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PLAINTIFF'S EXHIBIT

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In accordance with the policy adopted in 1928, the July and August issues will be omitted.

EXHIBIT 324

THE OCCURRENCE OF PULMONARY FIBROSIS AND OTHER PULMONARY AFFECTIONS IN ASBESTOS WORKERS

(Concluded)

E. R. A. Merewether, M.D.

H. M. Medical Inspector of Factories

Morbid Anatomy

Professor M. J. Stewart of Leeds has kindly supplied me with a detailed description (dated March 23, 1929) of postmortem appearances of several cases. From this account the following points are extracted:

The gross morbid anatomy of this lesion, as seen in persons who have been at work for many years in the dusty atmosphere of an asbestos factory, consists in a widespread pulmonary fibrosis affecting especially the basal region of both lungs. There are extensive, densely fibrous adhesions throughout the pleural sacs, which may, indeed, become completely obliterated. In particular, the bases of the lungs are firmly adherent to the diaphragm, and between these two structures there is often a thick, homogeneous layer of fibrous tissue, similar in appearance and consistence to yellow fibrocartilage. As a result of these two processes—pulmonary fibrosis and pleural sac obliteration—bronchiectasis develops in the midst of the more grossly diseased tissue and may go on to the formation of multiple abscesses with smooth walls.

The distribution of the fibrosis, apart from the extensive basal lesions, is rather irregular, the peripheral, subpleural regions of the lungs being more affected than the central areas. In one case in which the patient had been away from work for four years (following nineteen years in the mill), the areas of dense fibrosis were extraordinarily sharply circumscribed. The nodular character of the lesion which is so striking a feature of silicosis in its earlier stages is not met with in this disease.

The totally fibrosed areas of lung show grayish-black mottling, owing to immobilization of carbon. This tissue is excessively dense and completely airless; but there is no evidence of calcification. Other portions of lung show varying grades of the same process, and even the least affected parts are definitely tougher than normal. In none of the cases was there any evidence of an active tuberculous lesion.

Histology

The essential lesion is a chronic interstitial fibrosis of the lung. In the earlier stages, there is fibroblastic proliferation in the alveolar walls, which become increasingly thickened in consequence; and there is catarrhal desquamation of the alveolar epithelium. At this stage of the disease peculiar golden-yellow "asbestosis bodies" are found in varying, usually considerable numbers, both in the alveolar spaces and in the thickness of their walls. Detached portions of bodies may often be seen engulfed by alveolar phagocytes, or a large unbroken body may be partially surrounded by these cells. In the more advanced stages of the disease, the fibrosis of the lung is complete. Alveoli can no longer be made out: but the asbestosis bodies still remain embedded in the fibrous tissue. Varying quantities of carbon are also present, chiefly in little masses as though it had been contained within phagocytes. Chronic bronchitic

changes are present and all grades of bronchiectasis may be seen, in cases of long standing, from simply bronchial and bronchiolar dilatation, with walls still more or less intact, to large spaces filled with pus in which the original bronchial walls have become converted more or less completely into fibrous and granulation tissue. Many of the smaller arteries show obliterative endarteritis.

Reversionary metamorphosis of alveolar epithelium is frequent, and such alveoli are often filled with albuminous fluid, in which clumps of "bodies" may be found.

Asbestos Dust in the Lungs

Dr. W. E. Cooke has supplied (under date of June 25, 1929) details of his researches on asbestos dust in the lungs, and on the constitution of the curious bodies, which have been freely used in the following notes:—

Sections of lung and the results of digestion of the lung with trypsin show an enormous amount of fine black granular dust, much of which is carbonaceous. In addition, there are two striking features. The first is the almost complete absence of the fine translucent spicules of fiber which make up a great proportion of asbestos dust in factories. The second feature is the presence of large fragments varying in length from 10 to 360 microns. They are found in fibrotic and necrotic areas, singly and in groups. The particles are so large—masses of them are seen in some sections—that they must have occluded small bronchi and resulted in fibrosis of the surrounding area.

Comparing these large particles in the lung with those found in asbestos dust, close resemblance in sizes, shapes, and colors is apparent. There are the same black, blue, brown, and translucent fragments. In fact it is easy to take such a single particle from the lung and immediately find its brother in a slide made from the dust.

Curious Bodies

In addition to the fine granular dust and larger fragments of asbestos, sections of the lungs show curious bodies. They are found in alveoli, bronchioles, fibrous and necrotic areas, and in phagocytes in sections of both lungs. If a portion of lung is teased and extracted with water, or digested with trypsin, or, as Professor Stewart pointed out (1928), if a smear is made from the cut surface of the fresh lung, these bodies are seen in myriads. The larger bodies measure from 20 to 100 microns or more in length, and are of a golden-brown color. They may have single clubbed ends or may appear as elongated dumb-bells; some are filamentous, while others suggest a series of disks.

Single coccal and spore-like forms, and aggregations of these, and streptococcal forms are not uncommon; the color varies in the smaller types from a very pale yellow to a yellowish-brown. The bodies do not stain with any of the aniline dyes, but in chrysotile workers, they give the Prussian blue reaction for iron in varying degrees of intensity. These curious bodies have been found in every autopsy in pulmonary asbestosis. It would be unprofitable to retrace all the steps which led down many by-lanes during the course of the work, but I will mention a few pertinent details of interest.

