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P29

EXHIBIT P-327

ASBESTOS EXPOSURE AND SMALL AIRWAYS DISEASE

GÖTEBORG — A high incidence of small airways obstruction is found in subjects exposed to asbestos; the obstructive pattern is best detected by flow volume curves (and can occur in patients with normal spirometry); flow volume curves might be used as a routine clinical test for early detection of airways obstruction in asbestos exposure.

These findings were reached by Drs. R. Rodriguez-Roisin, G.M. Cochrane,



Dr. Rodriguez-Roisin

Report from the SEPCR WORKING GROUP, Göteborg, Sweden.

and T.J.H. Clark of the Brompton Hospital, London, England, and reported to a meeting organized by the SEPCR (Societas Europaea Physiologiae Clinicae Respiratoire) Working Group. The symposium, "Epidemiological Follow-Up Investigations of Lung Function", held recently in Sweden, was addressed by Dr. Rodriguez-Roisin.

"Classically, the main functional effects of asbestos in the lung are a restrictive pattern and a low gas transfer," he said, "but airways obstruction has been reported to occur and it has been suggested that this is caused by asbestos deposition in and around small airways."

Random Sample

In the Brompton study, the incidence of airways obstruction was investigated by routine spirometry, static lung volumes and flow volume curves. These measurements were made in a random sample of 40 subjects who had been referred to the hospital having been exposed to asbestos dust for varying periods of time. Referral was based on respiratory symptoms and signs or radiological abnormalities.

Routine lung function tests permitted the classification of these patients into four groups: Normal ventilatory pattern (37 per cent of total subjects); restrictive defect (30 per cent); mixed defect (17 per cent); obstructive profile (15 per cent).

Flow Volume Curve: Test of Developing Asbestosis

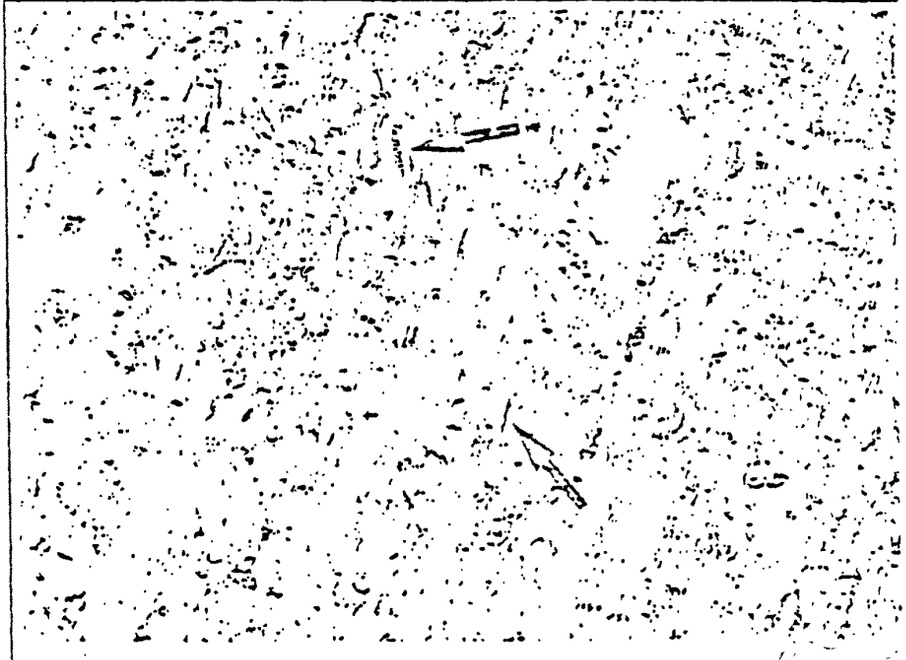
Epidemiological data (age, years of exposure to asbestos and smoking habits), clinical features (chronic cough, sputum, finger clubbing and basal rales) and radiological findings (according to UICC/ Cincinnati Classification, 1970) were analyzed in each group. No statistical differences were found between the groups, nor any correlation with the functional changes.

Flow volume curves were studied and V max at 50 per cent, expressed as TLC per second, was reduced in eight out of the 15 normal subjects and in half of the 12 restrictive ones. Adding these 14 subjects to the other obstructive groups (six pure obstructive plus seven mixed) gave an airways obstruction total of 67 per cent.

