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THE SILICA DUST HAZARD IN THE GRANITE CUTTING INDUSTRY*

L. R. THOMPSON, M.D.
Surgeon, United States Public Health Service

AND

ROLLO H. BRITTEN
Associate Statistician, United States Public Health Service

IN the past our knowledge of the effect of industrial hazards on the health of workers has been gained largely from occupational mortality statistics and the physical examination of sample groups of employees. It was believed that the dust hazard in this country offered an opportunity for an investigation of a more intensive character, by observing groups of workers over a period of time as to the character and severity of sickness which might be associated with the hazard.

In order to study the health of workers in the dusty trades, six specific investigations were undertaken, each dealing with a different type of dust. A list of these dusts follows:

1. Calcium dust (Portland cement plant)[1]
2. Silica dust (granite industry)[2]
3. Metal and other dusts (silver polishing)
4. Carbon dust (hard coal mining)[3]
5. Vegetable dust (cotton milling)
6. Municipal dust (street sweeping)

Two papers in regard to the study of calcium dust have been published in

* Received for publication Jan. 13, 1930.

[1] 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.

[2] Russell, A. E., Britten, R. H., Thompson, L. R., and Bloomfield, J. J.: The Health of Workers in Dusty Trades. II. Exposure to Siliceous Dust (Granite Industry). U. S. Pub. Health Bull. No. 187, 1929.

[3] Also a modified study in a soft coal district.

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

E. R. A. MEREWETHER, M.D.

H. M. Medical Inspector of Factories

INTRODUCTION

PRIOR to the commencement of this inquiry, in February, 1928, definite knowledge existed of only two deaths of asbestos workers about whom there was expert opinion that the inhalation of asbestos dust had at least contributed to, if not caused, the fatal outcome.

The first of these, in retrospect the most suggestive, only came to light some years after the occurrence, when full information was unobtainable. All that is known of this case, now referred to as "the Montague Murray Case," is contained in the evidence given by Dr. Montague Murray before the Departmental Committee on Compensation for Industrial Diseases in 1906 (1). From this source, we learn that:

> The patient, a male aged 32, came under the care of Dr. Montague Murray at the Charing Cross Hospital in the beginning of 1899. He had worked with asbestos for "some 14 years," 10 years as a cardroom hand, and the remainder in some other room of the factory, "where there was much less dust." He volunteered that, of the 10 people working in the cardroom when he went into it, he was the only survivor, and that all the others had died somewhere about 30 years of age. There is no note as to the nature of his work, previous to that in the asbestos factory.
>
> He was treated in the Charing Cross Hospital for two months, and then returned to work. After a few months, however, he became ill again, and was re-admitted to the Hospital in April, 1900, where he died. The post-mortem examination confirmed the clinical diagnosis of extensive pulmonary fibrosis. There was no evidence of pulmonary tuberculosis, and examination of the sputum for B. tuberculosis was negative.

The second case was reported by Dr. W. E. Cooke in 1924, eighteen years later (2):

> The deceased, a woman, aged 33, who died in 1924, had worked in asbestos for 18 years, but intermittently for the last 5 years, owing to periods of ill-health. The post-mortem examination revealed, not only extensive fibrosis of the lungs, but also much change due to pulmonary tuberculosis.

Although Cooke (3) and Stuart McDonald (4) were conclusively of the opinion that in this case the lungs showed a progressive dust fibrosis, together with a chronic tuberculous infection, the etiologic relationship between the inhalation of asbestos dust and fibrosis of the lungs would have been strengthened by the absence of a tuberculous infection.

Cooke's case is, however, of outstanding importance, not only because [illegible]

cause its publication again directed attention to the possibility that inorganic dusts containing little or no free silica may be productive of extensive pulmonary fibrosis. Of these dusts, the silicates form a very large class, of which asbestos is but one example. Many other members of the class, such as the feldspars, kaolin, French chalk, and pumice, are extensively used in industry. The importance, therefore, of delimiting the potentialities of the class as producers of pulmonary fibrosis is clear.

Cooke's case was the first to be generally reported in the medical press; the facts of the Montague Murray case, although contained in the evidence presented before the Departmental Committee in 1906, and published in 1907, were liable to be overlooked in the mass of important material on industrial diseases elicited by that Committee.

Since Cooke's case, Dr. I. M. D. Grieve has made a careful study of a group of asbestos workers in his practice, and has courteously allowed access to his records.

In February, 1928, Dr. MacGregor, Medical Officer of Health for Glasgow, drew my attention to an asbestos worker who was receiving treatment in one of the hospitals in that city. This case, the details of which have been reported by H. E. Seiler (5), presented, both clinically and radiologically, signs of a diffuse pulmonary fibrosis, with no evidence of a tuberculous infection. On further investigation into [illegible] industrial and medical [illegible]

This case, at that time the third of which the Factory Department had knowledge, was, however, the first in which the four essential conditions, necessary to establish a relationship between the inhalation of asbestos dust and the development of fibrosis, could be demonstrated. These conditions are:

1. Work involving exposure to asbestos dust.
2. The existence, demonstrable clinically and radiologically, of a definite pulmonary fibrosis.
3. The absence of previous or present infections known to cause pulmonary fibrosis—*e.g.*, tuberculosis, influenza, or pneumonia.
4. The absence of previous or present work involving exposure to other dusts which might cause pulmonary fibrosis.

These conditions being fulfilled, a relationship between the inhalation of asbestos dust and the development of the pulmonary fibrosis may be presumed.

The importance of establishing whether the supervention of this disease in an asbestos worker was an exceptional occurrence, or evidence of a grave health risk in the industry, was now apparent, and steps were taken, forthwith, to obtain *prima facie* evidence in proof, or disproof, of the existence of such a risk.

A number of workers in asbestos were selected and examined clinically and radiographically. The findings invited further investigation throughout the industry, with the result that a comprehensive inquiry involving the investigation [illegible]

with the carding, spinning, and weaving processes of the industry.

In the meantime, in March, 1928, the death of an asbestos worker (one of Dr. Grieve's cases) occurred in another part of the country, and, on post-mortem examination, a condition of widespread fibrosis of the lungs, without tuberculosis, was revealed. The microscopic examination also disclosed the presence of the curious bodies.

In 1928, also, Dr. F. W. Simson (6) reported a fatal case of fibrosis of the lungs occurring in a native working in an asbestos mill in southern Rhodesia.

In order that this brief survey of the events leading up to the present inquiry may be complete, it is necessary to review the investigation made in 1910 to 1911 by Dr. Collis and Miss Whitlock—the only previous inquiry into the subject.

In May, 1910, the Registrar-General drew attention to a death which had been certified as "acute pulmonary phthisis in an asbestos worker," and to a statement (for which he was unable to vouch) that seven other deaths from phthisis had occurred in the same factory.

Following this, extensive inquiries were made, a medical report on all the employees in the factory concerned was obtained, and Dr. Collis and Miss Whitlock investigated generally the various processes in the industry with respect to the evolution of dust, methods of ventilation, lost time due to sickness, etc. Also inquiries were made of the Canadian government as to the conditions in the asbestos quarries and mills in that Dominion, evidence of increased sickness and mortality rates among the workers, and any methods of ventilation which had been found of especial value.

The result of this investigation did not conclusively prove that asbestos possessed injurious properties, but it pointed to the probability that such was the case. It is interesting to consider why it was that no conclusive proof one way or the other could be obtained, then, as to the injuriousness of asbestos dust. Some, if not all, of the factors which affected the position then are, in varying degree, continuing factors, and have an important bearing on the present inquiry. Their operation, while providing a solution to this query, also affords an explanation to another pertinent question: Why is it that this industry, founded in antiquity, has only recently excited attention, by reason of its raw material becoming suspected as a cause of industrial disease?

The answer appears to be that in the past it was not practicably possible to obtain proof of the injuriousness of asbestos dust, considering the limitations imposed by the state of the industry, and the point reached by research work into the relationship between dust inhalation and diseases of the lungs.

The industry itself, not a large one today, was then (1910) considerably smaller. Certainly it had begun to grow rapidly, but the number of workers who could have been employed for a period of time long enough to allow of the development of definite physical signs of pneumonokoniosis must have been quite small, and dispersed over the country.