Professor Stewart suggested to me

that a better method than simple extraction of the lungs with water or saline would be to digest portions with trypsin. During this process it was found that if the specific gravity was kept at about 1,070, the black dust and larger fragments of asbestos, as well as the partially digested lung tissue, sank to the bottom of the tube. By decanting the apparently clear supernatant liquid, and by centrifugalizing, neutralizing, and washing the deposit, the curious bodies could be obtained in a pure state. This was done, and sufficient material for X-ray examination was obtained. The bodies were attached to a hair with gum and subjected to a seven-hour exposure by Professor Bragg's method. If the bodies were mineral, the X-ray film would have shown a definite translatable atomic pattern. The films, however, did not do so, and we were then able to exclude the suggestion that the bodies were altered asbestos fiber. The result definitely pointed to the greater proportion of their composition being of nonmineral origin.

The bulk of the curious bodies is soluble in strong acids and alkalies, and if the solution is observed under dark ground illumination, their bases are seen as extremely fine spicules some of which by transmitted light would probably be invisible. Under a dissecting microscope it is possible partially to fracture the larger bodies and to show a central fine core.

The greater portion of asbestos dust consists of slender translucent fibers. In sections and extracts of the lungs there is a remarkable paucity of these fine spicules. The end-results of digestion show the fine granular dust and the large black, blue, and brown particles and what appear to be pieces of quartz. Relatively few fine spicules are found; but curious bodies of all descriptions are present in enormous numbers.

All these facts lead us to imagine the bodies to consist of a central nucleus of asbestos spicules, upon which colloidal aggregate of blood proteins plus, possibly, soluble fractions of asbestos and, in the case of chrysotile workers, an iron salt have been adsorbed and molded by currents in the bronchi and the alveoli.

The method of formation would appear to be as follows: The fine spicules of asbestos cause, by mechanical action on the bronchioles and alveoli, either minute extravasations of whole blood, or serous exudates which envelop them. Solution of any soluble fraction of asbestos takes place. The total amount of asbestos that is soluble must be extremely small, as is proved by the X-ray pattern of the curious bodies, but in the case of chrysotile workers some solution is suggested by the free iron reaction. We must remember, however, that the Prussian blue reaction may be due to the iron of the hemoglobin. Any surface in contact with a colloidal solution may act as an adsorbent, and in the present case the fine spicules must be considered to do so. Interaction between the soluble fraction of chrysotile and plasma proteins takes place, syneresis occurs, and, with the loss of water, the adsorption is rendered irreversible. The adsorbent is permanently enshcathed with stable colloidal aggregates which become molded into the familiar

shapes by alveolar and bronchial currents.

Support for this idea is found in the fact that micro-organisms adsorb colloidal material in the presence of blood serum and colloidal asbestos. Staphylococci so treated appear as large round yellowish-brown disks; in the process their property of staining with aniline dyes is lost. The organisms coalesce and form masses; and I think it probable that some of the coccal and spore forms are similar organisms.

Finally, are the curious bodies diagnostic of pulmonary asbestosis? Asbestos is unique among minerals in being fibrous; and its dust, generated during manufacturing processes, is also unique. As can be imagined from the formation of the curious bodies, there is no reason why, given any silicosis, a fine spicule of mineral should not have colloidal matter deposited around it and become molded into a curious body. But as no other mineral dust is fibrous, this occurrence must be so rare as to be negligible from a diagnostic point of view. The conditions which, apparently, must obtain for the formation of curious bodies are the presence of plasma proteins and fine spicules soluble only with difficulty. These conditions are ideally found in asbestos workers; for this reason I believe that curious bodies, if found in any numbers, are pathognomonic of pulmonary asbestosis.—

W. E. Cooke and also Roodhouse Gloyne (20) have independently demonstrated the presence of a mineral core, evidently derived from asbestos fibers, in the curious bodies. The bodies have been found (Stewart and Haddow (21)) in the intrathoracic lymphatic glands; in material obtained by lung puncture during life, first suggested by S. A. Henry; in the sputa; and in smear preparations from the cut surface of the lung. In smear preparations they can be readily demonstrated.

The importance of these findings lies in the fact that the bodies have never been found, as yet, in any other human affection. That similar bodies may be found to occur in the lungs of workers exposed to other dusts, is quite possible, although they have not been found in silicosis, nor were they present, according to Simson (6), in the fibrosed lungs of a hematite miner.

They appear within a comparatively short period of time after exposure to asbestos dust; at the moment, their presence in the sputa, in the absence of clinical or radiologic evidence of pulmonary fibrosis, cannot be taken as indicating anything more than previous inhalation of asbestos dust. The presence of the bodies, however, in the sputa of persons with signs of a diffuse fibrosis, or their presence in numbers on postmortem examination of a fibrosed lung, has evident implications.