V max at 75 per cent, expressed as TLC per second, was reduced in the remaining seven normal subjects. "Using flow volume curves as a test, therefore," the speaker continued, "85 per cent — 34 subjects — presented evidence of small airways obstruction. MMFR was also reduced in 72 per cent of the total series. "If we exclude the 13 cases already detected by routine methods, 21 cases had small airways disease," the researcher pointed out.

Smoking Habits

While no statistical correlation between functional changes and epidemiological, clinical and radiological findings was obtained, a look at the smoking habits of the 34 subjects with reduced flow volume curves showed that 19 were smokers, eight had been non-smokers for less than 10 years and one for more than 10 years. Only six had never smoked. Classifying the one long-term ex-smoker with the non-smokers, the speaker noted that seven patients — 17 per cent of the total — had small airways obstruction and no smoking habits. These seven non-smokers with reduced expiratory flow rates had in common a long exposure to asbestos dust and few X-ray changes.



PULMONARY FIBROSIS in a patient with heavy exposure to asbestos dust. Within collagen tissue, many fibrils, probably of asbestos origin, can be observed (arrows).

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Of Chemicals to Man: asbestos*

The excerpts we present below continue a series highlighting published material which we believe will be of special interest to RNB readers.

A Working Group of the International Agency for Research on Cancer met in Lyon, France, in 1972 under the chairmanship of Dr. J.C. Gilson, Medical Research Council, Pneumoconiosis Unit, Llandough Hospital, Penarth, Glamorgan, U.K., to prepare a monograph on the carcinogenic risk of asbestos to man. Their research was part of a programme initiated by the IARC to provide government authorities, international agencies, industries and scientists with "expert, independent, scientific opinion on environmental carcinogenisis".

To a background of pertinent references regarding physio-chemical characteristics, use and occurrence of asbestos, as well as pharmacological, toxicological and epidemiological data, was added a review of published articles and papers; every effort was made to cover the whole literature. The nine members of the Working Group, who served in their individual capacities as scientists, provided a critical view and evaluation of the data reported.

Extracts from the monograph follow:

Observations in Man

In 1935, 50 years after the start of the use of asbestos in industry, suspicion of an association between asbestosis and lung cancer was reported by Lynch & Smith (1935) in the U.S.A. and by Gloyne (1935) in the U.K. About ten years later, case reports of pleural and peritoneal tumours associated with asbestos appeared (Wedler, 1943, a, b; Wyers, 1946). Epidemiological proof came from Doll (1955) and Knox *et al* (1968), who showed a ten-fold excess risk of lung cancers in those U.K. asbestos textile workers who had been employed before 1930, when new regulations produced improved dust conditions in factories. Similar findings, but including the development of mesotheliomas, were reported in the U.S.A. in 1961 and published later (Manuceso & Coulter, 1963). Possible variations in risk with different types of fibre were rarely considered in the early reports.

Exposure to Single Types of Fibres

In 1956 Wagner started investigating the occurrence of pleural and peritoneal mesotheliomas in the crocidolite mining areas of the North-West Cape Province in South Africa. It was shown that these tumours occurred among men working in the mines, mills, transportation and handling of the fibre, as well as in the non-mining population living in the vicinity (Wagner *et al.*, 1960). Asbestosis was not invariably present. The latent period between the first exposure and tumour development was long — a mean of 40 years. Searches in the other areas in South Africa where amosite and chrysotile had been mined for many years did not reveal these tumours (Sluis-Cremer, 1965). Subsequent surveillance of the mining population in all the asbestos-producing areas in South Africa has added support for a major difference in incidence of the mesotheliomas with the different kinds of asbestos (Harington *et al.*, 1971; Webster, 1973).

Exposure to Mixed Fibre

Pure exposures to a single type is rare, as in most industrialised countries different types of fibres are mixed during processing. Prospective mortality surveys of defined populations of asbestos textile and shipyard workers have provided the most concrete evidence concerning the association between bronchial cancer, pleural and peritoneal mesotheliomas and past exposure to asbestos. It has also been suggested that other factors such as iron oxide may be contributative (IARC, 1972).

The proportion of the highly exposed groups who eventually dies of asbestos-related cancers is still uncertain on account of the very long latent period, the difficulty of establishing population groups exposed earlier than about 1940, and the confounding effects of cigarette smoking. For mesotheliomas — a tumour which occurs rarely except following inhalation of asbestos — the proportion may be about five per cent in the higher risk groups.

Newhouse (1969) and Newhouse *et al.*, (1972) have shown that the cancer risk following mixed exposure to chrysotile, amosite and crocidolite is dose-related in men and women. Those with low or moderate exposure (as judged by their occupations) showed no excess lung cancer risk, whereas after more than 15 years' follow-up, those with heavy exposures had a six-fold excess among the men and twelve-fold among the women. The occurrence of mesotheliomas was also dose related.