Precise knowledge of the morbid affections of the lungs produced by the inhalation of dusts was more fragmen-

tary than it is today. The position was clearly stated two years later in evidence placed before the Royal Commission on Metalliferous Mines and Quarries. This Commission, when reporting in 1914 (7), stated "we do not know whether other dusts besides those containing free crystalline silica induce a pathological condition in the lungs, though the experiments of Professor Beattie in animals suggest that this may occur." It is only in the last year or two that research has produced some definite evidence as to the precise effects of some of these dusts on the lungs (8) (9) (10) (11) (12).

In 1910, radiography of the lungs, in both its technical and its interpretative aspects, was still in its infancy and, so far from having attained its present status of being an indispensable aid to the diagnosis of the dust diseases of the lungs, was an unused ally.

In addition, the existence of a measure of exhaust ventilation in the most dusty processes of the industry, incomplete as it was, had important results in modifying the onset, course, and duration of any pathologic lung conditions resulting from the inhalation of the dust. This influence has been much more pronounced in the period intervening between 1912 and the present inquiry, and will be referred to again.

Another factor which has tended and still tends to obscure the possible deleterious effects of the non-silica dusts, is the general use of the phthisis mortality rate as a comparative index of the degree of injuriousness of the dust encountered in the various dusty occupations. This rate, while of great value in separating dusty industries into two [illegible] show an excess mortality from phthisis, and those which do not—as well as being a comparative index of the industries belonging to the former group, not only is of little value as a means of classification in the latter group, but also tends to distract attention from it, and to result in the associated dusts being dismissed as more or less innocuous.

Evolution of dust is only one factor, though an important one, which may cause variations in the phthisis mortality rate in different industries. Wages, hours of work, aggregation of workers, amount of food, housing, and other social and environmental conditions are, however, powerful influences in the same direction, and are not necessarily comparable as between the workers in any two industries.

In thus reviewing some of the influences which have retarded recognition of the baneful effects of some dusts upon the lungs, the singular attributes of pulmonary fibrosis, the most important of the diseases caused by the inhalation of dust, must not be overlooked.

This disease, insidious in its onset, stealthily advances with but faint warnings of its progress; inexorably it cripples the essential tissues of the lungs, yet for a considerable period causes almost no inconvenience to the worker. As time goes on, however, the lungs find more and more difficulty in re-aerating the blood; and breathing is quickened on slight exertion. Still the worker is able to remain at work, but is aware of his undue shortness of breath on extra effort. Usually, however, he ascribes it to causes other than the dust he is inhaling.

As the disease progresses [illegible]

a stage is reached when the lungs can do little more than maintain life; and the shortness of breath is extreme. Even in its terminal stages, the disease, deceitful to the last, may masquerade as chronic bronchitis, pulmonary tuberculosis, bronchopneumonia, or the like.

While more or less acute cases of fibrosis closely simulating miliary tuberculosis have occurred, even in this country (13), they have all been associated with the inhalation of dense concentrations of free silica dust, and are, fortunately, the exception rather than the rule.

The difficulty of diagnosing pulmonary fibrosis, especially in its early stages, or if complicated by tuberculosis, has been stressed by a number of authorities, and has, undoubtedly, contributed to impede the attainment of precise knowledge of the extent to which the various industrial dusts affect the lungs.

Difficulties and obscurities still impede, though to a less degree than in the past, any investigation into the effects of an industrial dust upon the lungs of those exposed to it; but prior to the War, although there were certain slight indications that asbestos, in common with some other dusts, might produce permanent pathologic changes in the lungs, it was not possible to obtain evidence sufficient to prove or disprove this hypothesis.

At the outset of the present inquiry into what, if any, pulmonary diseases workers exposed to the inhalation of asbestos dust are more prone to contract than the general population, it was considered essential to view the problem afresh, and with complete detachment, because of the fact that asbestos dust has a totally different physicochemical constitution from that of dusts containing much free silica and causing silicosis. It was felt that although much valuable guidance could be obtained from the methods of investigation of silicious dusts, care had to be taken to keep an open mind, so as not to be unconsciously biased in the direction of assuming that the effects of the dust, if any, must be comparable in some degree to those of crystalline silica.

Asbestos and the Asbestos Industry

Asbestos

The term asbestos is a collective name, of no definite mineralogic significance, which has been applied to a variety of silicate minerals, which differ from one another in chemical composition and physical properties, but which resemble one another in their finely fibrous feature and flexibility (14). Their value depends on the facility with which they are capable of being split up into long and flexible fibers, which can be spun like cotton and woven into cloth; on their resistance to heat and acids; and on their insulating properties with respect to heat and electricity.

Varieties of asbestos possess these qualities in differing degree. Commercially, therefore, selection is made by the manufacturer of that variety and grade which is most suitable for the purpose in view, regard being paid to ordinary economic factors, such as the cost of the raw material, and the price which the finished article may be expected to command.

Consignments of asbestos of the same general variety, but with differ-

ent countries of origin, are frequently mixed in the preliminary processes of manufacture. Thus Canadian chrysotile may be mixed with Russian, or Rhodesian chrysotile, and so on. Less frequently, totally different varieties, such as amosite and chrysotile, may be mixed.

Practically speaking, all that goes under the name of asbestos, in commerce, is either fibrous serpentine or a fibrous mineral of the amphibole, or hornblende, group. The former is the most important commercially; but strictly, the mineralogists confine the term asbestos to fibrous forms of hornblende. These two types are sharply distinguishable, chemically and mineralogically.

Serpentine asbestos, or chrysotile, is a hydrated magnesium silicate, containing practically no calcium, a high percentage of combined water, and a low percentage of iron. This variety is very suitable for spinning, but is attacked by acids. Nearly 80 per cent. of the world's production of asbestos is derived from Canada, and is of this variety; the remainder comes mainly from South Africa and Russia.

The amphibole or hornblende, varieties [illegible] and combined water, and usually more calcium, aluminium, and iron. Members of this group are resistant to acids, but are more difficult to spin, some being quite unsuitable for this purpose. The most important members of this group are crocidolite, amosite, and tremolite.

Crocidolite and amosite are mainly silicates of iron, the former having a beautiful lavender-blue color, the latter being brownish-gray. Both are spun and the yarn is woven into cloth for various purposes, such as acid filtering, and for making into insulating mattresses, as well as for other purposes. Both are produced extensively in South Africa, from which quarter all required commercially is obtained. Amosite, a comparatively recent discovery, is found there in very large deposits; its use is steadily increasing, the initial difficulties associated with the manufacture of textiles from this variety having been overcome.

Tremolite, found in various quarters of the world, has been mined in northern Italy since the time of the Romans. Its chief use is in the manufacture of asbestos millboard and for filtering purposes.

TABLE 1.—COMPOSITION OF SERPENTINE AND AMPHIBOLE VARIETIES OF ASBESTOS

MINERAL GROUP	VARIETY	PERCENTAGE OF							
		SiO_2	Al_2O_3	FeO Fe_2O_3	MgO	CaO	Na_2O	K_2O	Combined Water
Serpentine	Chrysotile	39–42.5	0–3.7	0.7–4.4	39–43	0–0.35	...	...	13.3–16.5
Amphibole (hornblende)	Crocidolite	50.5–52.1	0–1	35.5–37.4	0–3	0.75	6.2–9	...	1.6–4.5
	Amosite	49–53	1.2–9.4	34–44	0.7–6.4	...	0–2.5	...	2–3.5
	Tremolite	57.2	0.9	3.2	22.8	13.4	0.6	0.3	2.4

Table 1 [illegible] from [illegible]

sources, shows the main differences in the composition of these four varieties.

Only a very small proportion of the world's production of asbestos, which is between 300,000 and 400,000 short tons per annum, is suitable for spinning, and the most desirable grades of spinning fiber, consequently, command a high price—now over £100 a ton. The shortage of this grade has led to improvements in manufacturing processes which have enabled less expensive grades of fiber to be utilized for spinning, some of which give rise to an increased amount of dust.

About four-fifths of the world's production of asbestos is fiber unsuitable for spinning, and this is used in the manufacture of asbestos millboard, tiles, sheeting, paper, and many other articles. It is the discovery of industrial uses for these very short fibers, and the dust-like waste, which has been responsible for the phenomenal expansion of the industry as a whole. The spinning and textile section has also shared, because of the extensive use of the yarn and cloth in the manufacture of steam packings, insulating mattresses, brake linings for motor cars, fireproof curtains, and the like.

