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BIOLOGICAL EFFECTS OF ASBESTOS*

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SECTION III. HUMAN EXPOSURE TO ASBESTOS: INDUSTRIAL POPULATIONS

ASBESTOSIS IN GREAT BRITAIN

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Our knowledge of asbestosis dates from a post-mortem examination carried out at the Charing Cross Hospital, London, 64 years ago. The findings of that post-mortem examination were given by Montague Murray¹ in evidence before a Departmental Committee on Compensation for Industrial Diseases in 1906. He gave a good description of the morbid anatomy of pulmonary asbestosis and he produced photographs taken from specimens showing spicules of asbestos in the lungs of his young patient who died after 10 years exposure to cardroom dust. Eighteen years were to elapse before another post-mortem examination again directed attention to the possibility that silicates may cause extensive pulmonary fibrosis. In the interval interest was concentrated on what was regarded as the more important and widespread risk associated with dusts containing free silica. The second case was reported by Cooke² in 1924, the first to appear in English medical literature. His case is frequently associated with the discovery of "curious bodies" but what Cooke demonstrated in the lungs of a young tuberculous female textile worker were actual particles of asbestos fibers and dust, and he always insisted that proof of the diagnosis did not include the presence of "curious bodies."

Then suddenly, between 1927 and 1931, a whole new literature relating to asbestos appeared in Britain. The clinical picture of pulmonary asbestosis was described by Oliver,³ and the clinical and X-ray findings by Seiler,⁴ Burton Wood,⁵ and Ellman.⁶ Stuart McDonald⁷ described the histology of pulmonary asbestosis, and referred to certain highly characteristic yellowish brown bodies abundant in all sections. He advanced the hypothesis that the bodies were portions of asbestos fibers in the process of alteration. Stewart⁸ described how the characteristic bodies could be detected immediately by making a lung "squeeze." Unfortunately he described them as "asbestosis" bodies. Later Roodhouse Gloyne⁹ suggested that "asbestos" bodies would be a better name. In the new literature the report by Merewether and Price¹⁰ in 1930 was outstanding. The report gave their conclusions and recommendations following an extensive investigation of workers and of conditions on the manufacturing side of the asbestos industry and was important on a number of counts. On the medical side the investigation established the occupational cause of asbestosis. Merewether found that one in four of the sample of 363 workers examined had evidence of pulmonary fibrosis attributable to asbestos dust and he calculated that on the sample, one in eight of all workers in the industry would be found to have evidence of fibrosis. It was made clear that the

London, England

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principal safeguard against the ill effects of asbestos dust in the lungs was to be found in improved ventilation and dust suppression, and a number of recommendations were made.

The results of the report were also important. Under section 79 of the Factory and Workshops Act 1901 the Secretary of State issued a certificate to the effect that "the manipulation of asbestos and the manufacture and repair of articles composed wholly or partly of asbestos and processes incidental thereto are dangerous." The section in the Workmen's Compensation Act of 1925, which provided for the application of that Act to workmen suffering from silicosis was extended to certain processes involving exposure to asbestos dust,¹¹ and in 1931 asbestosis became a compensatable disease. Compensation cover was fairly wide and the compensatable disease was defined as "fibrosis of the lungs due to asbestos dust or that disease accompanied by tuberculosis." Medical Boards of specially qualified medical practitioners were appointed to make the medical examinations and give the medical certificates, initial and periodical examinations of workers in certain scheduled processes in the industry were instituted, and the standard of physique required on initial examination was prescribed.¹² Special regulations which defined asbestos as "any fibrous silicate," lay down specific instructions as to exhaust ventilation, the prevention of dust gaining access to the atmosphere of workrooms, the damping of floors and benches, the cleanliness of floors and plants, the storage of asbestos, and the employment of young persons. The regulations¹³ came into force with general application on March 1, 1932.

Case finding in Great Britain then has arisen mainly in two ways — from the statutory periodical examinations carried out in certain high-risk occupations in the industry, and from examinations following applications for compensation under the Workmen's Compensation Acts and claims for disablement and death benefit under the Industrial Injuries Act,¹⁴ which superseded the Workmen's Compensation Act of 1948.

Under the Industrial Injuries Act the words silicosis and asbestosis were dropped and the comprehensive term pneumoconiosis was introduced. Pneumoconiosis was defined in the Act as "fibrosis of the lungs due to silica dust, asbestos dust or other dust..." The Act provided that the effects of tuberculosis (pulmonary) in a person suffering from pneumoconiosis were to be treated as the effects of the pneumoconiosis. Cover was provided for all the main occupations in the industry:

- (a) the working or handling of asbestos or any admixture of asbestos;
- (b) the manufacture or repair of asbestos textiles or other articles containing or composed of asbestos;
- (c) the cleaning of any machinery or plant used in any of the foregoing operations and of any chambers, fixtures and appliances for the collection of asbestos dust;

- (d) substantial exposure to the dust arising from any of the foregoing operations.