Professor Stewart, who is continuing his work on this subject, has devised the following method of examining sputum for asbestosis bodies:

> Half an ounce or so of sputum is added to an equal quantity of undiluted antiformin. This is gently agitated until the sputum is completely dissolved, after which it is diluted with 2 or 3 ounces of water and allowed to stand in a large test tube for three or four hours. The bulk of the supernatant fluid having then been decanted, the remainder is centrifuged at a moderate speed for ten to fifteen minutes. The whole of the supernatant fluid is now poured off, and the deposit transferred to an albumin-

ized slide, by means of either a platinum loop or a pipette. After thorough drying on a hot plate and final fixation over a bunsen burner, the film is very gently washed in water, dried, and mounted in Canada balsam. After a little experience the asbestosis bodies are readily picked up with the low power, and their true nature is then confirmed with the ⅛-inch or oil immersion lens. As a rule they are present in very small numbers, perhaps only one or two in a whole film. In the case of some of the older workers, however, numerous bodies, up to one or two per field in certain portions of the film, have been found. It is obviously necessary to cleanse thoroughly all glassware, etc., used in making these examinations; otherwise there is a risk of contamination of subsequent specimens.

The films can be treated with hydrochloric acid and ferrocyanide of potash to demonstrate the Prussian blue reaction given by the bodies.

Asbestosis an Occupational Hazard

Out of a total of 374 workers examined, 105, or 28.1 per cent., were found to be affected with fibrosis of the lungs, in greater or less degree. But when these figures are corrected by the exclusion of cases in which previous work in dusty occupations (*e.g.*, quarrying, coal mining) may have been the prime, or a contributory, factor in the development of the fibrosis, ninety-five, or 26.2 per cent., of 363 workers were found to be affected with pulmonary fibrosis due to the inhalation of asbestos dust, and a further twenty-one, or 5.8 per cent., showed precursive signs of this disease.

This percentage may well be compared with the corresponding figures found by Middleton (22) in his examination of metal grinders (46.9 per cent.) and by Sutherland and Bryson (23) (24) in their examinations of potters (39.9 per cent.) and of sandstone workers (58.1 per cent.).

The precise significance of the lower figure found in this inquiry is not easy to determine. At first sight it seems to indicate that asbestos dust is less potent as a cause of pulmonary fibrosis than are the dusts containing free silica. Other factors, however—such as the measure of exhaust ventilation, or other means of reducing the concentration of dust, in the several industries; differences in the average length of employment of the comparable groups in the samples examined in the respective inquiries; and the relative numbers employed in the more dusty and the less dusty processes—will affect the crude incidence rates expressed by these percentages.

From comparison of the amounts of dust evolved—determined visually and by means of dust counts—in dusty asbestos processes, uncontrolled by local exhaust ventilation, with obviously defective exhaust ventilation, and with comparatively good exhaust ventilation, there can be no doubt that this preventive measure, which has been applied in some degree for more than seventeen years, has been a positive factor in minimizing the production of fibrosis—probably in the direction of lengthening the period before the fibrosis becomes fully developed.

Consideration given to the distribution of the workers examined both according to length of employment, and according to age, together with the incidence rates of fibrosis in each case, indicates the outstanding importance of length of employment (and hence length of exposure to dust), and the negligible effects of age, on the production of fibrosis. Thus age groups 30 to

39 and 50 to 59 have incidence rates of 30 per cent. and 37.9 per cent., respectively—not a wide difference, since the groups include workers in various processes exposed to different concentrations of dust. Moreover, the average length of employment in these two groups, excluding the cases of fibrosis, is also similar, but considerably less than the average length for all the cases of fibrosis (13.5 years).

No special susceptibility to the development of fibrosis is shown by young persons, unless it is considered that the figure of 8.7 years, the average length of employment of the cases of fibrosis in age group 20 to 29, has been shortened by increased susceptibility at ages under 20. As, however, the average age of this group of cases of fibrosis is 26.7 years, and also because the establishment of fibrosis does not necessitate immediate retirement from work, this is largely discounted.

TABLE 5.—DISTRIBUTION OF WORKERS EXAMINED WHO HAD BEEN EMPLOYED FOR FIVE YEARS OR OVER, TOGETHER WITH CASES OF FIBROSIS, ACCORDING TO PROCESS IN WHICH WORKER WAS LONGEST EMPLOYED

PROCESS	NO. EXAMINED	CASES OF FIBROSIS		AVERAGE LENGTH OF EMPLOYMENT IN YRS.	
		No.	Per Cent. of Group	Group Less Cases of Fibrosis	Cases of Fibrosis
1. Crushing, opening, disintegrating, and mixing	21	9	42.9	8.9	10.9
2. Carding	39	16	41.0	9.1	13.2
3. Spinning, twisting, doubling, plaiting, etc.	87	12	13.8	10.1	18.7
4. Mattress making	15	7	46.7	7.3	13.0
5. Weaving and associated processes	88	40	45.5	10.0	12.7
a. Cloth weaving	28	21	75.0	9.5	14.1
b. Band weaving	21	4	19.0	10.9	13.5
c. Cloth and band and unclassified weavers	14	4	28.6	7.2	9.5
d. Warpers, beamers, loom tuners, charge hands, and others associated with weaving	25	11	44.0	11.2	10.8
6. Miscellaneous processes and remaining unclassified workers	24	11	45.8	8.8	13.8
Total	274	95	34.7	9.6	13.5

The effects of length of employment are such that after five years' exposure, the incidence rate of fibrosis mounts rapidly, and after ten years increases almost in geometric progression. Table 5 shows the distribution of 274 workers examined, and of those with fibrosis, according to the process

in which they were longest employed. This was a compromise necessitated by the common customs in the industry of having several different processes going on in the same room, and of workers transferring from one process to another. Since the intention was to obtain some measure of the *relative* incidence of fibrosis in workers in the various processes enumerated, and since no case of fibrosis clearly due to asbestos dust was found among eighty-nine workers examined, who had been employed for less than five years, inclusion of this group of workers would only have introduced a varying source of error.