In 1880, three years after the discovery of the large Canadian deposits, the world production of asbestos was little over 500 short tons; by 1900 it had risen to about 35,000 short tons, by 1920 to over 230,000 short tons, and by 1925 to over 330,000 short tons (15).

The imports of asbestos (all grades) into the United Kingdom rose from 18,591 tons in 1922 to 33,520 tons in 1927 (16). The figures for 1927 refer to Great Britain and northern Ireland only. Of these quantities 8,844 tons and 3,794 tons, respectively, were reexported. Thus the consumption of asbestos in this country trebled within five years.

The Industry

On considering the uses of asbestos one is astonished, not only at the wide range of articles manufactured from this mineral, in greater or less proportion, but also at the diversity of industries which nowadays find its use necessary, either in the form of the raw material, or as manufactured articles.

Evidently, therefore, with the multiplicity of processes and dusts encountered in the ramifications of the industry, discrimination would have to be exercised, and some limit set to the processes included in the inquiry.

The processes selected may be divided, roughly, into

1. Processes involving the manipulation of asbestos, either pure, or admixed with a small proportion of cotton, or other vegetable fiber.
2. Processes involving the manipulation of asbestos together with other dusty materials.
3. Processes involving the making up of asbestos cloth into other articles.

Group 1 entails exposure to asbestos dust mainly, and to cotton, or other vegetable dust, very slightly; group 2 entails exposure to asbestos dust in very varying amounts, and also exposure to divers other dusts, such as brick dust, magnesia, kieselguhr, fossil meal, and cement; in group 3 the exposure to asbestos dust—provided no other asbestos processes are being carried on in the vicinity—is, in the majority of cases, negligible. Processes belonging to all three groups may be carried on in the same factory, and workers may transfer from one department to another [illegible]

Investigation into the intricate question of the effects of mixed dusts, while possibly productive of some general corollary, would lose much of its value in the absence of knowledge of the effects of the several component dusts, and moreover would introduce an incalculable variable into the final results. For these reasons, and also because the Montague Murray case, Cooke's, Grieve's, and Seiler's cases all occurred in processes included in group 1, it was considered advisable to exclude, as far as possible, from the inquiry all workers exposed to the influence of mixed dusts, and all those not employed in processes included in groups 1 and 3.

The processes included, therefore, are the crushing, preparing, sieving, opening, mixing, carding, spinning, doubling, plaiting, braiding, and weaving of asbestos, together with the operations incidental thereto. Also included is mattress making, where the filling is asbestos, and the manufacture of some insulating materials, where the dust produced is asbestos.

The carding and spinning processes have many points of resemblance to the corresponding processes in the cotton industry, but with essential modifications and restrictions caused by the different physical characters of the asbestos fiber. These processes are aided by an admixture of cotton, or other vegetable fiber, and usually from 2 to 10 per cent. by weight of cotton is added. More rarely, and for special purposes, long-fibered asbestos is carded and spun with no admixture of vegetable fiber, but usually asbestos yarns contain a core of either cotton or metal wire.

Asbestos yarns are largely used for the weaving of fabrics, which themselves are used for a multitude of purposes, but, braided together, are made into ropes for use as steam packings and other purposes. The interstices and center of the rope may be filled with other materials such as talc, oil, or graphite, depending on the precise use to which the rope is to be put.

Asbestos mattresses, used for blanketing steam engines, and for other insulating purposes, are made of asbestos cloth stuffed with asbestos fiber. The stuffing material may be, however, slag wool, magnesia (containing approximately 15 per cent. of asbestos fiber), or kieselguhr with a small percentage of asbestos fiber. The manufacture of mattresses filled with mixtures of asbestos fiber and other materials has not been included in this portion of the inquiry.

The shortness, slipperiness, and lack of strength of the individual asbestos fiber, as compared with cotton, flax, wool, silk, and other textile fibers, have been the cause of much technical difficulty in manufacture. The efforts of manufacturers to cope with these difficulties, together with those due to wide variations in the physical properties of the raw material, are reflected in the methods employed by each. For these reasons, and because of the great number of patented and special products manufactured, one finds considerable differences in detail in the processes in use.

The majority of the processes mentioned result in the evolution of dust, although by no means to the same extent. Differences in plant, quality of asbestos used, methods of manufacture, type of finished article, and extent of application [illegible] ventilation,

all result in variations in the evolution of dust in similar processes in different factories.

Variations in the evolution of dust in different processes being of outstanding importance, a series of samples of dust from the air of workrooms was collected, by means of the Owens jet apparatus.

Population at Risk.—A calculation of the total number of workers employed in the processes already enumerated as included in this inquiry, but excluding those engaged in processes included in group 3 in which the exposure to asbestos dust is negligible, gives a figure of approximately 1,600; if we could add to this the number of workers engaged in handling pure asbestos fiber only, in the preliminary stages of the processes included in group 2, we should obtain the total population at risk from the effects of asbestos dust itself. Unfortunately, this latter figure is unobtainable; but a rough estimate, and most probably an overestimate, is 600. Thus, about 2,200 appears to be the total population at risk in this country, for the purposes of this inquiry. This figure, however, does not include the large number of workers engaged in the processes in group 2 which involved exposure to the influence of mixed dusts, of which asbestos is but one, and commonly not more than 20 per cent. of the mixture.

TABLE 2.—DISTRIBUTION, ACCORDING TO LENGTH OF EMPLOYMENT, OF (A) 775 WORKERS ENGAGED IN ASBESTOS PROCESSES AND (B) SELECTED SAMPLE OF 363 WORKERS

YRS. EMPLOYED	(A)		(B)	
	NO.	PER CENT.	NO.	PER CENT.
0–4	483	62.3	89	24.5
5–9	200	25.8	141	38.8
10–14	51	6.6	84	23.2
15–19	24	3.1	28	7.7
20 and over	17	2.2	21	5.8
Total	775	100.0	363	100.0

This estimate of the population at risk, although it may be excessive, is useful, since it enables us to judge of the adequacy of the sample of workers examined, and to apply more correctly the incidence rates of any pulmonary affections disclosed by the examination of the sample (30).

The sample examined (after eleven cases are excluded of fibrosis and prefibrotic conditions due to causes other than the inhalation of asbestos dust) numbered 363, representing 16.5 per cent. of the population at risk, estimated as above. The manner of selection of the sample must be referred to, since just interpretation of the results depends upon a due appreciation of the relationship of the sample to the whole population at risk.

The principle of selecting primarily those longest employed was adopted; but regard was also paid to the other end of the scale, so as to obtain information as to the length of exposure to dust necessary before effects are manifested, and also as to the particular process in which the worker was engaged. It was felt that, in this way, the maximum information would be obtained in the shortest time.

Table 2 shows the distribution, according to length of employment (not necessarily in one factory), of

775 workers engaged in the processes under review, and of the sample of 363 workers, distributed in the same way. The enormous preponderance numerically of workers employed under five years in these processes is striking, as is also the very low percentage of workers employed ten years or longer.

Comparison of the two parts of the table shows that the effect of this method of selection is that the number examined in each succeeding five-year employment group is a progressively greater proportion of the total number which *could* have been examined in each particular group. With a solitary exception, all those examined were at work on the day of examination.

Not only the value, but the necessity, of radiographic examinations of the chest in investigations into the effects of dust upon the lungs, has been emphasized repeatedly by Watkins-Pitchford, and reaffirmed by the Departmental Committee on Compensation for Silicosis (17). A high standard of radiography is essential; as Watkins-Pitchford phrases it, the radiograms must be "technically satisfactory." Indifferent films are useless, since it is the fine detail of the lung which is being studied.

In a general inquiry, such as this, which involves the examination of workers in factories, large and small, scattered over the country, it is not practicable, nor is it necessary, considering the special purposes of the inquiry, for radiograms to be taken of all the workers examined. The dislocation of work in a factory, caused by the absence of a number of hands for this purpose, cannot be viewed with unconcern, especially when, as is not infrequent, the factory is remote from a center which is equipped with the necessary X-ray plant, and where the services of a consultant versed in the science of radiography of the lungs are available. Furthermore, it must not be overlooked that such examinations are voluntary, and not, as in South Africa, compulsory. Moreover, repeated and protracted examinations only result in the exhaustion of all concerned, and much depends on the willing co-operation of employers and employees, since the only incentive is a desire to further the common welfare.

