Current Practice

RESPIRATORY TRACT DISEASE

Diagnosis and Management of Industrial Chest Disease

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Forty years ago industrial chest disease was practically synonymous with silicosis, a pulmonary fibrosis caused by the inhalation of fine particles of silica. Better and standardized radiology, extensive studies of necropsy material, the new scientific discipline of objective measurements of respiratory function, the study of host reaction in animals, and—last but not least—modern epidemiological techniques have enormously widened the field of occupational chest disease. Only a few examples can be given. Coal-workers' pneumoconiosis has been found to be a clinical entity with specific histological lung changes.¹⁻³ In a classical publication MacLoughlin *et al.*⁴ described the "mixed dust fibrosis" of foundry workers. Asbestosis was first recorded in this country by Cooke in 1924.⁵ Byssinosis was for many years overshadowed by the high prevalence of non-occupational bronchitis in Lancashire, but has now emerged as a separate entity.^{6,7} Farmer's lung⁸ has only recently been added to the steadily growing list of prescribed diseases. Hard metal disease (tungsten carbide)⁹ and aluminium fibrosis¹⁰ have been described in the last few years. The users of artificial grinding wheels (corundum and carborundum) do not appear to be affected by serious pneumoconiosis, but some workers in the manufacture of these instruments have been exposed to a very serious hazard.¹¹

During the last four decades some harmless industrial materials have been substituted for highly hazardous ones. Sandstone wheels—the "widow makers" of Sheffield—have been replaced by carborundum and corundum tools.¹² Steel castings are now cleaned by steel shot instead of by sand. The gravest risk in the pottery industry—kiln placing in the fine china section—has been eliminated by substituting alumina for flint as a placing medium.¹³

Ingenious methods of dust suppression at the coal face and in workshops have been devised. Their benefits will become apparent only in future years. Some industries such as coal-mining and cotton manufacture are undergoing drastic contraction, which will necessarily alter and possibly diminish the overall size of the present problem.

The "known" prevalence and attack rates of industrial respiratory disease depend to a large extent upon the scope and skill of epidemiological investigations. The need for great caution in the interpretation of the official statistics published by the Ministries of Power and of National Insurance is shown in Fig. 1. The large number of "first diagnosed" cases in pottery operatives between 1953 and 1956 coincided with the first comprehensive radiological surveys in Staffordshire. Eighty-five per cent. of the pottery workers who attended the mass radiography unit during that period had never been x-rayed before. After the backlog of "newly detected" cases had been dealt with by the panels official figures declined rapidly. Three successive comprehensive x-ray surveys of the pottery industry in Stoke-on-Trent suggest a rather low attack

rate of pneumoconiosis.¹⁴ Similarly the certification rates in the South Wales coalfield before 1946 were 40 times those of the other mining areas in Great Britain.¹⁵ The extension of comprehensive surveys to all coalfields, legislative changes, and other extraneous factors have since narrowed the gap considerably. Coal-miners still contribute 84% of all cases of pneumoconiosis (see Table), but in 1963 only 25% originated in the South-western division of the National Coal Board. The recent rise in asbestosis and byssinosis is undoubtedly due to the growing interest of research workers in these conditions, and also to legislative changes.

FIG. 1.—National Insurance (Industrial Injuries) Act, 1946. Pottery industry Great Britain, 1951–1963. Cases of pneumoconiosis diagnosed on first examination.

National Insurance (Industrial Injuries) Act, 1946. Cases of Pneumoconiosis and Byssinosis, Diagnosed on First and Re-examination

Occupation	1958	1959	1960	1961	1962	1963
Coal-mining industry..	2,902	3,523	3,279	2,760	2,171	2,268
Other mining and quarrying ..	89	84	86	89	81	73
Pottery manufacture ..	259	89	50	68	99	76
Refractories ..	46	22	16	28	24	24
Foundry workers ..	232	145	99	103	85	86
Steel dressers ..	44	32	19	32	20	24
Asbestos workers ..	27	37	29	43	52	67
Cotton workers (byssinosis) ..	288	466	418	388	381	354
Others ..	146	109	76	92	77	70

Sources: Ministry of Power (1964); Ministry of Pensions and National Insurance (1958–1963).