The processes differ, some very materially, in the amount of dust which they cause, and consequently in the amount inhaled by the operators (30). The outstanding point brought out by Table 5 is the relatively low incidence rate of fibrosis in "spinners" (group 3), as compared with each of the other groups. They are corroborated by counts of the dust particles in samples of air, a number of which have been taken and are discussed under *Dust Risk*.

There is also some indication in these figures that not only are "spinners" less likely to develop fibrosis, but when they do, it takes longer to develop. Study of the radiograms suggests that in such cases (*i.e.*, in the less dusty processes) the resulting fibrosis is, for a considerable period, much more linear in its radiologic features, and shows less mottling. The history and clinical features in these cases, and the comparative dust counts, all contribute to the view that with comparatively low concentrations of dust, the resulting fibrosis is longer in developing and remains longer in the milder, and radiologically linear stage.

This view is evidently of considerable practical importance since it suggests that in such cases, the rate of accumulation of dust in the lung has not greatly exceeded the rate of elimination, the changes still being centered in the main lymphatic system.

If this hypothesis is accurate, it should follow that in order to prevent the full development of the disease among asbestos workers, within an average working lifetime, it is necessary to reduce the concentration of dust in the air of the workrooms to a figure somewhat below that pertaining to spinning at the present time.

Fortunately, also, the spinning group is the largest individual group in this section of the industry—*e.g.*, out of over 400 workers employed by one firm, this group accounts for more than one-third.

Although all workers examined who have been included as spinners have been engaged in spinning, or in similar processes for convenience termed "spinning," for a longer time than on any other process, only a few have been employed solely on spinning, and in workrooms where no other, and more dusty processes, were being carried on.

These two factors, prior work in more dusty processes and present work in proximity to more dusty processes—especially the latter—have had, it is believed, some effect in raising the incidence rate of fibrosis for this group.

Dust Risk

In an effort to obtain additional data on the influence of concentration of dust in workrooms, a number of samples of air were taken and the dust con-

a shift, to determine the average concentration of dust. Repeated controls are also required.

Comparative data show that the dustiest processes are opening (with old-fashioned teasers), sieving (with no local exhaust ventilation), and shoveling, or otherwise handling asbestos fiber. The heaviest counts of all were found in this group, in filling sacks, by hand, with disintegrated fiber in a settling chamber. The visual cloud of dust was intense, and irritating enough to provoke immediate coughing on the part of the observer. The clumping of the dust particles of the sample on the cover slip was very marked, with the result that the total dust count arrived at was undoubtedly too low. With counts of asbestos particles of this order, however, the precise count is of little practical importance, since the dust concentration is far beyond the safe limit.

Next in order is cloth weaving, dry and without the application of local exhaust. This is undoubtedly a dusty process, and although local exhaust reduces the count at the breathing level of the weaver, much dust still escapes into the workroom. Further counts are necessary to estimate this. Weaving cloth wet, not merely damp, reduces the dust count to a remarkable degree. The figure for band (*i.e.*, narrow) weaving, wet, is not accurate since it was raised by dust from a neighboring dry cloth loom; still the improvement due to wet methods is noticeable. One asbestos weaver, who was formerly a cotton weaver, and who has to wear glasses, stated that whereas he used to clean his glasses about three times a day when cotton weaving, he has to clean them about five times a day when asbestos tape weaving dry, and only once a day when weaving asbestos wet.

Next in order comes mattress making, without any precautions such as exhaust ventilation, or damping floors, cloth, and tables. Application of exhaust ventilation and damping reduce the count considerably, but it is believed that the figures are too low, since counts for some subsidiary processes such as buttoning, sewing, and cutting out are not available.

The figures for spinning, plaiting, and braiding are believed to be rather too high, owing to contamination of dust from neighboring and more dusty processes, and further investigation is required here.

A number of the samples taken with the Owens apparatus were examined to determine the size of the particles, and the percentages of particles 2 microns and under, and 7.5 microns and under, were ascertained. The 2-micron standard was retained for purposes of comparison, and because of its accepted importance in silicosis. The 7.5-micron figure was also adopted because of the pronounced acicular character of much of the asbestos dust, and because, representing the diameter of the human red blood corpuscle, it is a standard easily distinguishable, and may conceivably represent the limit size of particles which can be conveniently engulfed by phagocytic cells, which are somewhat larger. Among twenty counts from various processes (Table 6), in two, between 60 and 70 per cent. of the particles were of sizes 2 microns and under; in two, 70 to 80 per cent.; in ten, 80 to 90 per cent.; and in six, 90 per cent. and upward. When the counts were analyzed ac-

cording to the 7.5-micron standard, all twenty showed 90 per cent. or more of the particles to be of this size or less.