Radiography, therefore, has a different function in these inquiries, than when it is applied to individual cases for compensation or other legal purposes. This function is not that of replacing careful clinical examinations, as has been recently foreshadowed (18, p. 40), but primarily that of being an indispensable aid in diagnosis, especially of doubtful cases, in the determination of complicating lesions, in measuring the extent and progress of the disease, in locating the point at which the earliest radiographic signs appear, and finally as a check upon the human factor presented by the examiner himself.

The examinations (except one) were carried out at each factory, in a room set apart for the purpose, suitably warmed, and with the necessary appointments. On occasions the noise of traffic or from the adjoining factory was a hindrance, but in one way or another these difficulties were overcome or minimized.

All the selected workers were examined by the writer. At three factories, however, a number of workers were examined jointly with Dr. E. L. Middleton. His far-reaching experience of the

pathologic changes in the lungs, produced by the inhalation of various dusts, was of great value, and his assistance was much appreciated.

Every effort was made to complete the clinical examination of the workers before the onset of winter introduced difficulties due to ephemeral bronchitis, colds, and influenza, which would tend to obscure the main issues. Thus the clinical, and two-thirds of the radiographic examinations were completed by the middle of November, 1928, prior to the commencement of the influenza epidemic early in 1929.

Clinical Examination

Percussion

The inhalation of asbestos dust originates changes in the lungs, which may be looked upon as a measure of the efforts of the living tissues to repel, or incarcerate, the irritant particles of dust. These changes modify the percussion note. It is true that the note elicited may be similar on both sides, but the note is not normal. It is thinner and higher pitched than normal, and there is a sense of resistance imparted to the plexor finger. In other words there is a diffuse, but slight, impairment of resonance.

This alteration in the percussion note, however, is more difficult to recognize because it is bilateral, and extends over a wide area: consequently the aid of contrast percussion is denied. It is best elicited by rapidly, and very lightly, percussing the back of the chest from apex to base on each side. It will be found that the extreme apexes remain clear, but below the apexes the impairment [illegible] these, it diminishes in intensity, but still persists. In other words, we find tacked on to the paravertebral dulness an area, above and below, of impaired resonance, which is much more extensive than that usually associated with old inactive hilar tuberculosis.

Impairment of the percussion note was found to be constantly more marked in the right side; in fact, at first it was thought that in the earliest degrees of fibrosis it was confined to that side, but later, and more extended observations lead to the conclusion that the earliest detectable cases are bilateral, although the signs on the left side are tenuous.

This change is so constant that it has been adopted as the most reliable single clinical sign presented by this type of pulmonary fibrosis, and no case has been classified as fibrotic in its absence.

Considering the frequency with which signs indicating what may be termed "enlarged roots" were found in asbestos workers, it may be that paravertebral dulness is one of the earliest signs produced by the inhalation of asbestos dust, indicating congestive changes in the root areas and a choking of the lymphatics with dust. Reflex impairment of note, due to irritation of the lung tissue by dust, is, however, an attractive explanation. It does not follow, of course, that workers presenting these signs will ever develop a definite asbestos fibrosis.

Clearly, the physical signs presented by any case of diffuse pulmonary fibrosis not only may be modified by changes produced by some intercurrent lung disease, but, *ab initio*, will vary [illegible]

the fibrosis may be implanted upon a perfectly normal chest, in which case the problem of diagnosis is straightforward. In other cases, however, preexisting root changes, so common in an industrial community, may be present, or the lungs may be already the seat of emphysema and chronic bronchitis, or of definite old tuberculous lesions, or there may be a massive pleural thickening, the result of an old pleural effusion. All these examples have been noted in the present investigation, and others will readily come to mind.

Although in most of these cases the dust fibrosis, if moderate in degree, can be confidently diagnosed, especially with the aid of radiography, it must be admitted that the problem becomes very difficult when the dust fibrosis is comparatively slight, and the changes produced by other conditions are pronounced and diffuse. Three types of these cases have caused most difficulty among those examined; fortunately, numerically they were few.

The first of these is where the dust fibrosis has been implanted upon lungs already emphysematous. It seems that, at any rate in the case of the asbestos fibrosis, until the fibrotic changes get the upper hand, clinical diagnosis of the fibrosis in these cases is impossible. Nevertheless, although the problem of the diagnosis of a fibrosis implanted upon an emphysematous chest has caused some difficulty in this inquiry, it seems likely that it can arise only under exceptional circumstances.

The second type of case which has [illegible] difficulty in clinical to healed tuberculosis. That there is an extensive fibrosis is clear enough, and that most of it is not due to asbestos is strongly suggested when examination of the lower portions of the lungs shows that they are comparatively slightly affected. Further help in these cases, of course, may be obtained from the history and symptoms.

The third type, also rare, is that in which changes following an old massive pleural effusion on one side so obscure the physical signs of any dust fibrosis as to render that side useless clinically for diagnostic purposes. If the side affected by the pleurisy happens to be the right, the radiographic picture is also curtailed by the normal partial obscuration of the left lung base by the heart shadow.

These three types were, with the possible exception of the first, uncommon, and are mentioned merely to call to mind some of the ways in which an asbestos fibrosis may be masked, in greater or less degree, by changes due to other disease.

Only passing reference need be made to the other physical signs found in the asbestos fibrosis, since they do not differ materially from those presented by silicosis. Chest expansion is diminished and may be reduced to one-half inch or even less in advanced cases. Retraction of the apexes is common, and sometimes shows a peculiar feature differentiating it from that found in fibroid phthisis. Instead of the immobile and sunken apexes seen in the latter disease, the apexes are seen to descend during inspiration, and to rise [illegible] expiration. This seems [illegible] the slackening of normal

mation of this was obtained radiologically.

Auscultation

In the majority of the cases of fibrosis, the respiratory murmur is weakened, much or little, generally, more on the right side, and often still more at the base; the expiratory sound is weaker than the inspiratory, and often becomes less and less audible as one approaches the bases.

Transitional phases between this and harshened breath sounds and prolonged expiration are not uncommon, even in the same chest. The latter may be noticeable in the upper portions of the lungs, but progressively diminish toward the bases. Other combinations were also noted, however.

The dry character of this type of fibrosis during most of its course is rather striking. Scattered fine râles and clicks in the root areas, axillae, and bases were not infrequent; slight edema of the lower halves of the lungs was noted in one of the more advanced cases; but in a number, no adventitious sounds at all were heard.

Pleural crepitations, and rarely a slight pleural rub, were noted—these attacks seem to cause little pain—and in one case a little fluid at the right base.

No doubt this variability in the sounds heard on auscultation reflects underlying changes, temporary or permanent, in the lung, and is dependent, *inter alia*, on the extent of the fibrosis with its associated pleural thickening—changes due to past disease, catarrh, or other intercurrent affection, and to the degree of compensatory emphysema present.

Symptoms

The symptoms exhibited by these cases of fibrosis, as might be expected, closely resemble those of silicosis. The distribution of the main symptoms, and of one sign, cyanosis, which is included with the symptoms for convenience, is given in Table 3. A few cases have been excluded on the grounds that one or other of the symptoms complained of might be assigned to causes other than the fibrosis, such as a complaint of shortness of breath in a case with a past history of mild thyroid intoxication, or of dyspnea associated with obesity.

TABLE 3.—DISTRIBUTION OF FOUR COMMON SYMPTOMS, AND OF CYANOSIS, AMONG CASES OF FIBROSIS

SYMPTOM	NO. EXAMINED[1]	CASES IN WHICH SYMPTOM WAS PRESENT	
		No.	Per Cent.
Cough	91	54	59 3
Cyanosis	93	52	55.9
Dyspnea	91	47	51.6
Expectoration	91	31	34 1
Pain	94	10	10 6

[1] Excluding those in which the presence of the symptom is referred to other causes.

Between 50 and 60 per cent. of the cases complained of cough, or of shortness of breath on slight exertion, or showed some degree of cyanosis, whereas only about one-third complained of expectoration, and one-tenth of pain, or discomfort, in the chest. Also, while 14.6 per cent. of the cases had no complaints and showed no cyanosis

cent. presented all four complaints and were cyanosed. 60.6 per cent. presented two, three, or four of the five items.

Clearly, none of the four complaints, or the presence of cyanosis, can be an infallible indication of the existence of fibrosis; but the presence of two of them, shortness of breath on slight exertion, and cyanosis in some degree, in an asbestos worker, is highly suggestive, in the absence of other evident cause.