In a naval dockyard population, Harries (1968) showed that while there was no excess cancers associated with asbestos exposure between 1959 and 1968, there has since that time been a steep rise in mesotheliomas. Extensive so that any effects on cancer incidence would be expected to be detectable in the 1970's and onwards.

Non-Occupational Exposures

Asbestos fibres and 'bodies' are present in the lungs of most adults who have lived in urban areas (Thomson *et al.*, 1963, Gross *et al.*, 1969; Davis & Gross, 1973; Um, 1971, Oldham, 1973). The total number of fibres in the lungs may be large, but the mass is small. There is no evidence at present that this lung burden is a cause of excess morbidity or mortality in the general population.

Mesotheliomas have occurred after short exposure to asbestos and in those exposed at home to dusty clothing or to a neighbouring source of asbestos air-pollution (Newhouse & Thomson, 1965; Bohling & Hain, 1973). However, studies of the geographical distribution of cases of mesothelioma in the U.K. over a 10-year period indicate that the new cases are nearly all from areas where there had been a recognised occupational exposure to asbestos in the past (Gilson; 1970).

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FIGURE III

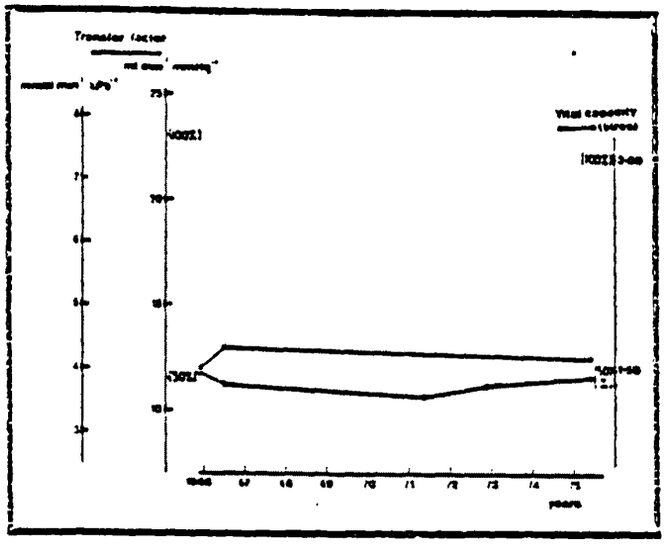
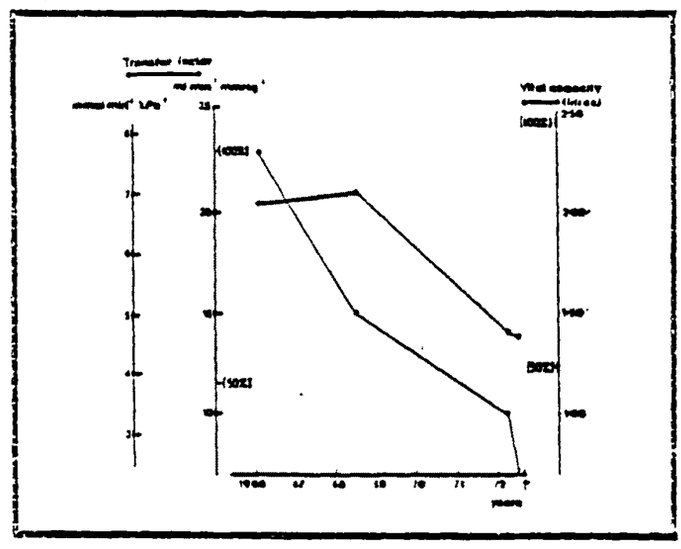


FIGURE IV



Both Vital Capacity and Transfer Factor were already impaired to 50 per cent of the predicted values, and have remained constant in absolute values for the past nine years.

"The changes seen in these three patients may represent the disease process at different stages of its development," Dr. Britton postulated. "In the last patient, the disease process may have burnt itself out." Death may occur at any time along this progression and is rarely caused by asbestosis alone, the study indicates. Death is usually due to carcinoma of the lung (of the 17 patients with serial tests, seven have died, six from a carcinoma) or possibly due to a superadded dust infection.

