more disabled by shortness of breath and died of a respiratory infection in 1973, aged 82 years. Before death his chest radiograph had shown the development of larger nodular shadows, consistent with progressive massive fibrosis.

Pathology

There was considerable hypertrophy of the right ventricle of the heart. The lungs were enlarged and heavily pigmented. Palpation revealed bilateral subapical fibrosis in the upper lobes and small

fibrotic nodules in the apices of the lower lobes. The hilar lymph nodes were enlarged and hard. There was mucocoele of the gall bladder.

The fixed lungs showed on sectioning irregular fibrosis and cavitation in the upper lobes and at the right apex a dense black fibrotic lesion 2×3 cm. A similar smaller lesion was present in the left upper lobe. Throughout both lungs were many small, palpable fibrotic nodules.

Histology showed confluent masses of dense collagen, accompanied by accumulation of heavy black dust pigmentation (fig 4). Brown particulate material was not prominent. The dust transmitted polarised light. The centres of the larger masses of fibrosis were patchily necrotic. Fibrosis was surrounded by emphysema. The smaller nodules were centriacinar dust foci with varying amounts of fibrosis, between 2–3 mm diameter. The lymph nodes contained fibrosis, calcification, and dust pigmentation.

A 3.5 cm diameter tumour was found adjacent to an area of fibrosis in the left upper lobe. This was a well-differentiated squamous carcinoma, ulcerating an otherwise normal small bronchus but probably

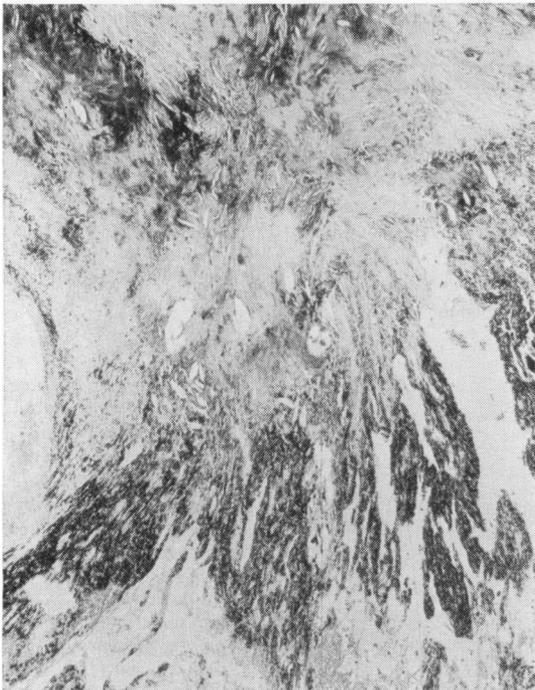


Fig 4 Patient 2. Part of the large lesion from right apex, showing dense collagen admixed with pigment (top) with aggregates of pigment-filled macrophages at the margin (bottom). Haematoxylin and eosin, original magnification × 45.

arising from the scar. The tumour was locally invasive but there was no distant spread.

PATIENT 3

Mr JC had worked as a shale miner for 49 years. He had a long history of cough and sputum production and was in receipt of disablement benefit for pneumoconiosis. In 1973, at the age of 76 years, he was admitted to hospital with jaundice and died two days later.

Pathology

At necropsy he was found to have died from acute suppurative cholecystitis, supervening on chronic cholecystitis with a solitary gall stone. He had moderate right ventricular hypertrophy. The lungs were voluminous and grey-black, and the intrathoracic lymph nodes were jet black on section. The lungs showed generalised centriacinar emphysema, most marked at the apices, and small numbers of palpable fibrotic nodules, the largest being slightly greater than 1 cm diameter. One of those at the right apex showed dense fibrosis with central necrosis. This lesion contained a group of dilated bronchi showing metaplastic and pre-malignant epithelium, in continuity with a poorly differentiated squamous carcinoma. Local invasion by the tumour was very limited and there was no distant spread. The lungs were heavily pigmented with black dust particles which transmitted polarised light.