Nevertheless an advanced degree of fibrosis may be present, and little complaint made. Symptoms, unless distressing, are so often a function of the introspectiveness of the patient. Slight degrees of fibrosis, too, give rise to no symptoms, in the absence of an intercurrent bronchitis, since the remaining sound lung tissue is still amply sufficient for all purposes.

Cough was the most frequent symptom, and was of two types. For a few weeks after commencing work in a dusty process, asbestos workers often find the dust irritating, and cough while in the dusty atmosphere; this lasts for a few weeks, and then usually disappears. The writer has noticed this effect on himself, but only while in a very heavy cloud of asbestos dust. The dust seems to be only slightly irritant in this way. One or two men who had previously worked in cotton card rooms, or blow rooms, and were affected by that dust, stated that they were unaffected by asbestos dust. Certainly asbestos dust does not cause asthmatic attacks like those seen in cotton card room workers. One man, who migrated from a cotton card room ("hard waste") nine years ago to an asbestos card room, on account of the [illegible] of the dust, does not find asbestos dust irritating, but to this day cannot work with a cotton scutcher (as he occasionally does) without precipitating a coughing attack within an hour or two. Although he suffers from winter cough and some shortness of breath, he stated that his health has been much better since the change.

Asbestos dust, therefore, has only very mild powers as a reflex irritant of the upper respiratory tract, and is, in this respect, comparable to free silica. This is an unfortunate attribute, since it leads to the assumption that the dust is more or less innocuous.

The second type of cough is more intimately associated with the development of fibrosis, and occurs, or perhaps is noticed, after a varying number of years' work. Usually it is present only in the morning on getting up, when after rather a sharp attack of coughing, a little viscid sputum "like an oyster" is brought up. A similar bout may occur at night after ceasing work; at times it is sufficiently sharp to cause retching. It is generally rather worse in winter, and may be noticed only then. Although it closely resembles smokers' cough, its features are precisely the same in nonsmokers.

Persons giving a history of cough dating from an attack of pneumonia or other illness in childhood, almost invariably state that the cough has not become worse since working with asbestos. One worker succinctly described the cough as "first in the throat; later catches the chest."

Generally, this cough causes very little inconvenience and may pass unnoticed until the development of some other symptom directs attention to the general state of health. Thus, out of

sixty-six cases of fibrosis, in thirty-three (50 per cent.) the cough was stated to have preceded the onset of shortness of breath, in twelve (18.2 per cent.) to have developed contemporaneously, and in twenty-one (31.8 per cent.) was either not noticed until after the onset of shortness of breath, or, although the latter was complained of, cough was not admitted.

Complaints such as "colds go to the chest," and "frequent colds on the chest," were not uncommon. Complaint of spitting of blood was exceptional; and in all cases in which this complaint was mentioned, the exciting cause was, primarily, some disease other than the fibrosis—*e.g.*, in one, it was due to pulmonary tuberculosis; in another, it occurred only during a prolonged attack of pleurisy; and in a third, the hemorrhage was gastric in origin. Cyanosis, a valuable sign when present, rarely amounts to more than a duskiness, or slight blueness, of the lips; it contrasts, however, with the general pallor of the face with which it is often associated in these cases. Cyanosis in some degree was noted in 56 per cent. of the cases. It may be absent even when there is a considerable degree of fibrosis, or it may come and go.

Pain in the chest is rarely complained of, and then it is usually described as "tightness of the chest," "soreness," or "aching." It was noted in 10.6 per cent. of the cases.

Well-marked clubbing of the fingers was noted in a few cases. The general nutrition is hardly affected, except in the latest stages. Only ten (10.5 per cent.) were noted as being thin. In one, the nutrition was [illegible] twenty-[illegible]

and in the remainder of the cases, good.

Radiography

Radiograms of the chest were obtained of 133 workers, or 35.5 per cent. of the number examined clinically. Of these, over 100 were taken by Dr. E. W. Twining of Manchester, and Dr. N. Tattersall of Leeds, and the remainder (except one) by Dr. F. L. Henderson of Glasgow. To all three, I am much indebted. Dr. Twining and Dr. Tattersall have devoted many hours to the joint study with the writer of the radiograms, and have drawn freely on their wide experience of radiography of the chest in furthering the purpose of the investigation.

Dr. R. S. Paterson of Manchester, too, added his experience to a final review of the films, and Dr. E. Barclay, now of Cambridge, lent his assistance in interpreting some difficult films; to both, grateful acknowledgment is made.

The standard of radiography was very high; indeed, a high standard is indispensable, if it is desired to trace the cause of such a fine and diffuse fibrosis as that produced by asbestos.

The films are superficially, but only superficially, comparable to silicosis. In the earliest negatives studied with Dr. Tattersall, a characteristic slight obscuration of the lung fields, a general lack of translucency, was noted. This appearance, for want of a better term, was denominated as "veiling." Dr. Burton Wood has independently noted (19) and confirmed this, referring to it as the "ground glass appearance." [illegible]

varying size seen in the striation in the mid-zones in some of the films.

There was general agreement as to the fine and delicate nature of the characteristic mottling. Dr. Twining (in a personal communication dated Aug. 2, 1929)—while lamenting that there has been, as yet, neither time nor opportunity to study the changes from every aspect, and therefore his present views may be modified on consideration of all the evidence—states:

> In general my opinion is that the earliest stages are not characteristic radiologically, but that the stage of fine dusty stippling is almost certain to be eventually provable as an early asbestos lesion. We saw it constantly in a large series of films, and after a little experience it is quite easy to detect it.
>
> Some of the other cases showed a few grouped lesions, like rosettes or leopard markings, each component being about the size of a primary lobule. These are similar to lesions sometimes seen in tuberculosis. In the asbestos cases I regard them as being groups of primary lobules making up a lobule, the walls of which are infiltrated. They certainly seem to correspond in size with the macroscopic lesions seen in the pathological specimen.
>
> The advanced cases show heavy basal and mid-field mottlings, common in pneumonoconiosis, with a tendency to avoid the apices.
>
> On the whole the lesions are distinctly less dense than those of silicosis, and are less easy to group into well-defined stages, but I think we can recognise:
>
> 1. A very doubtful stage of increased linear striations.
> 2. Fairly definite fine dusty stippled appearance.
> 3. Coarser mottling with increased linear striations.
> 4. Gross lesions with pleural changes and [illegible] due to the pull of the fibrous

The radiographic appearances in the earlier stages are most marked on the right side at the base or in the central zone. This corresponds with the clinical findings. This preference for the right side has been noted also in silicosis, by the South African observers. The cases showing radiographic signs of a dust fibrosis have been classified in three broad groups. This grouping, although unscientific, is convenient practically, considering the main purpose of this investigation. Indeed more precise classification based on the particular radiographic changes noted might well be misleading at the present time.

Much combined clinical, radiologic, and pathologic study is required into the whole subject of these fine types of dust fibrosis, which have been noted in workers exposed not only to silicate dusts other than asbestos, but also to other inorganic dusts containing no silica, before it will be possible to classify the radiologic changes as has been done so successfully by the South African workers in respect to silicotic fibrosis.

The hypothesis that, because asbestos dust produces a pulmonary fibrosis with consequent deviations from the normal in the lung skiagram, the degree and potentialities of this fibrosis can be assessed by comparison of its radiologic picture with those of standard silicotic films, is untenable.

At least two general types of pulmonary fibrosis caused by inorganic dusts can be recognized. A third, representing the purely peribronchial [illegible] of fibrosis, might be added; [illegible] dust fibrosis will

types. These types are (1) that produced by combined silica dust, an example of which is seen in asbestos fibrosis, and (2) that produced by free silica dust, represented by silicotic fibrosis.

These two types, while resembling one another in clinical signs and symptoms, differ materially both in the nature of the lesion produced in the lung, and in the character of the associated radiographic picture. Thus, attempts to weigh, consciously or unconsciously, asbestos fibrosis, or any dust fibrosis other than silicosis, by means of the standard radiographic changes found in silicosis, is unsound and is likely to lead to a misconception of the potentialities of the dust in question.