Main Types of Industrial Chest Disease

There are four main types of industrial chest disease:

(1) *Mineral dust pneumoconiosis*: the dusts range in their effects from those which are highly fibrogenic, such as silica, to the inert dusts of tin and iron. Pneumoconiosis caused by coal dust and silicates, such as talc, kaolin, diatomaceous earth, and asbestos, are the other most important types in this group.

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(2) *Pulmonary disease caused by organic dust*: the most common forms in this country are byssinosis and farmer's lung.

(3) *Pulmonary disease caused by industrial gases and fumes*: typical examples are manganese pneumonitis,¹⁶ chronic cadmium emphysema,^{17 18} and silo-fillers' disease.¹⁹ In general the population at risk in these occupations is small. On the other hand, the number of workers in heavy and dusty industries who suffer from bronchitis is enormous.

(4) *Occupational lung cancer*.

Diagnosis of Industrial Chest Diseases

The classical triad of diagnostic criteria are a precise occupational history, radiological examination, and symptomatology. To these must now be added objective measurements by respiratory function tests. They allow in many cases an assessment of the type and degree of respiratory disablement and in some instances they are helpful in making a differential diagnosis. In the words of MacKerrow and Gilson, "the pendulum is swinging away from the shadows in the radiograph towards the objective measurements of lung function as the most fruitful means of investigating industrial pulmonary disease."²⁰ Some of these tests can be carried out in a general practitioner's surgery.^{21 22} For a detailed account of the increasing range of these tests a modern textbook should be consulted.²³

Occupational Histories

In 1700 Ramazzini²⁴ asked his pupils to add to the traditional bedside inquiries the question: "What occupation do you follow?" This sound advice is still frequently neglected. Furthermore, often only the patient's last employment is recorded, whereas his respiratory disease is frequently due to an antecedent occupation. Accurate occupational histories taken in chronological order from school-leaving age, together with an inventory of tools and materials used during each employment, will often be rewarding. A "housewife" may have worked as a tilemaker or enamel sprayer before marriage. Building workers and plumbers are liable to inhale asbestos dust. A bricklayer's main job may be the ripping out of highly siliceous refractory bricks from furnace linings. The mystery of typical "classical silicosis" on the x-rays of two lorry drivers was solved when close questioning revealed a short term of employment in a flint mill and the manual loading of finely ground flint.

Radiological Evidence

In most of the *mineral* dust pneumoconioses radiological changes are the most important single piece of evidence. The "International classification of radiological changes caused by mineral dusts"^{24a} is most useful in epidemiological research and of great value in assessing the radiological progress of pneumoconiotic changes. It is an adaptation of the classification of coal-workers' pneumoconiosis by the Pneumoconiosis Research Unit,²⁵ and there are some difficulties in applying the scheme to conditions caused by other dusts. Furthermore, the specificity of some of its categories, such as "increased lung markings" and "linear opacities," is still somewhat controversial. The classification distinguishes between small shadows—"simple pneumoconiosis," and larger opacities—complicated pneumoconiosis or progressive massive fibrosis. Each main division is graded into categories according to the profusion of lesions in simple pneumoconiosis, and to the total size of shadows in progressive massive fibrosis.

In coal-miners the opacities are closely related to the natural history of the conditions, and for simple pneumoconiosis a fairly

good relation has been found between x-ray changes and the dust content of lungs.²⁶⁻²⁸

The classification is purely radiological, and contrary to a widespread misunderstanding by patients—and sometimes by their doctors—its categories are not a measurement of disablement or of a percentage assessment of "loss of faculty." The dramatic "snowstorm" changes caused by highly radio-opaque but inert dusts (tin, iron) are not usually associated with impairment of respiratory function; a minor degree of nodular shadowing in silicosis or minimal radiological changes in asbestosis can be accompanied by considerable disablement.