With regard to the effect on the lungs of different varieties of asbestos, no evidence was found to indicate that any one of its varieties is more potent in producing fibrosis than the others, other factors, such as concentration of dust, being equal. There is now no doubt, however, that both chrysotile and crocidolite will produce fibrosis; while the third, the comparatively newly discovered amosite, resembles crocidolite so closely in its chemical constitution, and in the characteristics of the dust, that there can be no reasonable doubt, also, with respect to it.

Length of Dust Exposure

No case of diffuse fibrosis clearly due to asbestos was discovered with under 5 years' employment. Three cases were found with 3, 3½, and 4½ years' work, respectively, who showed clinical signs of fibrosis. Definite confirmatory radiologic evidence was obtained in the first, definite but slighter radiologic changes in the second, and indefinite suggestive changes in the third. The previous occupation in the first case was flax weaving for 18 years; in the second, coal hewing for 16 years; and in the third, work in an iron foundry for 1 year. All these occupations are dusty; and in the last two there may be some exposure to free silica. Also increased silica content has been found in the lungs, after incineration, of flax dressers. For these reasons, these three cases could not be certainly ascribed to asbestos dust, although it was, probably, at least a contributory factor. Among the cases of fibrosis found, thirty-six had been employed in asbestos for periods ranging from 5 to 10 years; two of these cases, one employed for 9 years and the other for 7 years, show that a considerable degree of fibrosis may occur with less than 10 years' exposure.

F. W. Simson (6) examined the lungs of a guinea-pig which had been experimentally dusted by Mavrogordato for two hours a day on each of fifty days between February and April, 1925, and which died from other causes in December, 1927. He states that histologic sections showed a slight generalized fibrosis. This observer also examined portions of the lungs of two native asbestos mill workers; one had been employed for twelve months and had died from a miliary tuberculosis, and the other, employed for two years, had apparently never recovered from an attack of lobar pneumonia a year before death.

In commenting on the amount of fibrosis found, he states that "a comparison between the human cases and the experimental animal showed that the fibrosis was more rapid and extensive in the human cases than in the experimental animal," and, again, that "the amount of fibrosis in two of the human cases . . . . was quite definite, and, if due to the presence of asbestos dust, the initial rate of production was rapid when compared with present-day non-infective silicosis on the Rand."

Experiments by Professor Beattie, in 1912, demonstrated that the lungs of guinea-pigs exposed to asbestos dust for forty-three and sixty-seven hours showed "definite cellular proliferation, though not very extensive, and this is

certainly a preliminary stage in the production of fibrosis."

These pathologic and experimental findings suggest that the inhalation of asbestos dust rapidly causes some changes in the lungs. But no evidence was obtained that asbestos dust can produce an acute type of fibrosis comparable to that formerly noted in South Africa after repeated exposure to massive concentration of free silica, a few cases of which have occurred recently in Great Britain, causing death in from two to four years.

The amount of disablement produced by the development of pulmonary fibrosis in these workers is surprisingly slight for a number of years, even more so than is generally the case in silicosis. The nature of the work, however, in the majority of the processes does not involve much physical exertion—in fact, in this respect it is "light work." The affected person may, and often does, continue at work —with occasional intermissions, latterly, due to exacerbations of bronchitis—until the condition is advanced, although he suffers increasing inconvenience from shortness of breath on the slightest exertion.

Usually these cases cease work a year or more before death, but sometimes a terminal bronchopneumonia, or other acute infection, commences while they are still at work, and there is no long period of invalidism.

Fatalities

Particulars of eight deaths have been collected, and are summarized in Table 7. In six of these, advanced pulmonary fibrosis was the primary cause of death. In the remaining two cases, pulmonary tuberculosis was a contributory factor. In six of the eight cases the diagnosis was verified by postmortem examination; in the other two cases, confirmatory radiographic evidence was obtained.

Information has recently been obtained in regard to three other cases, in which death occurred in 1929. The details of one have been published by Wood and Page (25). Postmortem examinations have been made in all three cases, and in all, the presence of pulmonary asbestosis, without tuberculosis, was verified. In one case, the pulmonary fibrosis was the primary cause of death; in one, information as to the extent of the fibrosis has not yet been obtained; and in the third case, a lobar pneumonia was superimposed on the fibrosis.

It is not suggested that these few fatalities, in which the cause of death has been verified by strict inquiry, are any criterion of the true effect of the disease on the mortality rates of asbestos workers. Others are known to have occurred in which the existence of the asbestos fibrosis has been determined in life, but no postmortem examination has been possible.