Badham (8) gives an example of this source of error in reporting the unexpectedly early death of a man affected with a fine fibrosis caused by an orthoclase basalt containing no free silica. Referring to some of the radiologic differences between the fine fibrosis of dusts other than silica and the nodular fibrosis of quartz dust (*i.e.*, true silicosis), he states:

> Moreover, the coarse fibrosis of silica gave clear interspaces of normal lung, while the fine fibrosis presented a uniform granular mottling leading to the conclusion which is probably erroneous that the actual development of fibrous tissue was greater in a nodular fibrosis as silicosis than in a generalised fine fibrosis caused by silicates. To me it appears that the mechanical damage to the lung is greater in a fine fibrosis than in a coarse fibrosis when both are well developed.

The evidence obtained in the present inquiry amply confirms this general statement.

On studying the development of asbestos fibrosis as displayed in a series of radiograms, and especially in the few available taken a year or more before a fatal termination, one cannot help asking the question, "What is there here which could have this effect?" The answer is that the damage to the lung is much greater than it appears to be when judged by the silicotic standard. Asbestos fibrosis is much more diffuse; it spins its fine web, as it were, crisscross throughout the lung, enveloping and eventually strangling the ultimate lobular structure, rather than depositing itself in numerous more or less isolated foci, as in silicosis. Thus, at any rate in the less advanced stages, the radiologic picture of the lesions does not impress the eye, unconsciously viewing it from the standpoint of silicosis.

The radiographic picture may show soft and fairly coarse nodulation, but it is never so impressive as the nodulation in a silicotic film.

Paradoxically, the distinctive feature, both clinically and radiologically, of the asbestos fibrosis is its uniformity. The modesty of the symptoms; the unobtrusive, but diffuse, impairment of the percussion note; the homogeneous stippling of the skiagram; all are fragments of an entity, unmistakable when assembled, but enigmatic when divorced.

Only two references to the radiographic appearances of the chest in asbestos workers have been traced: one in a report by Pancoast and Pendergrass (10), and the other in an article by Burton Wood (19).

Pancoast and Pendergrass together with Miller and Landis examined "17

first stage changes and the other 15 definite second stage appearances. Of the men longest at work, one after seventeen years' occupation showed very definite diffuse, 'soft' spots throughout both lungs, and another showed about the same appearance after fourteen years' occupation. Very slight nodular shadows were found in one man after only two years' occupation. Most of these second stage cases showed also well-marked first stage appearances still present, indicating a persistence of free drainage hilumward. In all instances the nodular shadows were characteristically 'soft' and varied considerably in size." Their first stage appears to correspond with stage 1, and their second stage with stages 2 and 3 mentioned above.

It appears from the context that these observers regard asbestos fibrosis as being really a silicosis due to admixture of free silica derived from the original rock—a view difficult to substantiate.

Burton Wood (19) in a series of fifteen skiagrams of asbestos workers notes: "The most noticeable feature of skiagrams of workers exposed longest to asbestos dust is the presence of shadows suggesting a diffuse fibrosis affecting chiefly the lower two-thirds of the lungs. The fine quality of the shadows is worthy of note. Some of the cases exhibit a 'ground glass' appearance, though on close inspection fine mottling is evident. . . . when more definite mottling is present it lacks the coarse quality described in the skiagrams of chests showing pneumonokoniosis, e.g. South African gold miners. . . ."

[illegible] maturation of asbestos [illegible] period of years and by the time the stage is reached when the features discussed above are, in varying degree, positive, and the radiologic picture is recognizable, the fibrosis is not in its inception, nor even in its earliest stages, but is developed and fairly widespread.

We must, therefore, recognize an earlier state when the fibrosis is present in slight degree, and also when there is evidence of choking of the lymphatics, and of a measure of pulmonary catarrh. This stage may be referred to conveniently as the prefibrotic stage.

The indications of this stage are indefinite, and some, at any rate, not specific. If, however, they can be determined and applied with only a moderate degree of accuracy, information of practical value will be available. Efforts have been made, therefore, to distinguish workers in this stage. The following tabulation shows that twenty-one workers out of 365 examined (5.8 per cent.) were so classified:

Clinical Examinations (365):	*No.*	*Per Cent.*
Fibrosis	95	26.2
Prefibrotic conditions	21	5.8
Radiologic Examinations (133):		
Fibrosis	52	39.1
Suggestive changes not definitely diffuse fibrosis	22	16.5

Many of these cases present a slight diffuse impairment of percussion note—perhaps better described as a slightly increased sense of resistance felt on percussion, mostly of the right lung, at least as contrasted with the left, and associated with some weakening of the respiratory murmur. Probably [illegible] early stage could be detected [illegible] by means of

comparison with a radiogram taken prior to commencing work with asbestos. By such means the earliest stages of silicosis have been worked out by the South African observers. There, the radiograms taken at periodic six-monthly medical examinations can always be compared with initial radiograms taken before the employees are permitted to work underground.

group is dismissed from any further consideration.

A general summary of the findings in the 374 workers who were examined clinically, classified under the most important lesion, is presented in Table 4.

Illustrative Cases

The salient points of a few cases are set out below to illustrate clinical and

TABLE 4.—GENERAL SUMMARY OF FINDINGS CLASSIFIED UNDER THE MOST IMPORTANT LESION

YRS. EMPLOYED	NO. EXAMINED	CASES OF FIBROSIS		PRE-FIBROTIC CONDITIONS		PULMONARY TUBERCULOSIS			CASES OF OLD HILAR TUBERCULOSIS, OR SUGGESTING ENLARGEMENT OR CONGESTION OF LUNG ROOTS	PULMONARY AND BRONCHIAL CATARRH, AND BRONCHITIS	OTHER PULMONARY LESIONS			WITHIN NORMAL LIMITS
		Due to Asbestos	Due to Other Causes	Due to Asbestos	Due to Other Causes	With Evidence of a Dust Fibrosis	Other Active Lesions	Other Inactive Lesions			Pleurisy[1]	Emphysema[2]	Other Lesions	
0–4	92	0	3	5	0	0	0	0	13	7	1	0	1	62
5–9	142	36	1	12	0	1	1	4	3	3	5	2	2	72
10–14	89	27	5	3	0	3	0	3	5	0	3	2	0	38
15–19	30	15	1	1	1	0	0	1	0	2	1	1	0	7
20 and over	21	17	0	0	0	1	0	0	0	1	0	1	0	1
Total	374	95	10	21	1	5	1	8	21	13	10	6	3	180
Percentage	100	25.4	2.7	5.6	0.3	1.3	0.3	2.1	5.6	3.5	2.7	1.6	0.8	48.1

[1] One case of thickened pleura due to old gunshot wound.
[2] One case of emphysema due to gassing.

In other cases included in this group, diffuse weakening of the respiratory murmur was noted, with fine sticky râles in the root areas. At present no stress can be laid upon this group. The clinical changes are so slightly marked that until comparative radiograms are available it would be unwise to draw any deductions

other features. Five radiograms are reproduced to illustrate the radiologic appearances of some of the cases. There are insuperable difficulties, however, in reproducing the finer changes depicted in the original negatives.

Case 1. This worker

and as a doubler nine and one-half years. Her family and personal medical history contain nothing of note. She has no complaints and feels quite well. Her nutrition is good. She is pale, but shows no cyanosis.

Chest.—She has a chest expansion of 1¼ inches. No impairment of the percussion note is detected. The breath sounds in the right lung are weaker, generally, than those in the left. Expiration is prolonged at the right root; no added sounds are heard. A skiagram showed some very slight and doubtful changes in the direction of increased striation.

Case 2.—This man, aged 24, has been employed in asbestos for five years, carding and mixing. Previously he worked for four years in the cotton trade, yarn weighing. His family and personal medical history contain nothing of note. He has had a slight morning cough and expectoration for a year. His nutrition is good. His color is fresh, and there is no cyanosis.

Chest.—There is some impairment of the percussion note over the middle third of the right lung, behind, and slightly at the right base. The breath sounds are harsh, and expiration is prolonged in the upper half of the right lung, behind; expiration is also prolonged over the left upper lobe, behind. The respiratory murmur is weakened at the right base, and the breath sounds are of a "whiffing" character. There are no added sounds. A skiagram showed enlargement of the right root, and slight haziness and striation in the central zones in both lungs, suggesting early fibrosis.

Case 3.—This man, aged 32, has been employed in asbestos for six years in the card room, and as a stripper and grinder. Previously he was employed in a cotton card room for nine years. He had army service for five years, but was not gassed. He gives a family history of asthma; otherwise there is nothing of note in his family or personal medical history. He has no complaints and feels quite well. His musculature is very good. His color is pale, but he shows no cyanosis.