The diagnosis and disablement questions arising on claims for disablement benefit under the Industrial Injuries Act are decided by the Pneumoconiosis Medical Panels of the Ministry of Pensions and National Insurance. The Panels also advise Insurance Officers on death claims. Post-mortem examinations are carried out in the vast majority of cases and the lungs are always examined by two members of the Panel; thus in a number of cases not seen in life the diagnosis of asbestosis has been made post-mortem. A number of cases have also been found following examinations under a special benefit scheme for older workers who were time-barred for compensation under the Workmen's Compensation Acts. The Panels also carry out the periodical medical examinations at the prescribed interval of two years. These different kinds of examination provide the only source of information about the prevalence of asbestosis in the country as a whole.

Between 1931 and December, 1963, 748 cases of asbestosis were diagnosed in life under the Workmen's Compensation Act and Industrial Injuries Act.

TABLE 1 shows the number of cases diagnosed each year under the Industrial Injuries Act. It shows that the number of new cases is increasing. The average of 45 for the last five years suggests that the incidence rate may be at least five per thousand exposed.

Four of the Ministry's Pneumoconiosis Medical Panels account for the

TABLE 1
NUMBER OF NEW CASES OF ASBESTOSIS DIAGNOSED IN LIFE
UNDER THE INDUSTRIAL INJURIES ACT, 1948

1950 (9 months)	17*
1951	17
1952	15
1953	23
1954	31
1955	48
1956	31
1957	56
1958	27
1959	37
1960	29
1961	43
1962	52
1963	67
Total	494

*Detailed statistics of cases under the Industrial Injuries Act were not maintained until April 1950.

TABLE 2
NEW CASES OF ASBESTOSIS 1955-63 ANALYZED
BY PRINCIPAL OCCUPATION*

Principal occupation	Grand total	
	No.	%
Opening, disintegrating	41	16.6
Insulating	72	41.0
Laggers		
Sprayers		
Mattress makers		
Others	12	
Weaving	16	21.5
Carding, spinning, etc.	37	
Slab and pipe making	20	8.1
Brake lining	4	1.6
Miscellaneous	27	11.0
Total	247	

*At four Panels—London, Manchester, Newcastle, Sheffield.

majority (63 per cent) of asbestosis cases. They are also the Panels mainly concerned with periodical medical examinations. In the nine-year period 1955-1963 these Panels diagnosed 247 fresh cases of asbestosis including 34 female workers. They have been grouped according to the processes in which each was mainly employed and with similar processes grouped together.

TABLE 2 shows the cases analyzed by principal occupation. It shows that the insulating section of the industry gave rise to 41 per cent of the total. The occupations of opening etc., carding and spinning, and the miscellaneous group which includes maintenance men were important causes of asbestosis.

TABLE 3 shows whether diagnosis was the result of initial or periodical examination, or the result of a claim. Well over half the cases resulted from direct claims, and the majority of these (60 per cent) were from the insulating section of the industry.

TABLE 3
NEW CASES OF ASBESTOSIS DIAGNOSED 1955 - 1963*

Principal occupation	Diagnosis resulted from		Total
	Initial or periodical exam.	Claim to benefit	
Opening, disintegrating	24	17	41
Insulating Laggers	3	69	72
Sprayers	—	13	13
Mattress makers	5	—	5
Others	7	5	12
Weaving	13	3	16
Carding, spinning, etc.	29	8	37
Slab and pipe making	8	12	20
Brake lining	2	2	4
Miscellaneous	13	14	27
Total	104	143	247

*At four Panels—London, Manchester, Newcastle, Sheffield.

TABLE 4 shows the cases analyzed by principal occupation and date of entry into the industry. The year 1933 was chosen because it was considered that by that date the regulations regarding ventilation and dust control were effective. It is recognized, however, that there were enormous difficulties in this regard during the war years. The majority (66 per cent) of the group entered the industry in 1933 or after.

TABLE 5 shows the average exposure and the range of exposure in the principal occupations. The figures of average exposures in opening, and disintegrating and weaving are an improvement on Merewether's figures for these occupations in 1930, but there is no room for complacency. Since 1933 the disease has been produced over short exposures. In practically every principal occupation asbestosis has developed following exposures of under 10 years. It is still developing.

from	Total
time benefit	
17	41
19	72
13	13
-	5
5	12
3	16
8	37
12	20
2	4
4	27
3	247

Sheffield.