PATIENT 4

Mr DMcL worked as a shale miner from 1909 until 1956, except for a spell of two years in the army and was not employed after leaving the shale industry. He smoked 20 cigarettes daily and had morning cough productive of sputum. He was examined by a Pneumoconiosis Medical Board in 1958 with a complaint of minimal exertional dyspnoea. Chest radiograph at that time showed simple pneumoconiosis, category 2/1q. A 10% disablement assessment was reached. He was re-examined regularly by the Board between 1961 and 1967, when he and his chest radiograph remained unchanged. In 1963 his FEV₁ was 2.4 l and his vital capacity 3.4 l and in 1965 2.6 l and 3.7 l respectively. In 1970 he complained of aches and stiffness in his shoulders and arms and the radiograph showed the development of multiple larger (3–10 mm) nodules in the upper zones. These appearances remain essentially unchanged at the time of writing, when for a man of 83 years he is reasonably well. Although it was suspected that his complicated pneumoconiosis was of the Caplan type, he has no physical signs of rheumatoid disease and rheumatoid factor is absent from his blood. Disablement assessment was increased to 40% in 1976.

Analysis of lung dust

Small pieces of lung from patients 1, 2, and 3 were analysed for their mineral content. The samples had been previously embedded in wax for histological examination. They were dewaxed with chloroform and ethanol and the tissue removed by hydrolysis with 11.3N hydrochloric acid. The dusts were recovered, dried and the ash contents determined after ashing in a low-temperature oxygen plasma. Qualitative mineral analyses were carried out by X-ray diffraction and the quartz, kaolinite, and mica contents were determined quantitatively by infra-red spectrophotometry.

The results of the infra-red analysis are given in the table. The dusts retained in the lungs of the three miners were similar, containing over two-thirds ash. The ash contained approximately 15 to 20% quartz,

20 to 40% kaolinite, and 15% mica, with about 20% unidentified material. These findings are compared in the table with the mean measurements from a series of lungs from coalminers who had worked in collieries mining low rank (low carbon content) coals, recently investigated at this Institute.

X-ray diffraction confirmed the infra-red analyses, and showed in addition a substantial amount of illite/montmorillonite, a complex clay mineral with a mixed layer sheet silicate structure. This material would contribute both to the mica and the unidentified ash in the infra-red analysis.

For comparison with the lung dusts, four samples of oil shale from different seams in the Lothians have been analysed similarly. These all contained quartz, kaolinite, and mica in similar proportions to those found in the lung dusts. The illite/montmorillonite was also found by X-ray diffraction (fig 5).

Table *Infra-red analysis of lung dust from shale miners*

Patient	Total weight (mg)	% Ash	% Quartz	% Kaolinite	% Mica	% Unidentified
1	138	91.7	17.7	36.3	16.4	21.3
2	3298	69.3	11.2	21.9	15.7	20.5
3	1185	66.5	8.6	19.3	12.6	26.0
Coalminers	—	54.4	10.1	33.4		10.9

Note: the above figures represent the percentage of the dust obtained from the lungs that was ash (non-combustible material) and its principal silicate components, quartz, kaolinite, and mica.