Chest.—There is slight impairment of the percussion note generally, particularly

old tuberculosis of the lung roots and general increased striation, especially in the lower half of the right lung, suggesting early fibrosis.

Case 4.—This worker, a male, aged 23, has been employed in asbestos for nine years, mostly mattress making. His family and personal medical history contain nothing of note. He complains of occasional pain in both sides of the chest, and perhaps a little undue shortness of breath on exertion. His color is normal. His musculature and nutrition are good.

Fig. 1.—Case 4: Moderate fibrosis. Undue degree of striation and fine mottling in central zones of both lungs; calcified glands in roots, with rather coarse striation in upper lobes; ? interlobar pleurisy on right side, between the lower and middle lobes; emphysema at bases.

Chest. There is no retraction of the apexes, but there is a slight flattening below the right clavicle. The percussion note is slightly impaired generally, behind, particularly on the right side. The breath sounds are harsh, generally, and expiration is prolonged. No added sounds are detected.

the upper lobes; ? interlobar pleurisy on the right side, between the lower and middle lobes; and emphysema at the bases. The case was diagnosed as moderate fibrosis.

CASE 5.—This woman, aged 40, has been employed for eleven years in asbestos, for most of the period mattress making. Previously she was employed in a laundry. She has recently been ill for three months with pleurisy—no history of tapping; otherwise her family and personal medical history are negative. She complains of having had a winter cough for three or four years, and of shortness of breath on exertion since her attack of pleurisy. Her nutrition is good; her weight is stationary. She has normal color, and no cyanosis.

Chest.—There is some general impairment of the percussion note over both lungs, behind, more noticeable at the bases. The respiratory murmur is weakened generally. Persistent crepitations and medium rales are noted low down in the right [illegible]. A skiagram showed diffuse [illegible] and fine nodular mottling [illegible]

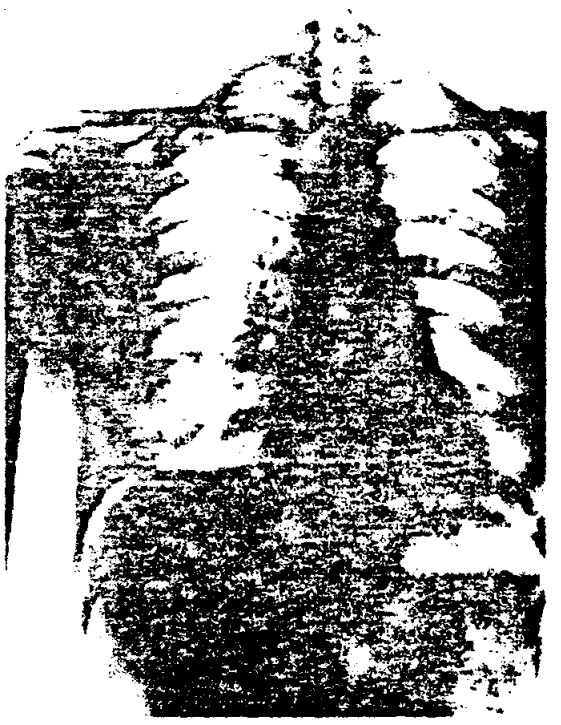

FIG. 2.—Case 6: Moderate, but fully developed, fibrosis. Enlarged glands in both roots; diffuse fine mottling in both lungs, especially the right.

moderate fibrosis, most marked toward the bases.

CASE 6.—This worker, a female, aged 31, has been employed in asbestos for seven years, opening, and has been exposed to much dust. Her previous employment was non-dusty. Her family and personal history disclose nothing of note. She complains of having had a cough for five years, and of shortness of breath on hurrying. She is thin and undernourished, and pale. Dust is present on her hair and in her nostrils.

Chest.—She has a chest expansion of one-fourth inch. There is slight general impairment of the percussion note, definite at the apexes and the bases. The breath sounds are weak; expiration is slightly prolonged. No added sounds are heard. Her heart is not enlarged, and no murmurs are detected. The skiagram (Fig. 2) shows a number of enlarged glands in both roots, and a diffuse fine mottling in both lungs, especially the right. This is a case [illegible]

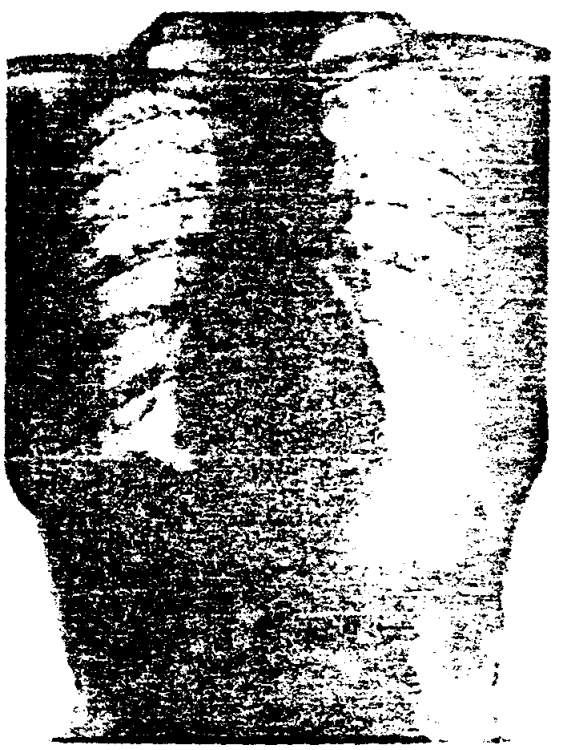

FIG. 3.—Case 7: General fibrosis, mostly linear, but with little mottling of right lung. Old puerile tuberculous scars at apexes.

employed in asbestos for thirty-two years—about three years on cards, and the remainder as a spinner. She has done no other factory work. Her family medical history discloses nothing of note. She has a personal history of nephritis seven or eight years ago, causing three months' illness. Her complaints are: shortness of breath on hills, and winter cough for about six years. Her nutrition is fair. She is rather pale.

Chest.—She has a chest expansion of one-half inch. No retraction of the apexes is noted. There is general slight impairment of the percussion note, behind. Breath sounds are harsh and expiration is prolonged. No added sounds are heard. No gross enlargement of the heart is noted, but the second sound is found to be accentuated over the aortic area. The skiagram Fig. 3 shows a general fibrosis, mostly linear, but with a little mottling of the right lung. There are also very old puerile tuberculous scars at the apexes. The case was diagnosed as general fibrosis.

This case should be compared with Case 6. In Case 6 there was a *heavy exposure to* dust extending over a few years; in the present case there was *exposure to a very much less concentration of* dust, except possibly in the first three years, but extending over *many years*.

Case 8.—This worker, a male, aged 62, has been employed in asbestos for twenty years as a weaver. He was previously a cotton and silk weaver. His family and personal medical history show nothing of note, except that he was regarded as a delicate child. His complaints are: shortness of breath on exertion, and on going upstairs, for three years; morning cough and a little expectoration for the last three winters. He is thin and pale, with some cyanosis.

Chest. Chest expansion is 1 inch; there is retraction of the apexes. The percussion note is impaired over both upper lobes in front, and over the upper two-thirds of the right lung, behind. Expiration is prolonged at the bases. Pleural rub and inconstant rales are noted at the left base, and a few rales at the right base. A skiagram [illegible] diffuse fibrosis [illegible]

employed for twenty years in asbestos in many capacities. There is nothing of note in his family history. He gives a history of pneumonia a number of years ago. His complaints are: a little shortness of breath on exertion for the last two or three years; otherwise none. His nutrition is fair only. His color is fresh, but there is slight cyanosis of the lips, at times.

Chest.—The skin is poorly elastic; there is some retraction of the apexes. There is an impaired, rather "dowdy," note on percussion, generally, especially over the upper half of the left lung, and over the upper two-thirds of the right lung, behind, not in front. Respiratory murmur is generally weak. Expiration is slightly prolonged, but diminishing toward the bases. Pleural crepitations are noted on deep inspiration at the right axilla. A skiagram shows [illegible] diffuse fibrosis. Neither lung expands well, and throughout both lungs is seen the diffuse reticulated appearance of moderate, but fully developed, fibrosis. The fine diffuse character of the lung appearances is particularly [illegible]

Case 10.—This man, aged 46, has been employed for twenty-five years in asbestos, during nine years of which he was not exposed to dust. For eleven years of the remainder he was employed in the card room, and mattress making. His family and personal medical history discloses nothing of note. His complaints are: shortness of breath on exertion for three months; cough after a few hours' work, during the last two or three years; [illegible] expectoration; and pain in the left side of the chest for the last three months. His nutrition is good. He is pale, with slight duskiness of the lips.