Figure IV charts the serial studies of a patient who died in 1972, six months after the diagnosis of cancer was made. Her only exposure to asbestos had been for three months in 1942, in making gas mask filters. In 1966 her lung function tests were within the normal range, but with abnormal basal shadowing on her chest X-ray. The initial fall in Transfer Factor came in 1968, while her Vital Capacity was still maintained. A gradual decline in both measurements followed over the next four years.

The emergence of a carcinoma in this patient, and in the others, did not appear to make any significant difference in the lung function tests, according to the researchers, although in the terminal stages of the illness there may be a more rapid decline in either or both measurements.

"While the serial lung function tests were not helpful in assessing prognosis in this limited group of patients with asbestosis," Dr. Britton concluded, "there is obviously a need for further longitudinal studies with a more frequent measurement and

Smoking Habit Magnifies Cancer Risk in "Lightly Exposed" Asbestos Workers

The important enhancement of lung cancer risk when cigarette smoking is coupled with asbestos exposure was the principal point of an address given by Irving J. Selikoff, M.D., of New York's Mount Sinai Hospital, to the American Medical Association eight years ago. The dramatic facts profiled by the study* of 307 asbestos insulation workers remain unrefuted: asbestos workers as a group face a high risk of dying of bronchogenic carcinoma (about seven or eight times the expected risk); asbestos workers who smoke have about 92 times the risk of men who are neither exposed to asbestos or are cigarette smokers.

Among Dr. Selikoff's comments at that time, however, are some which have special pertinance today when "heavy exposure" has been seen for the hazard it is and steps taken to avoid it. "The increased risk of neoplasia (mainly bronchogenic and mesothelioma) among insulation workers reported here," he said, "should be evaluated in light of the knowledge that these men have comparatively light exposure as asbestos trades go. Primarily employed in construction work, many of the materials they use contain little or no asbestos and others have only five to 15 per cent ... these men often work

Heavy exposure is unlikely to be the most important problem in the future, he said, rather "light exposure" similar to that of the insulation workers of the study will be much more common, both in the direct asbestos working trades and the indirect occupational exposures such as construction and ship-building industries.

And Dr. Selikoff sounded an early warning against another type of "light exposure" which, he remarked, might eventually affect many more people than those industrially exposed. "In the past several years," he noted, "it has been demonstrated that asbestos bodies can be found in the lungs of 25 to 50 per cent. of adults examined at autopsy in large cities such as Belfast, Cape Town, Miami and Montreal." He cited "asbestos air pollution" by fibres derived from industrial spillover (e.g. dust from construction sites or factory wastes) or from end-product use, as well as living within half a mile of an asbestos plant or in the household of an asbestos worker as environmental contaminations which can be associated with some risk of mesothelioma. "What is not now known," he pointed out, "is whether the minimal amount inhaled by the general public carry a similar risk."

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FIGURE I

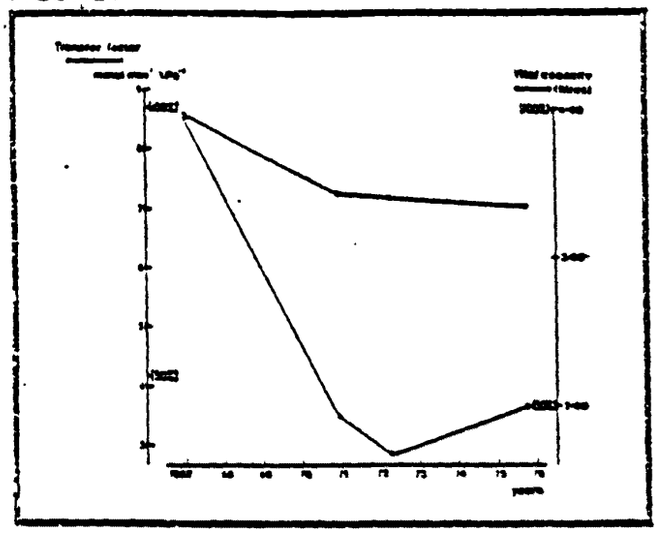
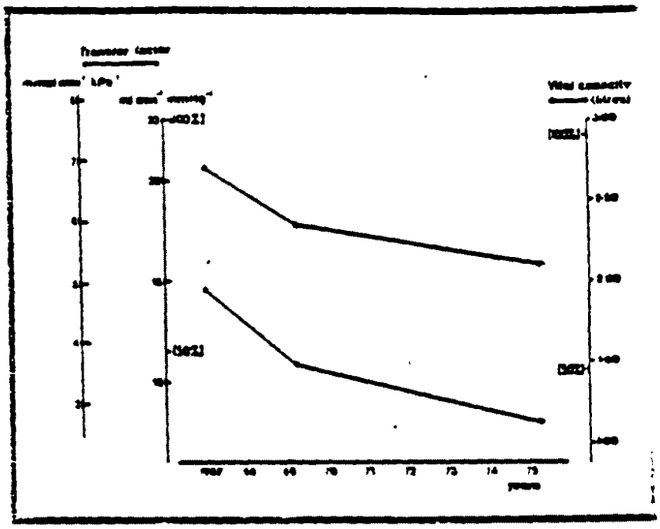