Difficulties in Diagnosis

Many factors have contributed to impede both the recognition of individual cases of this disease, and the establishment of asbestos dust as the determining cause. These factors are consequent, partly on the nature of the disease, partly on the nature of the dust, and partly on the industry itself, its rapid growth and internal conditions. Difficulties in diagnosing the disease, its points of resemblance, in its latest stages, to fibroid tuberculosis, and the liability of those affected to be

TABLE 7.—PARTICULARS OF FATAL CASES

NO.	CASE	DATE OF DEATH[1]	SEX	AGE	YRS. EMPLOYED	MAIN PROCESS	CONDITION OF LUNGS	POST-MORTEM EXAMINATION	INQUEST	REMARKS
1.	Montague Murray case	6/13/1900	M	34	14	Card room 10 yrs.	Advanced pulmonary fibrosis; no tuberculosis	Yes	No	.....
2.	Cooke's case	3/14/24	F	33	18[2]	Spinner latterly	Extensive pulmonary fibrosis and tuberculosis	Yes	No	Exposed to dust from other processes.
3.	H. S.	1927	F	48	24[2]	Mattress maker	Pulmonary fibrosis and tuberculosis	No	No	Two radiograms confirm diagnosis of pulmonary fibrosis.
4.	M. S.	3/ 7/27	F	34	20+	Mattress maker	Chronic pulmonary fibrosis; no tuberculosis	Yes	No	.....
5.	Grieve's case (W. L.)	3/24/28	M	34	13½	Mattress beater	Advanced pulmonary fibrosis; terminal bronchopneumonia; no tuberculosis	Yes	Yes	.....
6.	M. M.	8/25/28	F	34	15-16	Mattress maker	Advanced pulmonary fibrosis; no tuberculosis	Yes	Yes	Worked for less than the period stated, as a mattress maker.
7.	J. C.	10/13/28	M	36	18	Weaving 1 yr.; mattress dept. 7 yrs. and 2 mos.	Advanced pulmonary fibrosis; no tuberculosis	No	No	Two radiograms confirm diagnosis.
8.	L. H.	2/ -/29	F	41	10	Mattress maker	Advanced pulmonary fibrosis; no tuberculosis	Yes	Yes	.....

[1] Arrangement of dates follows the American custom—i.e., month, day of month, year.
[2] Estimated.

carried off by some intercurrent disease—which alone is noted on the death certificate—are all of importance.

A concrete example encountered during the inquiry will illustrate one of these points. J. C., Case No. 7 in Table 7, was known by his own doctor and by the Chief Tuberculosis Officer of the town to be affected with advanced fibrosis of the lungs. Four or five months before his death it was thought that a change of air would be of advantage to him. Through the generosity of the firm with which he had been employed, he was transferred from the town to a farm in the country, where he died. The death certificate, given locally, stated that pulmonary tuberculosis was the cause of death. In another case chronic bronchitis was given as the cause of death.

In the less advanced stages of the disease, the symptoms are so unobtrusive that the worker rarely consults his doctor. In the later stages, bronchitis, or the supervention of acute bronchopneumonia, pulmonary tuberculosis, or other acute infection, all so much more common, masks the underlying fibrosis; and the terminal condition is noted as the cause of death.

In the second place, the silicate dusts—of which asbestos dust is one—representing silica in the combined form as opposed to free silica, have naturally been overshadowed by the silica dusts, since they do not produce the picture of silicosis and have not been shown to be associated with an increased mortality from pulmonary tuberculosis.

Moreover, although the asbestos industry has very rapidly expanded, it is still comparatively small and scattered over the country. A large proportion of the workers have been employed in asbestos only for comparatively short periods, and of the processes considered so far, the less dusty spinning group is at the same time the largest. Thus only now the existence of a health risk in the industry is beginning to be recognized.

Does Asbestos Dust Predispose to Other Pulmonary Diseases?

The important question as to whether asbestos workers show an increased liability to other diseases of the lungs, such as pneumonia, pleurisy, and pulmonary tuberculosis—especially the latter—requires further investigation.

A history of pleurisy was given by ten workers, in eight since commencing work in asbestos: in addition, one worker had a slight pleural effusion at the time of examination, and one showed signs of old pleurisy, but no history was obtainable. Of the two with attacks prior to work in asbestos, one had normal lungs, and the other showed signs of a bronchiectasis. Of the eight giving histories of pleurisy, three showed signs of fibrosis, four showed signs of the old pleurisy only (one being due to a gunshot wound during the War), and the eighth showed signs of an old inactive tuberculous lesion of the right upper lobe.

A history of pneumonia was obtained in sixteen workers—in ten prior to commencing work in asbestos, and in six since. Of these six, two showed signs of a thickened pleura only, one showed merely enlarged lung roots, two had diffuse fibrosis, and in one the lungs were normal. Of the remaining ten, one had a thickened pleura, two bronchiectasis, two diffuse fibrosis, one

commencing fibrosis, and in four the lungs were normal.

With regard to evidence of bronchitis, and bronchial and pulmonary catarrh, fifty-eight of the 363 workers (16 per cent.) showed signs of one or other of these conditions, all being quite mild. Of these, thirty-one also showed signs of diffuse fibrosis, representing 32.6 per cent. of the fibrotic group, and eight were in the prefibrotic stage (38.1 per cent. of that group). The fact that the clinical examinations were conducted during the warmer months of the year, no doubt operated to reduce the incidence of these complaints. Under the circumstances, the preponderance among the cases of fibrosis is the more noteworthy, as indicating persistent irritation resulting from the dust.