The clinical and prognostic significance of "simple pneumoconiosis," especially in miners, is still a matter of some contro-

FIG. 2a

FIG. 2b

FIG. 2a.—Coal-miner aged 43 years, 28 years underground. Category 2M, simple pneumoconiosis. Chest x-ray film taken just before this man ceased to work underground. FIG. 2b.—Chest x-ray of the same man after 11 years' work as a gardener and park attendant. Category C progressive massive fibrosis.

versy, but certain facts have emerged from the intensive research of the past 20 years. The lower categories cause little if any respiratory disablement in men below the age of 60 years and simple pneumoconiosis does not shorten life.²⁹⁻³¹ On the other hand, progressive massive fibrosis is frequently a severely disabling disease, and is associated with a high mortality, especially in young people with extensive lesions.³²

From the point of view of prognosis some further findings are important: progressive massive fibrosis rarely attacks men with category 1 simple pneumoconiosis except in pneumoconiosis associated with rheumatoid disease.³³ Once the stage of category 2 or 3 has been reached progressive massive fibrosis may develop whether or not there is a continuation of exposure to dust (Figs. 2a and 2b). The miner with radiological evidence of category 2 or 3 simple pneumoconiosis is therefore clearly a man at risk whether he is disabled or not.

The hypothesis of the tuberculous aetiology of progressive massive fibrosis, which is based on necropsy findings^{2, 34} and animal experiments,³⁵ has not been confirmed by epidemiological studies.³⁶⁻³⁸ The falling incidence of tuberculosis in mining and industrial communities is no automatic guarantee of future lower attack rates of progressive massive fibrosis.

A recent study by Cochrane and Thomas³⁹ also suggests that contrary to previously held belief simple pneumoconiosis of coal-miners can progress in the absence of further exposure to dust.

Radiological Differential Diagnosis

A number of non-industrial systemic and pulmonary conditions produce radiological changes closely resembling those of simple pneumoconiosis.⁴⁰ The most important are sarcoidosis, haemosiderosis, fibrosing alveolitis, carcinomatosis, and miliary tuberculosis. Careful occupational and clinical histories, the course of the disease, laboratory investigations, respiratory function tests, and in some cases biopsy of superficial lymph nodes will establish a diagnosis in many doubtful cases. Lung biopsies should rarely be necessary.

The earliest stages of progressive massive fibrosis cannot usually be distinguished from tuberculosis without repeated bacteriological tests. The differential diagnosis between unilateral progressive massive fibrosis and bronchogenic carcinoma is notoriously difficult, especially when no previous chest films are available. The difficulties and observer errors in diagnosing asbestosis from chest x-rays have been described by Williams and Hugh Jones.⁴¹

Cavities in massive pneumoconiotic lesions are no proof of tuberculosis infection: they are often due to ischaemic-necrotic changes, sometimes preceded by melanoptysis, or to Caplan's syndrome.^{33, 42}

Symptomatology

Undue dyspnoea is the most common symptom of most types of industrial chest disease. Others are cough, pain and tightness in the chest, and lassitude. In an industrialized and cigarette-smoking society these are non-specific symptoms, but some conditions show a more distinctive pattern. In asbestosis dyspnoea is often out of all proportion to the physical signs and the x-ray abnormalities. In the byssinosis of cotton and flax workers, which may be caused by the action of plant debris on smooth muscles,⁴³ workers usually complain of tightness of chest, dyspnoea, and malaise in the afternoon of Mondays or on the day they return to work after holidays. Complete or partial recovery may follow, until after long exposure permanent impairment of lung function supervenes. Massoud and Taylor⁴⁴ found a higher level of antibody against an antigen in the cotton plant in the sera of workers with byssinosis than in the general population.