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TABLE 4
 NEW CASES OF ASBESTOSIS DIAGNOSED 1955 - 1963*
 ANALYZED BY DATE OF ENTRY INTO INDUSTRY

Principal occupation	Entered industry before 1933	Entered industry 1933 or after	Total
Opening, disintegrating	3	38	41
Insulating			
Ladders	41	31	72
Sprayers	1	12	13
Mattress makers	2	3	5
Others	3	9	12
Weaving	6	10	16
Carding, spinning etc.	10	27	37
Slab and pipe making	3	17	20
Brake lining	3	1	4
Miscellaneous	10	17	27
Total	82 (33%)	165 (67%)	247 (100%)

*At four Panels—London, Manchester, Newcastle, Sheffield.

TABLE 6 shows an analysis by some of the principal occupations and date of entry into the industry after 1950 — well after war conditions applied. Two of the insulating workers who entered in 1956 had only four years exposure. The sprayer who entered in 1953 had seven years exposure. The worker who entered carding and spinning in 1952 had five and one half years exposure. One of the sprayers who entered in 1951 and had nine years exposure has since died and asbestosis was confirmed at post-mortem.

Scrutiny of the material suggests that age at entry into the industry was not a factor in the development of asbestosis. At periodic and other examinations the facts about duration of exposure and dates of exposure can be elicited, but these examinations have not so far provided the means of relating working conditions as measured by dust counts, to development of the disease. The tables, and the knowledge of the processes involved in

TABLE 5
NEW CASES OF ASBESTOSIS DIAGNOSED 1955 - 1963*
(WORKERS WHO ENTERED THE INDUSTRY IN 1933 OR AFTER)

Principal occupation	Average exposure in years	Range of exposure in years
Opening, disintegrating	14	4-20
Insulating		
Laggers	26	8-44
Sprayers	8.3	3-12
Mattress makers	11	5-22
Others	13	9-27
Weaving	15	9-23
Carding, spinning, etc.	16	6-27
Slab and pipe making	16	5-30
Brake lining	14	12-16
Miscellaneous	16	4-26

*At four Panels—London, Manchester, Newcastle, Sheffield.

the principal occupations suggest that development of the disease was related to dustiness of the job. Scrutiny of the working histories suggests that "range" of exposure depended on, for instance, whether raw material was freely handled, whether laggers and their laborers made mattresses on the site and broke down old asbestos lagging, and whether high speed cutting and sawing were carried out in the sectional departments.

Diagnostic Criteria

Diagnosis was made on adequate exposure to asbestos dust plus two positive findings from the following — presence of basal râles, finger clubbing, radiological appearances and pulmonary function studies.

Experience has shown that in some occupations recent exposure of three to four years is adequate, and this may be related to type of asbestos. Generally speaking, however, something like eight years exposure is required. On the other hand, very short past exposures 20 years ago are also adequate.

TABLE 6
NEW CASES OF ASBESTOSIS DIAGNOSED 1955-1963*
ANALYZED BY PRINCIPAL OCCUPATION AND DATE
OF ENTRY INTO THE INDUSTRY AFTER 1950

Principal occupation	Year worker entered industry						Total
	1951	1952	1953	1954	1955	1956	
Opening, disintegrating	1	—	—	—	1	1	3
Insulating							
Sprayers	2	—	1	—	—	1	4
Mattress makers	1	—	—	—	—	1	2
Weaving	—	1	1	—	—	—	2
Carding, spinning, etc.	1	3	—	—	—	1	5
Slab and pipe making	2	—	—	—	—	—	2
Total	7	4	2	—	1	4	18

*At four Panels—London, Manchester, Newcastle, Sheffield.

Characteristic persistent crackling râles at the bases were very commonly present. Finger clubbing, though not so common as basal râles, was noted in 35 per cent of the total. This finding, however, was subject to inter and intra observer error.

The radiological appearances were very variable. A large number of films showed the changes characteristic of interstitial fibrosis or of pleural thickening, or both. A honeycomb appearance just above the diaphragm, a "ground glass" appearance made up of linear markings and small discrete opacities in the lower lobes, obliteration of the costo phrenic angle, shaggy heart shadow, and thickening of the interlobar septae were common findings. Calcified plaques were also fairly common and were seen mainly in the films of insulation workers. The Pneumoconiosis Medical Panels consider that bilateral calcified pleural plaques are almost diagnostic of asbestosis. The absolute symmetry in many cases and the slow progression can hardly be explained except on the basis of occupation.

The Pneumoconiosis Medical Panels had found that workers with asbestosis had little tendency to asthma and other forms of airways obstruction but that they did not do a stepping test well. The pathology of asbestosis — the small firm lung that stands up on the table, that cuts with a clean edge, that shows parenchymal fibrosis without significant pleural

thickening, or shows marked pleural thickening without or with very slight macroscopic parenchymal fibrosis but with extensive microscopic fibrosis suggested that a useful diagnostic aid and a much better assessment of disablement would be provided by using multiple tests. Since 1960 the Panels have referred asbestos workers to special centers for full investigation of lung function. The tests have shown a highly characteristic and consistent combination of changes, e.g. small inspiratory capacity, M.B.C. well maintained for lung volume, high F.E.V.% V.C., reduced diffusing capacity, and loss of compliance. These are specific to a small group of diseases, of which asbestosis is one.