Of other diseases, eight gave histories of rheumatic fever, and of these, two had definite valvular disease of the heart. In addition, valvular disease of the heart was noted in one worker, but no history of rheumatic fever was obtained.

With regard to pulmonary tuberculosis, so definitely an added risk in silicosis, this disease group has been drawn up to include all workers presenting signs indicating a past or present pulmonary tuberculous infection, but excluding cases of old inactive hilar tuberculosis. Under this heading are included not only workers with definite clinical signs of active tuberculous lung infections, or of old inactive lesions, equally definite, but also those workers showing signs of old apical collapse, usually attributed to a past tuberculous infection.

Signs of the latter are not uncommonly found in quite healthy persons, and are of no importance unless it is shown that the inhalation of asbestos dust is associated with pulmonary tuberculosis to an increased degree, when such cases, as indicating a group of workers with a latent infection, would assume a greater significance. No close association is apparent, however, since only thirty-seven, or 9.9 per cent. of the 374 examined, showed evidence of this disease, excluding cases of old inactive hilar tuberculosis, even when those showing the minor changes mentioned above are included. In only four was there a family history of pulmonary tuberculosis; and three out of four active cases belonged to this group. Thirty-three cases were inactive, of which twenty-one presented no evidence of dust fibrosis, and twelve presented evidence.

Out of the 374 persons examined, fourteen, or 3.7 per cent., gave a family history of tuberculosis. Evidence of active pulmonary tuberculosis was present in three; in two of these three the source of infection appears to have been a near relative.

Thus, no outstanding susceptibility to pulmonary tuberculosis was disclosed, either among asbestos workers as a class, or among those with fibrosis, considering the frequency of old healed apical lesions among the general population.

We have, however, by no means disposed of the question. The supervention of a tuberculous infection on a lung already the subject of fibrosis produces an increase in symptoms, previously unnoticed or disregarded, and causes the worker to seek his doctor's advice. He is then appropriately advised to give up his dusty employment, migrates from the industry, and

may or may not accept sanatorium treatment. Thus there tends to be a drift of such cases from the fibrosis producing industry.

During the course of the inquiry, information was obtained of a number of persons, previously employed in asbestos, who were either at home or in sanatoriums, suffering from chest complaints. Where an examination of workers is confined to those at work, a certain number of advanced cases of fibrosis, and a certain number of cases of pulmonary tuberculosis, with or without an asbestos fibrosis, in persons who have either given up work, or who are off work temporarily, will be missed.

It is necessary, therefore, to leave this question of increased susceptibility to pulmonary tuberculosis in abeyance, pending further investigation.

With respect to any increased susceptibility to the supervention of other lung diseases in workers with an asbestos fibrosis, the data obtained were insufficient to lead to any conclusion, except as to bronchial catarrh. There are indications, however, that when pneumonia or bronchopneumonia supervenes on a fibrotic lung, the prognosis is grave.

Asbestosis and Silicosis Contrasted

In view of the relationship between the asbestos fibrosis and silicosis, since they are both varieties of fibrosis of the lungs caused by the inhalation of dusts, it seems desirable to summarize shortly the main points of similarity and dissimilarity between the two diseases, so far as has been ascertained.

1. Inhalation of asbestos dust, under favorable conditions of concentration of dust and length of exposure, will produce a serious and even fatal degree of pulmonary fibrosis.

2. The type of fibrosis produced, however, exhibits a number of divergences from that caused by the inhalation of free silica dust, and the outcome of these variations is not yet apparent.

3. The divergences noted so far are exhibited in the radiologic picture, and on postmortem examination of the affected lungs, both in the gross morbid anatomy and in microscopic sections.

4. In the latter case the appearances of the asbestos fibrosis seem to be sufficiently distinctive not to permit of confusion between it and silicosis.

5. The radiologic appearances of the asbestos type of fibrosis, even in the more developed stage, are more delicate, softer, and more diffuse than the silicotic fibrosis.

6. It follows, therefore, that an opinion as to the degree and intensity of an asbestos fibrosis, based on a comparison of the radiologic changes with those shown in standard silicosis films, will be an underestimate.

7. In the absence of a full medical and industrial history, possibilities of confusion of the two types of fibrosis will arise in the interpretation of radiograms.

8. There is evidence that other inorganic dusts, containing no free silica, may be productive of this fine type of fibrosis in varying degree.

Summary

1. There is a definite risk of the development of a diffuse pulmonary fibrosis in persons exposed to the inhalation of asbestos dust.

2. This risk varies directly as the length of employment (and hence

length of exposure to dust), and is unaffected by the age of the worker.

3. There is a considerable difference between the least dusty and the most dusty processes investigated, and there is a corresponding variation in the relative risk of the development of fibrosis in the workers in these processes.

4. With continued exposure to high concentrations of dust, the disease may be fully developed in seven to nine years, and may cause death after about thirteen years' exposure, exceptionally in a shorter period.

5. Fibrosis as it occurs in asbestos workers differs considerably from the disease as it occurs in workers exposed to free crystalline silica dust; it differs in the radiologic picture, and also on postmortem examination of the affected lungs.

6. The more dusty processes are those involving the preparation and manipulation of the raw asbestos (other than crushing), dry cloth weaving, and mattress making.