FIGURE II



LONDON — A review of serial lung function tests carried out in a group of asbestosis patients shows that, after a varying interval of time following exposure, there is a marked fall in the Transfer Factor which may precede any significant fall in the Vital Capacity.

M. G. Britton, M.B. and D. T. D. Hughes, M.A., reached this conclusion as the result of a retrospective study of the 119 patients who had had lung function tests for possible asbestosis at The London Hospital, England, between 1960 and 1975. Dr. Britton presented their findings to a meeting of the Thoracic Society.

While the principal features of pulmonary function abnormality in asbestosis are a lowered diffusing capacity, reduced Vital Capacity mainly due to reduction in the inspiratory component, and hyperventilation on exertion (often with arterial oxygen desaturation) the sequence of events in the development of asbestosis is far from clear, Dr. Britton said. He cited a 1965 report by Bader *et al* which presented follow-up measurements on 13 asbestos workers using Vital Capacity, maximum breathing capacity and blood gas measurements, in which Vital Capacity was concluded to be the most sensitive index of progression of the disease, correlating well with X-ray changes and progression of dyspnoea in half the cases.

"We have been unable to find in the literature any study including serial measurements of Transfer Factor," Dr. Britton said, "and this prompted us to do the retrospective study."

Of the 119 patients, 48 had been diagnosed as having asbestosis according to Parke's criteria. Of the 48, 17 had had [illegible] from these 17 patients was studied to determine whether serial pulmonary function tests, especially changes in Vital Capacity and Transfer Factor, might be helpful in predicting the prognosis in such cases.

The period from first exposure to asbestos and referral to the laboratory varied between 17 and 56 years, with a mean of 32 years. "All were withdrawn from further exposure before or soon after their first tests," Dr. Britton remarked, "and thus represent the natural history of asbestosis rather than any changes from further exposure."

According to results of their initial tests, the 17 patients could be divided into four groups:

1. Three patients having normal values for both Vital Capacity and Transfer Factor (assuming normal to be greater than 75 per cent of the predicted values);
2. Eight patients with a normal Vital Capacity but an impaired Transfer Factor;
3. One patient with an abnormal Vital Capacity but a normal Transfer Factor;
4. Five patients with impaired values for both.

No correlation was found, considering all 17 cases, between the functional changes seen initially and the date and duration of exposure.

Dr. Britton described to the meeting findings for representative patients from each of the three main groups (1, 2, 4):

Figure I shows the results of the serial lung function tests in absolute values against time in years for a male patient from Group 1. Now 65, he was a lagger working with asbestos between the years 1927-19[illegible], who presented in 39 years after his initial exposure. [The values of the Transfer Factor are on the left of the graph (closed circles) and those of the Vital Capacity on the right (open circles). The figures in brackets refer to the percentage of predicted values at the time of the initial tests, all percentages referred to having been corrected for aging.]

On first testing, both Vital Capacity and Transfer Factor were approximately 95 per cent of the predicted value; within four years there was a marked fall in the Transfer Factor to 41 per cent of predicted values, accompanied by a smaller and less significant decline in the Vital Capacity to 85 per cent of predicted values.

Figure II presents the serial results of a female patient, now aged 63, from Group 2. She worked as an asbestos weaver from 1931 to 1935 and again for ten months in 1939. In 1967 she presented with a history of increasing shortness of breath, and grossly clubbed fingers. At this time, her Vital Capacity was 92 per cent of predicted value, but her Transfer Factor, at 63 per cent, was already impaired. Over the next eight years there was a decline in both measurements of a much greater order than would be expected from the effects of aging alone: to 77 per cent of predicted value for Vital Capacity and to 39 per cent of predicted value for Transfer Factor.

Figure III represents the findings for one of the five patients in Group 4. A female factory worker, now aged 54, she had worked at an asbestos factory from 1946 to 1961 making asbestos mattresses. Eighteen years after her first exposure she was diagnosed as

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by Dr. A.N. Walker, M.D., Royal Northern Hospital, London, England

Pulmonary damage caused by dusts, fumes or noxious substances and inhaled by workers in specific occupations is known as occupational lung disease.