In farmer's lung symptoms simulating those of an acute influenzal attack or of pneumonia follow the inhalation of mouldy and dusty hay. Severe and sometimes permanent dyspnoea may follow, particularly after repeated exposure. The initial granulomatous pneumonitis may lead to permanent interstitial fibrosis. Sufferers seem to be hypersensitive to certain thermophilic actinomycetes, and serum precipitins against mouldy hay have been demonstrated.^{45, 46}

In most pneumoconioses clinical signs are not specific or distinguishable from those of chronic airway obstruction and space-occupying lesions. Finger clubbing is rare in most mineral pneumoconioses but is sometimes seen in asbestosis.

Respiratory Function Tests

The dust deposits and slight fibrosis around the small bronchioles and the focal emphysema² in simple coal-miners' pneumoconiosis usually cause little change in pulmonary function. Many miners, however, suffer from chronic bronchitis, and function tests on them show the pattern of non-specific obstructive airway disease.

In progressive massive fibrosis the findings correspond to space-occupying lesions, distortion of airways, and irreversible airway obstruction; there may also be a reduction in the diffusing capacity of the lungs.

In silicosis and most of the mixed dust pneumoconioses the amount of fibrosis is more extensive than in coal-workers' pneumoconiosis. Lung-function tests reveal the pattern of parenchymatous pulmonary disease, frequently accompanied by signs of airway obstruction.

In byssinosis ventilatory capacity is often reduced during exposure to dust. Asbestosis causes reduced respiratory capacity, lung stiffness, and a lowered diffusing capacity. The characteristic findings in farmer's lung are a low diffusing capacity with high exercise ventilation.²³

Occupational Lung Cancer

In this country a significant aetiological relationship has been established between lung cancer and occupation for workers engaged in certain nickel-refining processes, chromate workers, retort house personnel in the gas industry, and persons exposed to asbestos dust.⁴⁷⁻⁴⁹ The main interest is now centred on the association between asbestos and malignancy. Inhalation of asbestos seems to be related not only to a high incidence of bronchogenic carcinoma but also to pleural and peritoneal malignant tumours.^{50, 51} These rare tumours have also been found in residents of asbestos-mining districts not directly exposed to occupational risks.

Contrary to a widespread belief there is no evidence of a causal relationship between coal-miners' pneumoconiosis or silicosis and lung cancer. Indeed, the attack rate in South Wales coal-miners seems to be lower than in non-mining men.^{52, 53}

Medical Treatment

No therapeutic measures are usually needed in cases of simple pneumoconiosis. The value of aluminium inhalations has not been convincingly proved.⁵⁴

The treatment of chronic airway obstruction, the most common complication of pneumoconiosis and of chronic bronchitis generally, will be discussed elsewhere in this series.

The results of standard anti-tuberculous therapy in cases of sputum-negative progressive massive fibrosis are not convincing; on the other hand, many pneumoconiotics with positive sputum for *Mycobacterium tuberculosis* undoubtedly benefit from energetic and prolonged anti-tuberculosis drug treat-

ment.⁵⁵ Generally, however, the presence of pneumoconiosis has an adverse effect on the response of tuberculosis to standard drugs, and more relapses occur than in uncomplicated tuberculosis.⁵⁶ Whenever possible treatment should be continued for at least two years. A *restitutio ad integrum*, which drug treatment achieves in many cases of uncomplicated tuberculosis, cannot be expected in cases of progressive massive fibrosis with Koch's infection. The patients will be left with space-occupying, massive, and in many instances growing lesions. The occurrence of so-called "anonymous mycobacteria" in coal-miners^{57 58} may cause difficulties, as many of these bacteria are resistant to a number of first-line and second-line anti-tuberculous drugs.

Steroid treatment has been found effective in cases of early asbestosis and farmer's lung.