7. The less dusty processes are spinning and similar processes, and processes, other than dry weaving, involving the manipulation of the spun yarn.

8. Further investigation is required to determine whether there is any increased susceptibility to the supervention of pulmonary tuberculosis associated with the development of the asbestos fibrosis.

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BIBLIOGRAPHY

1. Departmental Committee on Compensation for Industrial Diseases. Minutes of Evidence, Appendices and Index, 1907. Cd. 3496, p. 127; Report, 1907. Cd. 3495, p. 14.
2. Cooke, W. E.: Fibrosis of the Lungs Due to the Inhalation of Asbestos Dust. Brit. Med. Jour., 1924, *2*, 147.
3. Cooke, W. E.: Pulmonary Asbestosis. *Ibid.*, 1927, *2*, 1024.
4. McDonald, S.: Histology of Pulmonary Asbestosis. *Ibid.*, p. 1025.
5. Seiler, H. E.: A Case of Pneumoconiosis. Result of the Inhalation of Asbestos Dust. *Ibid.*, 1928, *2*, 982.
6. Simson, F. W.: Pulmonary Asbestosis in South Africa. *Ibid.*, 1928, *1*, 885.
7. Royal Commission on Metalliferous Mines and Quarries. Second Report, 1914. Cd. 7476, p. 146.

8. Badham, C.: Notes on a Fine Type of Fibrous Pneumonokoniosis Produced by Silicates and Other Minerals. Studies in Indust. Hyg. No. 13, Rep. Dir.-Gen. Pub. Health, New South Wales, for 1927, Section I.-E, Indust. Hyg., p. 102. Sydney, 1929.
9. Clark, W. I.: The Dust Hazard in the Abrasive Industry: Second Study. This Jour., 1929, *11*, 92.
10. Pancoast, H. K., and Pendergrass, E. P.: Pneumoconiosis (Silicosis). A Roentgenological Study. New York, Paul B. Hoeber, Inc., 1926, p. 155.
11. Thompson, L. R., Brundage, D. K., Russell, A. E., and Bloomfield, J. J.: The Health of Workers in Dusty Trades. I. Health of Workers in a Portland Cement Plant. U. S. Pub. Health Bull. No. 176, 1928.
12. Goadby, K. W.: Fibrosis of the Lung in Iron Miners. Jour. Roy. Mier. Soc., 1925, p. 432.
13. Annual Report of the Chief Inspector of Factories and Workshops for 1928, p. 94. London, H. M. Stationery Office, 1929.
14. Penny, F. W.: In "Industrial and Manufacturing Chemistry" by G. Martin. 1917, Part 2, p. 281.
15. Compiled by *Asbestos*, a monthly trade journal published in Philadelphia, Pa.
16. Annual Statement of the Trade of the United Kingdom, for 1922 and 1927. London, H. M. Stationery Office.
17. Departmental Committee on Compensation for Silicosis dealing with the Refractories Industries (Silicosis) Scheme 1919. First Report, 1924, p. 29.
18. Smith, A. R.: Silicosis among Rock Drillers, Blasters, and Excavators in New York City. This Jour., 1929, *11*, 39.
19. Wood, W. B.: Pulmonary Asbestosis. Tubercle, 1929, *10*, 353.
20. Gloyne, S. R.: The Presence of the Asbestos Fibre in the Lesions of Asbestos Workers. *Ibid.*, p. 404.
21. Stewart, M. J., and Haddow, A. C.: Demonstration of the Peculiar Bodies of Pulmonary Asbestosis ("Asbestosis Bodies") in Material Obtained by Lung Puncture and in the Sputum. Jour. Path. and Bact., 1929, *32*, 172.
22. Macklin, E. L., and Middleton, E. L.: Report on the Grinding of Metals and Cleaning of Castings with Special Reference to the Effects of Dust Inhalation upon the Workers. London, H. M. Stationery Office, 1923.
23. Sutherland, C. L., and Bryson, S.: Report on the Incidence of Silicosis in the Pottery Industry. London, H. M. Stationery Office, 1926.
24. Sutherland, C. L., and Bryson, S.: Report on the Occurrence of Silicosis among Sandstone Workers. London, H. M. Stationery Office, 1929.
25. Wood, W. B., and Page, D. S.: A Case of Pulmonary Asbestosis. Tubercle, 1929, *10*, 457.

For the geologic and the manufacturing and industrial aspects of asbestos, *see*:

26. Cirkel, F.: Chrysotile-Asbestos, its Occurrence, Exploitation, Milling, and Uses. Second edition. Ottawa, Govt. Printing Bureau, 1910.
27. Asbestos, its Sources, Extraction, Preparation, Manufacture and Uses in Industry and Engineering. Berlin, Becker and Haag, 1923.
28. Thorpe, E.: A Dictionary of Applied Chemistry. London, Longmans, Green & Co., 1912.
29. *Asbestos*, a monthly trade journal published in Philadelphia, Pa.

For description of processes, and further discussion on the incidence rate of fibrosis, and effect of work in different processes and also preventive measures, *see:*

30. Merewether, E. R. A., and Price, C. W.: Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry. London, H. M. Stationery Office, 1930.