In the 10-12,000 litres of air inhaled by the average human subject each day, there is suspended particulate matter. The majority is exhaled. A quantity, nevertheless, is deposited and the nature of these particles varies considerably: some are chemically inert but still capable of damage; others act as allergens; some cause fibrosis and yet others, like asbestos are potential carcinogens.

Investigation of Occupational Lung Disease

Diagnosis and assessment of occupational lung disease depends on four factors:

1. The physician's awareness of patient's occupation and nature of risk.
2. Chest radiography.
3. Pulmonary function studies:
   (a) spirometry
   (b) diffusing capacity.
4. Distinctive signs and symptoms.

As compensation is available for patients with some occupational diseases, because there may be a need to recommend changing jobs, and because industry's attitudes are now directed to protect workers, the physician is neglectful if a disease is not documented thoroughly.

The chest radiograph is of great importance as are pulmonary function studies as stressed by the International Labour Organisation Report of Meeting of Experts[1]. They underlined the importance of these studies in diagnosis, prophylaxis, assessment and clinical management of respiratory impairment. The armamentarium of investigations can aid correct diagnosis of the underlying lung condition, often without need for biopsy.

Chest Radiograph

This is a useful diagnostic tool when linked with symptoms and knowledge of occupational exposure. In pneumoconioses caused by coal dust and silica, the International Labour Office Classification[2] uses three parameters: type of opacity, their quality and quantity. The UICC/Cincinnati classification[3] has been developed for use in pneumoconioses including asbestosis. It can easily by appreciated that, however, in many occupational lung diseases the chest radiograph is either normal or exhibits changes which are not diagnostic.

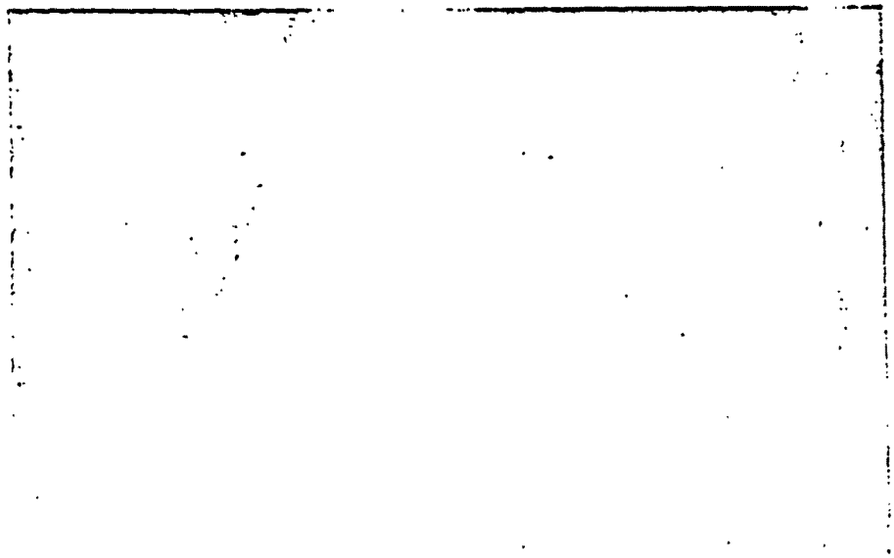
Diffusing Capacity (DL_{CO})

This is reduced in the earlier stages of asbestosis and farmer's lung, and also in progressive massive fibrosis. It is commonly abnormal towards the later stages of most of the pneumoconioses.

Sputum Examination

Tuberculosis (frequently the anonymous mycobacteria) is a secondry invader of patients with coalworkers' pneumoconiosis and silicosis, so sputum must be cultured at intervals.

The presence of asbestos fibres in sputum is evidence of recent exposure, and of asbestos bodies, of past exposure. Neither is evidence of lung damage. Although carcinomata are uncommon in occupational lung disease, they must be suspected in high risk groups.



ASBESTOSIS with calcified plaques is shown in this X-ray of the lower lung fields of a sixty year old patient. Her asbestos ...