General Management

"... The board says that I have bronchitis and cardiac hypertension. Dr. X in the hospital says silicosis, Dr. Y tuberculosis, Dr. Z thinks I have silicosis, you say bronchitis. I have worked as a pottery caster for 40 years, and after 14 years of ill-health, the Ministry tells me that doctors do not make mistakes."

(From a recent letter to the writer.)

Such outcries are not uncommon. In 1963 77% of coal-miners and 53% of workers in other scheduled occupations who applied for disability benefits under the Industrial Injuries Act were "not diagnosed" as the result of a scrutiny of their chest radiographs. Of those who were clinically examined by the panels 60% were not awarded a disability pension.⁵⁹ Many of them will have applied on the advice of their own doctors, of chest physicians, or as the result of a routine mass x-ray examination.

The often-heard question, "Why is my younger workmate who has breathed less dust than I have done certified while I am not?" should not be brushed off with a remark about inconsistent procedures by the pneumoconiosis panels. The question poignantly points to our present ignorance of the biological factors which determine the widely varying individual susceptibility to the hazards of industrial environments. It is, however, important to know that the panels rarely diagnose pneumoconiosis unless category 2 simple pneumoconiosis has been reached.⁶⁰ Generally it is inadvisable to encourage workers whose radiographs show less than that category to submit an application for disability benefit. They should be kept under regular supervision at chest clinics or attend for routine chest x-raying at the many centres and field units which are now available.

Chronic bronchitis in miners and in other workers in dusty industries causes at present a great amount of resentment and frustration. Official figures show a high mortality and morbidity from bronchitis in miners and foundry workers, but their wives seem to be similarly affected. Community surveys reveal a higher prevalence of bronchitis in urban than in rural areas and a significant association with the smoking of cigarettes. However, middle-aged and elderly miners have a lower maximum breathing capacity than non-miners of the same age. The difference cannot be explained by smoking habits. The age-specific degree of breathlessness in miners and ex-miners shows little correlation with the degree of simple pneumoconiosis. Miners without x-ray evidence of pneumoconiosis are often more breathless than those with category 1 or 2 simple pneumoconiosis.^{29 61-65}

These statistical facts have been well summarized by Rogan and his colleagues⁶⁶ in these words: "When the prevalence of bronchitis in a mining population exceeds that of the non-mining population in the same area, only the excess can be regarded as occupational." This implies of course that in the individual case the respective contribution of occupational,

general environmental, and possibly genetic factors cannot at present be accurately assessed. Whilst it is undoubtedly true that "the chief need is prolonged and patient explanation,"⁶⁷ the psychological skill of a medical adviser will be severely tested in explaining statistical facts to anxious or resentful patients, who are often confused by conflicting medical opinions. It is important to point out that the term "pneumoconiosis" covers a very wide spectrum of abnormalities, some of which are purely radiological. Fortunately there is now no lack of examples of workers who live to a ripe age despite their large shadows on the chest radiograph. Their film should be shown to the anxious man with "simple pneumoconiosis" and compared with his own.

There is, however, a growing number of experts who consider the legal distinction between pensionable industrial and non-pensionable chronic chest disease as unrealistic, and suggest benefits for all those seriously disabled by respiratory insufficiency from whatever cause as a more logical approach.⁶⁸ In highly industrialized and cigarette-smoking societies such as ours the financial consequences of such legislative changes must not be underestimated.

In the meantime the medical practitioner faces the difficult task of dispersing the shadows in the mind, caused by shadows on the radiograph, of dissuading his patients from smoking, and last but not least of advising them about future employment.

Entire removal from dust and fumes, though desirable, is rarely practicable for social reasons. Furthermore, in most of the mineral-dust pneumoconioses no guarantee can be given that the progress of the condition will be halted once irreversible changes have been established. However, young persons who show signs and symptoms of industrial chest disease after only short exposures will be well advised to look for alternative employment. Middle-aged and skilled craftsmen will usually decide to stay in their job, whatever advice they get, and many will be found work in "approved conditions." Even severely disabled persons can often perform a reasonably satisfying job if given the opportunity. They can ask to be placed on the disabled persons' register. Often they will find the problem of travelling to and from work more difficult than doing it.