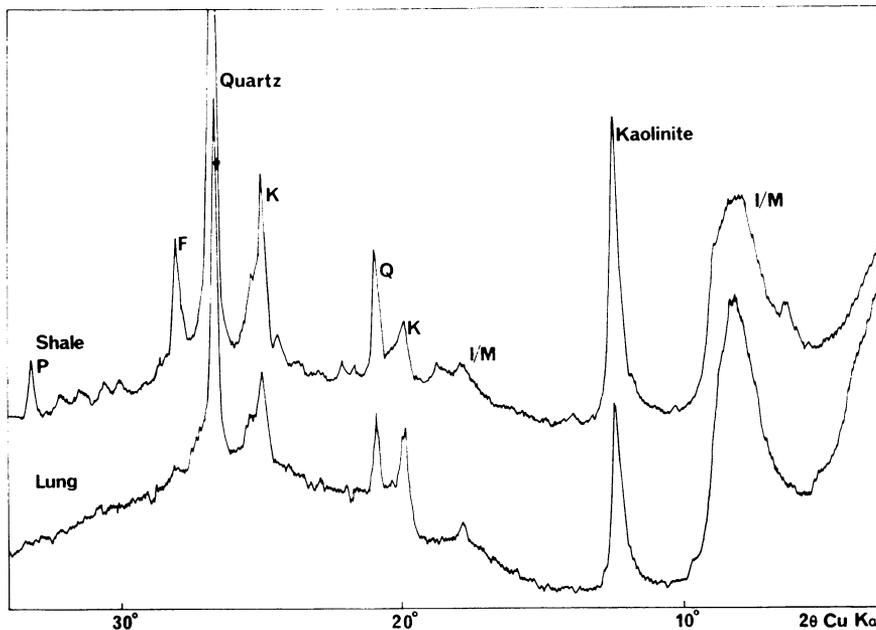


Fig 5 *Portion of trace from X-ray diffraction analysis of dust from lung of patient 1 (lower trace) and from sample of shale from the seam in which he had worked. Note the presence of identical peaks for quartz (Q), kaolinite (K), illite/montmorillonite (I/M), and feldspar (F). Pyrite (P), present in shale, would be metabolised and not remain in lung.*

Discussion

We have described four men who worked exclusively in shale mining and who developed pneumoconiosis towards the end of their working lives. Three had massive fibrosis (complicated pneumoconiosis) proven by histological examination while the fourth developed larger nodules on his radiograph on a background of simple pneumoconiosis after leaving the industry. Two of the men, in addition, had localised peripheral, well-differentiated squamous carcinomas in their lungs at necropsy.

Complicated pneumoconiosis has not been described previously in shale miners in the English language literature. Indeed, the condition was believed not to exist.¹¹ And yet there is no reason to suppose it would not occur. Shale mining inevitably liberated dust, especially in drilling and blasting and, although dust measurements were not generally made, the composition of the dust would be expected to include silicates. Some elemental analysis was carried out in the 1920s, showing shale to contain 70% ash but not defining the mineral composition.¹² The British Mines Inspectorate has records of an analysis of shale mine dust carried out in 1947 which showed less than 3% free silica. This figure is lower than that in the samples of shale that we analysed, which varied between 6 and 12% quartz, the principal minerals present being kaolinite and mica.

It is interesting that the dust we analysed in the lungs of three of our subjects was of similar composition—namely, approximately 70% ash, composed largely of the silicates kaolin and mica with about 10 to 15% free silica. The composition was also similar to that of dust obtained from the lungs of miners who had worked in low rank coal. Thus it would be anticipated on the grounds of dust composition that shale mining would be likely to cause a pneumoconiosis similar to that occurring in kaolin workers⁸ and coalminers. We have shown this to be the case, there being striking similarities in clinical course and pathology between our patients and those described in the Cornish and American china clay industries.^{8 13 14}

In spite of the general belief that shale dust is not toxic, some inhalation studies in animals have been carried out in the USSR and USA.^{15 16} In both countries these studies have shown that shale dust causes a granulomatous reaction in the lungs, and in the USSR a fibrotic reaction has also been reported. In addition there has been a report of diffuse interstitial fibrosis in Estonian shale miners.⁵ There is thus now good evidence that pneumoconiosis occurs in shale miners, and it would seem wise to concentrate further animal studies on investigation of the different toxicities of different types of shale dust and in

particular on the possible risk of carcinogenesis.

It is interesting to speculate why shale miners' pneumoconiosis has not been recognised earlier. This may be because the industry was in decline before much was known about pneumoconiosis generally and also because the disease probably usually presented late in life, often after retiral, perhaps related to the method of promotion in the industry which kept men away from the dustiest jobs in the earlier years of their careers. In addition, it seems unlikely that pneumoconiosis was a widespread problem, there being little local folklore about the harmfulness of the industry to the lungs. The mines were regarded as not very dusty; ventilation was said to be good in order to reduce the risks from accumulation of methane, and support for its efficacy comes from the fact that very few explosions were recorded in shale mines. The low dustiness may also have been related to the soft clay-like consistency of shale, which made it less likely to break up into respirable particles than coal or silica. Moreover, mechanisation was low, most work being done by hand drilling and shovelling. Nevertheless, the disease appears to have been regarded locally in Scotland as a variant of coalworkers' pneumoconiosis. Being a mixed dust fibrosis of the lungs, it falls within the legal definition of pneumoconiosis¹⁷ and those suffering from it have been able to submit claims for benefit for the prescribed disease to the Department of Health and Social Security.