Walker's paper, "Occupational Lung Disease". The complete text is available in booklet form to readers of RNB. Six charts are included: Commoner Pneumoconiosis, Irritant or Toxic Vapours, Organic Dusts/Fungi, Etiology of Commoner Conditions Caused by Occupational Exposure, Established Causes of Carcinoma Affecting Respiratory Tract, and Possible (Suspected) Causes of Carcinoma Affecting Respiratory Tract. To obtain a copy, simply address a request to Vitalograph Ltd. Publications Department, Maids Moreton House, Buckingham (England) or 8347 Quivira Road, Lenexa, Kansas (U.S.A.).

Skin Tests

There is a specific skin test for berylliosis. The Kveim test should be borne in mind, as sarcoidosis, although not an occupational disease, mimics many such diseases.

Lung & Pleural Biopsy

The high speed drill[4] for lung biopsy has made histological diagnosis possible in difficult cases of miliary mottling of chest radiographs and diffuse lung fibrosis of unknown etiology. Likewise pleural biopsy using the Abram's needle is helpful in suspected mesothelioma and tuberculosis.

Immunological Techniques

These are particularly useful in cases of suspected extrinsic allergic alveolitis. Here circulating antibody can be measured by techniques to differentiate specific precipitins. This is invaluable in the determination of etiology.

Spirometry

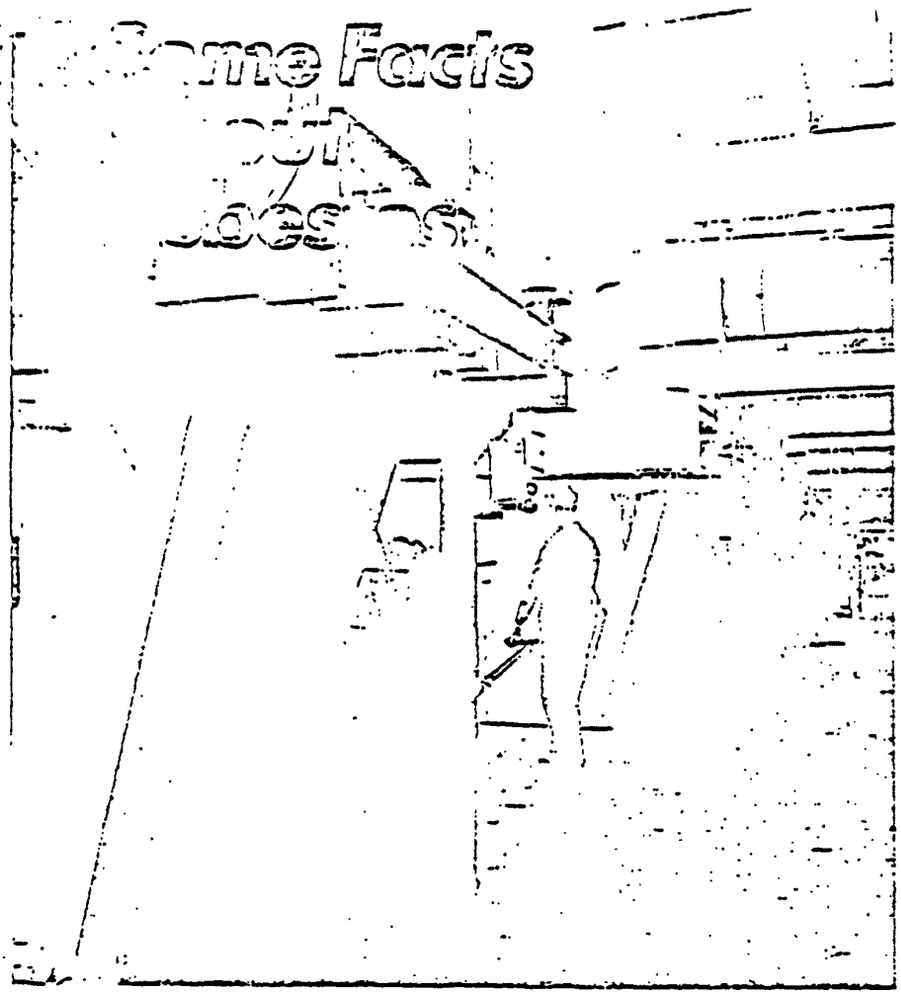
The physician can use the timed spirometer (Vitalograph) in his office, at the first and many follow-up visits to record and document the $FEV_{1.0}$ and FVC in his patients. By this means one has a sensitive indicator for assessing normality or abnormality, progression of disease, the effect of removal from exposure to inhaled material, together with efficacy of specific therapy. All occupational diseases demand this investigation. It would seem prudent to examine at intervals workers without the disease, but who are in potentially hazardous occupations, as in this way, the best prophylactic occupational medicine can be practised.