It needs no emphasis that the answer to the vast medical and social problem of chronic chest disease caused or aggravated by industrial conditions is prevention, an aspect which is not within the terms of reference of this contribution. The record of this country with regard to the basic principles of protection against noxious industrial dusts and fumes is impressive, and the epidemiological approach to the testing of these measures is mostly based on the pioneer work of British scientists. However, the thought that some men will soon breathe safely on the moon, whilst their earthbound fellows still become breathless in mines and factories, casts some doubts on the soundness of our sense of priorities.

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ANY QUESTIONS?

We publish below a selection of questions and answers of general interest.

Barrier Creams and Occupational Dermatitis

Q.—Have barrier creams a useful place in preventive dermatology?

A.—Users of a barrier cream, chiefly for oily or dirty work, often find that the skin is more easily cleansed if the cream has been applied before work is begun. That, however, is not what the question implies. The more essential object of a barrier cream is to reduce the incidence or severity of occupational dermatitis, and to this crucial question there is not enough evidence to give a firm answer.

The various barrier creams commercially available differ from each other, and the nature and extent of the risk of dermatitis varies from one occupation to another according to the substances handled and the degree to which the skin is exposed to them. Thus it is understandable that a particular cream might be helpful in one type of work and useless in any other. For example, Dr. Newhouse's observations¹ in an automobile factory suggested that the barrier cream used at that time was giving some protection against oil but not against chromate. Unfortunately the solvents and alkalis which most often damage the skin are likely to be the very substances which most easily remove a layer of barrier cream.

Barrier creams are sometimes themselves in danger of being potential irritants or sensitizers, and in this case may, of course, be actively harmful. Some emollient substances—e.g., lanolin—promote the absorption of foreign materials into the skin, and unless a barrier cream is formulated with this consideration in mind, and used only in connexion with suitable occupations, it can be appreciated that the risk of dermatitis could actually be increased by its use.

Tests of the efficacy of barrier creams carried out under controlled conditions have sometimes been encouraging, but other investigators have reached opposite conclusions. Whatever the result, these tests are artificial and do not answer the question whether barrier creams prevent occupational dermatitis. Controlled experiments on the factory floor are very difficult to arrange. A recent experiment of this kind, designed by a group of industrial medical officers and dermatologists, proved impossible to carry out because the insurance companies concerned were unwilling to accept the responsibility for cases of industrial dermatitis which developed under conditions which, it must be admitted, were experimental. Conclusions cannot be drawn from retrospective studies, because they seem always to be vitiated by the fact that too many variables are involved, of which a change in barrier cream is only one.²

At most it can be said that the protection afforded by barrier creams is only partial and in some occupations very slight or nil. In some cases positive harm may result. Their importance has been exaggerated by the advertising of certain manufacturers, and accepted too readily in some quarters. Probably most industrial medical officers and dermatologists to-day would still prefer to reserve judgment until better evidence is forthcoming.

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- ¹ Newhouse, M. L., *Studies of the Epidemiology of Non-infective Skin Diseases in an Automobile Factory*, 1963, M.D. thesis, University of London.
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Pregnancy in Older Age

Q.—A 43-year-old woman had two normal pregnancies and confinements at the ages of 24 and 26. She now wishes to remarry. How great are the risks of obstetric complications and foetal damage or abnormality?

A.—There is no need to be unduly pessimistic about the outcome of a third pregnancy in this patient. The greatest risk to her and her child is probably from placental insufficiency associated with maternal hypertension. She should be seen at regular and frequent intervals throughout her pregnancy, and she should be confined in a fully equipped and staffed maternity unit. There need be little anxiety about her labour and delivery provided that she and the foetus are observed closely.