From our patients it is clear that shale mining causes a pneumoconiosis that may progress to massive fibrosis even after the man has left the industry. Histologically the disease is similar to those cases of coalworkers' pneumoconiosis where the coal dust inhaled has had a relatively high ash (silica and silicate) content or to pneumoconiosis in workers exposed to kaolin (hydrated aluminium silicate). There is no longer any possibility of assessing the prevalence of the disease in Scotland though a mortality study of ex-shale workers would be possible. Clearly prospective epidemiological studies of shale miners are highly desirable in other countries, such as the USA, where shale mining is becoming of increasing importance. Moreover, it should now be assumed that exposure to respirable dust in shale workings is hazardous and measurements of dust levels coupled with attempts to reduce those levels where necessary are desirable. In the absence of any epidemiological evidence of dose-response relationships it would be sensible to introduce a dust standard no less stringent than that for coalmine dust, as has been done in the British kaolin industry. This standard is based on long-term epidemiological studies in the British coal industry.¹⁸

One further point should be emphasised. Two of

the patients we describe had well-differentiated, peripheral squamous carcinomas in their lungs. The peripheral site of these lesions and their association with scars suggest that they have been related to the dust or the pulmonary reaction to it more than to smoking. It is not, of course, possible to be sure of this, and the finding may just be a coincidence, as was presumably the occurrence of gall bladder disease in three of the four patients. However, the kerogens contained in shale dust are the precursors of the shale oils that were well-known skin carcinogens. It is conceivable that similar squamous tumours could arise in the lungs of miners and the skins of those extracting and using the oil. Appropriate dust control and surveillance of the workforce should be applied wherever shale is extracted or processed.

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References

- 1 Cameron IB, McAdam AD. *The oil shales of the Lothians, Scotland: present resources and former workings*. London: HMSO, 1978.
- 2 Bell J. Paraffin epithelioma of the scrotum. *Edin Med J* 1876; **22**:35.
- 3 Scott A. On the occupational cancer of the paraffin and oil workers of the Scottish shale oil industry. *Br Med J* 1922; **2**:1108-9.
- 4 Southam AH, Wilson SR. Cancer of the scrotum: the aetiology, clinical features and treatment of the disease. *Br Med J* 1922; **1**:971-3.
- 5 King VA. Morphological investigations of fibrogenic action of Estonian oil shale dust. *Environ Health Perspect* 1979; **30**:153-6.
- 6 Weaver NK, Gibson RL. The US oil shale industry: a health perspective. *Am Ind Hyg Assoc J* 1979; **40**:460-7.
- 7 Costello J. Morbidity and mortality study of shale oil workers in the United States. *Environ Health Perspect* 1979; **30**:205-8.
- 8 Hale LE, Gough J, King EJ, Nagelschmidt G. Pneumoconiosis of kaolin workers. *Br J Ind Med* 1956; **13**:251-9.
- 9 Liddell FDK, Lindars DC. An elaboration of the ILO classification of simple pneumoconiosis. *Br J Ind Med* 1969; **26**:89-100.
- 10 Dodgson J, Whittaker W. The determination of quartz in respirable dust samples by infra-red spectrophotometry. *Ann Occup Hyg* 1973; **16**:389-95.
- 11 Hunter D. *The diseases of occupations*. Sixth edition. London: Hodder and Stoughton, 1978: 1012.
- 12 Department of Scientific and Industrial Research. *The oil shales of the Lothians*. Third edition Edinburgh: HMSO, 1927.
- 13 Lynch KM, McIver FA. Pneumoconiosis from exposure to kaolin dust: kaolinosis. *Am J Pathol* 1954; **30**:1117-22.
- 14 Morgan WKC, Seaton A. *Occupational lung diseases*. Philadelphia: WB Saunders, 1975.
- 15 Bogovski P, Janes H. *Toxicology and carcinogenicity studies of oil shale dusts and shale oils*. Estonia, USSR: Institute of Experimental and Clinical Medicine, 1975.
- 16 Holland LM, Smith DM, Thomas RG. Biological effects of raw and processed oil shale particles in the lungs of laboratory animals. *Environ Health Perspect* 1972; **30**:147-1.
- 17 Social Security Act. Schedule 20. London: HMSO, 1975.
- 18 Jacobsen M, Rae S, Walton WH, Rogan JM. New dust standards for British coalmines. *Nature (London)* 1970; **227**:445-7.