The example, par excellence, where spirometry detects earliest disease is in byssinosis. At this time it is reversible, and long before any radiographic changes are seen. A similar group are the extrinsic allergic alveolitides.

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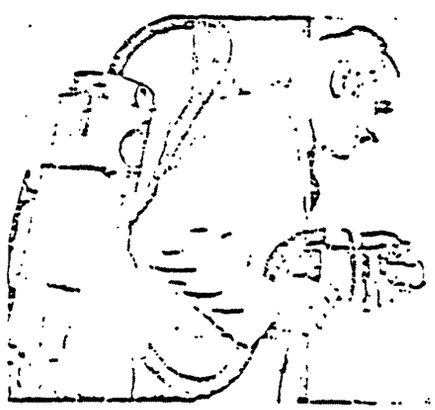
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REDUCED EXPOSURE to asbestos dust: adherence to government regulations which lay down rules for the protection of workers in the modern asbestos industry — and the enlightened self-interest of the industry itself — have prompted innovations in the handling of this potentially carcinogenic material. ABOVE, asbestos fibre is fed into carding machines under controlled conditions from a remote feedroom. BELOW LEFT, a portable exhaust-ventilation unit is used for drilling asbestos insulation board.

Photos courtesy of Turner & Newall Limited.



Asbestos is the generic name given to a class of natural fibrous silicates of which the two principal mineralogical groups are serpentine and amphibole asbestos.

Chrysotile, by far the most abundant form of asbestos, is a fibrous form of serpentine asbestos. Chrysotile asbestos fibres are flexible and have a high tensile strength, with diameter varying between 10 to 80 nm, but with a mean value of 30 to 40 nm. Chrysotile fibres are made up of bundles of fibrils, usually exhibiting a curved and twisted morphology in air samples and in histological sections (Timbrell, 1970). The macro-fibres — hundreds of thousands of fibrils in parallel array — are relatively easily separated into fine, flexible bundles.

The properties of chrysotile asbestos which make it particularly valuable to industry are length, strength, flexibility and a minimum of electrically conductive particles. Its resistance to alkaline attack makes it useful as a reinforcing fibre in cement. Asbestos textiles are almost entirely based on chrysotile fibres.

Production of chrysotile asbestos accounts for 95 per cent of the total of nearly five million tons of asbestos mined annually. Production of amosite and crocidolite (blue asbestos) fibres accounts for most of the rest.

The amphibole asbestos, crocidolite, differs from chrysotile in containing more silica, iron, aluminium, sodium and calcium and less magnesium. It is the strongest of the asbestos fibres and has a superior resistance to acid attacks. While no longer imported into the U.K., blue asbestos was widely used in the construction of older buildings as lagging and insulation. Risk of exposure to crocidolite is present as these buildings are demolished.

Amosite, another amphibole asbestos, has good tensile strength and in certain product formulations can have a higher heat resistance than chrysotile or crocidolite.

Amosite, anthophyllite and crocidolite fibres in air samples and histological sections are generally straight (Timbrell, 1970; Timbrell *et al.* 1970).

The largest commercial deposits of chrysotile are in the Ural Mountains and in Quebec Province. Most of the crocidolite fibre comes from the North-Western Cape and Transvaal areas of South Africa. Amosite, too, comes from the Transvaal, and anthophyllite from Finland. World production of asbestos has increased ten-fold in the last 40 years.

The modern industrial use of asbestos dates from about 1880 when it was first used to produce heat and acid-resistant fabrics. Present-day uses of asbestos run into the hundreds. A partial list:

friction materials (clutch plates and brake shoes), insulation of high-temperature vessels and pipes (e.g. in ships and power stations), protective clothing (against flame, heat and molten metal splashes), safety curtains, fire blankets, fire and smoke roof barriers, asbestos webbing (for door seals, thermal insulation, fuse box linings, lamp and stove wicks), asbestos tubing (switch-box seals, heat resistant cable covering), asbestos millboard, compressed fibre jointing (in boiler connections, sealing materials in internal combustion engines), rubber-proofed packings in hydraulic applications, dry asbestos packings (flame resistant sealing strip between walls and ceilings of hospital buildings), asbestos-cement pipe (for water supply, sewerage and drainage), asbestos insulating board (fire protection in buildings and in ships), asbestos paper and felts (flooring, roofing, automotive gasket materials), asbestos-reinforced thermoplastics (water filter units, door chimes, cooling fans, road signs, aircraft components) floor tiles, spray insulation, industrial talc, paper making.

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