The disablement at the date of diagnosis was assessed as slight in 140 cases, moderate in 81 and severe in 26. Two cases were complicated by active tuberculosis at the date of diagnosis, two by pleural tumor and two by carcinoma of the lung.

Follow Up

Of this series of 247 cases 59 have died. The average age at death from all causes was 57 years. One death was not reported to the Pneumoconiosis Medical Panel and in one other case there was no post-mortem examination. The causes of death in the remaining 57 were as follows:

Asbestosis (cor pulmonale)	8	
Asbestosis + acute pulmonary infection	9	
Cancer of lung	21	} 40 per cent
Mesothelioma of pleura	2	
Mesothelioma of peritoneum	1	
Other cancers	3	
Coronary artery disease	5	
Other cardiovascular conditions	5	
Acute intestinal obstruction	1	
Acute meningitis	1	
Mesenteric thrombosis	1	

In three cases the diagnosis of asbestosis was not confirmed post-mortem. In one with a history of 12-years exposure to dust in coal mines and an adequate exposure to asbestos dust, the slight radiological changes were due to coal miners' pneumoconiosis. In two cases the diagnosis of asbestosis during life was based mainly on a low diffusing capacity. Post-mortem examination showed only gross emphysema. Excluding these cases the average age at death of those who died from asbestosis was 57. The average age at death of those who died from cancer of the lung or pleura was 55 and the average age of those who died from other causes was 59. These findings are similar to those in 138 consecutive post-mortems on asbestos workers in the area on the London Panel. The average age at death from

all causes in the London group of 106 males was 57 and of 32 females 58. The average age at death of the males who died of asbestosis or asbestosis plus infection was 56 and of females 59. The average age at death of males who died from lung cancer was 57 and of females 59.

Under the National Insurance (Industrial Injuries) (Prescribed Diseases) Regulations, sequelae of prescribed diseases are treated as prescribed diseases. The Pneumoconiosis Medical Panels accept lung cancer as a sequela of asbestosis. In view of the abnormally high prevalence of lung cancers associated with asbestosis the risk of including lung cancers due to other causes has been accepted and this attitude seems not unreasonable. In the 21 cases who died of lung cancer in the 1955/63 series the average interval between first exposure and death from cancer was 27 years. There was only one in which the period was less than 15 years. This worker entered the industry at the age of 59 and was exposed to asbestos dust for six years. There was very slight asbestosis post-mortem. Was this an industrial cancer? We would appreciate the views of the Conference on this problem.

In addition to the figures of death from lung cancer, four of the survivors in the series are at present permanently incapacitated by asbestosis and pleural effusion. In two, lung cancer has been diagnosed, and in the other two mesothelioma of pleura is suspected. Seven other survivors are permanently incapacitated by asbestosis and "bronchitis," and one is permanently incapacitated by asbestosis and tuberculosis. Scrutiny of the benefit history sheets (giving certified cause of incapacity) of the other survivors, shows that asbestotics are frequently incapacitated for long periods by "bronchitis" and "chest infection." However, a small number have reached retiring age without severe disablement and 27 have had no recorded chest illness since 1955.

Initial and Periodical Examinations

Against this background of the effects of asbestos dust the role of initial and periodical medical examinations of workers must be considered. Our Pneumoconiosis Medical Panels consider that these examinations of asbestos workers are the most worth while of all. No doubt the workers are happier when they see that they are being kept under observation by the Panel doctors. Regular visits to factories afford Panel doctors opportunities to meet factory doctors and to study the processes at first hand. Asbestosis is the most difficult of the pneumoconioses to diagnose and periodical examinations provide experience in film reading and in the clinical aspects of the disease. In this regard the initial X-ray and lung function tests carried out when the worker enters the industry can be used as controls. May these examinations, however, give rise in the industry to a false sense of security on the idea that the doctors have everything under control? What evidence

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is there that periodical examinations have prevented the development of asbestosis and its sequelae? As regards initial examinations the Pneumoconiosis Medical Panels have to decide whether new entrants are suitable for employment in the industry. The question that should be asked is whether the employment is suitable for the worker? In our experience very appreciable improvements in working conditions have been made in some sections of the industry by some employers. What some can do, others can do. Let us remember the answer and the warning given by Merewether 30 years ago. When asked whether two years exposure was sufficient to cause asbestosis in a young girl, he replied, "Yes, if she lives long enough."

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