Chest. There is some retraction of the apexes. The percussion note generally is impaired, and of a "dowdy" character. Breath sounds are harsh and expiration is prolonged over the right upper lobe; elsewhere the respiratory murmur is a little weakened and expiration is not materially prolonged. No added sounds are heard [illegible] The [illegible]

has been employed for twenty-two years in asbestos—during the first eleven years on cards and as a weaver, during which he was exposed to a considerable concentration of dust. During the remainder of the time he was employed on a non-dusty process, but was exposed to a less concentration of dust derived from other processes. His family and personal medical history give nothing of note. His complaints are: shortness of breath on climbing stairs for the last five years; slight morning cough and expectoration for about the same time; and slight aching of the chest in front, below the left clavicle. *His nutrition is good. There is* slight cyanosis of the face.

FIG. 4.—Case 10: Fully developed fibrosis. Diffuse striation and fine speckled mottling throughout both lungs.

Chest.—Chest expansion is 1 inch; there is retraction of the apexes. There is impairment of the percussion note generally, most evident over the whole of the right lung, behind, and over the right upper lobe in front. The note is "hoxy" over the remainder of the chest. The breath sounds are slightly weakened generally, except at the right apex, but there is no gross change. Expiration is prolonged [illegible] crepitations [illegible]

over the right upper lobe, in front. The heart is normal. Sputum is mucoid, but is negative for Bacillus tuberculosis. Clubbing of the fingers has been noted for about two years. A skiagram showed an extensive diffuse fibrosis—fibrosis in a fairly advanced stage.

CASE 12.—This worker, a male, aged 60, has been employed in asbestos for twenty-six years in the card room. Previously he was a sawyer's laborer. There is nothing of note in his family and personal medical history. He complains of slight shortness of breath on exertion of recent years, and of winter cough for the last three or four years; expectoration is stated to be nil. His musculature and nutrition are fair. There is slight cyanosis of the face.

Chest.—He has a chest expansion of three-fourths inch. The percussion note is impaired generally, especially over the left upper lobe in front. The respiratory murmur is weakened over the whole of the chest except the left upper lobe, where there are whistling breath sounds, and medium râles. The heart is normal. A skiagram showed very extensive changes throughout the chest of a coarse type without fine stippling. The roots showed a choked appearance. There was heavy mottling at the base. One old tuberculosis focus was noted at the left base, and some calcified glands in the left hilum. The diagnosis was fibrosis in the advanced stage.

CASE 13.—This man, aged 66, has been employed in asbestos for twenty-one years as a weaver. For about sixteen years previously he was a cotton and silk weaver. His father died of "chest trouble" at the age of 45; his sister died aged 15 of pulmonary tuberculosis. He has only been away sick a week or two in twenty-one years, and has never had any serious illness. His complaints are: undue shortness of breath on exertion during the last two years; cough for the last two or three winters. Expectoration is creamy, and occurs a fair amount of times. He has lost weight, and is thin. He is pale, but with some cyanosis of the face, and with a malar flush.

Chest.—Chest expansion is 1 inches; there is retraction of [illegible] Dullness [illegible]

upper lobe, behind, and over both upper lobes in front. Breath sounds are rather weak, with prolonged expiration. Scattered variable rhonchi and râles are heard over the right lung, behind, over the left upper lobe in front, and over the left root. The skiagram showed a definite diffuse fibrosis plus tuberculosis. Sputum was negative (one examination). The diagnosis was dust fibrosis in the advanced stage, with tuberculosis, probably active.

CASE 14.—This worker, a male, aged 36, commenced work in an asbestos plant in 1910, but was employed on other work until January, 1914. During the next five years, until early in 1919, about three years and nine months were spent in army service, for nine months of which he was a prisoner of war, but he had two short periods of work in asbestos—eleven months in the weaving, and six months in the mattress, departments. From early in 1919 to November, 1927, he worked in asbestos processes, but for only six years and ten months in a dusty one (mattress making). He then ceased work. He was referred to a tuberculosis dispensary on Oct. 19, 1927, when he gave a history of bronchitis in 1916 and 1917 during the War, and of never being very well since; complaining of dyspnœa, pain in the right side, and a slight cough of only about six months' duration, with a small amount of grayish sputum. Dr. N. Tattersall states that at that time:

"Dyspnœa was very marked, even on talking, and there was definite cyanosis. He had marked lolllowing and respiratory retraction at both apices, especially the right; loss of note over most of the right lung and the upper half of the left; bronchial breath sounds at both apices; fairly numerous crepitations, mostly on the right side, and a pleural rub at the right base. He had a systolic bruit at the apex and pleuropericardial friction.

"I examined him a number of times, the last occasion being April 20, 1928. In that time he had lost 9 pounds, felt very weak, and the dyspnœa was increasing. The [illegible] remained much the same, though [illegible] and a cough, the sputum had remained small in amount, and its appearance did not suggest tuberculosis.

"My diagnosis from the beginning was one of pulmonary asbestosis with possibly added tubercle.

"I took two X-rays of him, neither of which showed appearances suggestive of tubercle, everything pointing to a very extreme degree of fibrosis."

He died on Oct. 13, 1928. No post-mortem examination was obtained.

FIG. 5.—Case 14: Massive fibrosis, with pleural thickening, retracted apexes, and pneumothorax on left side with incomplete collapse of lung.

It will be noted that the length of exposure to asbestos dust is unlikely to have been more than about ten years and two months, and of this time he was employed only about eight and one-half years in a dusty process. War service, of course, may have reduced his resistance. The skiagram (Fig. 5) shows a massive fibrosis with pleural thickening, retracted apexes, and pneumothorax on the left side with incomplete collapse of the lung.

[illegible] of fibrosis of the type pre-

finally to a fatal termination, even in the absence of a superadded tuberculous infection.

The primary effect of the fibrosis would appear to be that of causing defective aeration of the blood, resulting in an added strain on the heart. For many years, and even with an advanced degree of fibrosis this may be of little inconvenience, provided physical exertion is limited, and acute illnesses are avoided. Ultimately, however, the margin of safety, already diminished, is lost and the circulation slowly fails, with the usual signs of edema of the lungs. More often, however, an attack of bronchopneumonia, influenza, or other acute infection adds too much to the strain, impairs the cardiac musculature, and results in a fatal termination.

(To be concluded)

BOOK REVIEWS

BLEIVERGIFTUNG. By Prof. Dr. *Paul Schmidt*, Direktor des Hygienischen Instituts der Universität Halle a. d. S.; Priv.-Doz. Dr. *Adolf Seiser*, Oberassistent am Hygienischen Institut Halle a. d. S.; und Priv.-Doz. Dr. *Stilfried Letzner*, Assistent an der Medizinischen Klinik Halle a. d. S. Paper. Pp. 79 with index, illustrations, and bibliography. Berlin and Vienna: Urban & Schwarzenberg, 1930.

This monograph is divided into a short introductory or general section and a special section. The first part is devoted principally to a discussion of the outstanding diagnostic symptoms of lead poisoning and of the work done by Dr. Schmidt and his collaborators in the microchemical determination of lead in blood, urine, and feces. The second part discusses in detail the usual sources of lead poisoning; the absorption, distribution, and excretion of lead; the pathology and clinical findings associated with lead poisoning; and, finally, the diagnosis, prophylaxis, and therapeutic treatment of lead poisoning.

Dr. Schmidt's monograph is particularly apposite. It brings the literature of the subject down to the present date and represents the mature thought of an investigator who has long made this field particularly his own. The discussion of the diagnostic features of lead poisoning is especially clear and to the point. In this connection it is of interest to note that emphasis is placed on the importance of the determination of lead in the blood.

No attempt is made to discuss the details of technic. The monograph presents instead a critical review of the more recent experimental work relating to lead poisoning and will, in consequence, doubtless have a wide appeal to all investigators in this field. The bibliography, which contains 298 references to the recent work and brings the literature of the subject down to 1930, is particularly valuable. "Bleivergiftung" may well be recommended as a valuable addition to the library of students of public health.—*L. T. Fairhall.*

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PERSONAL QUALITIES IN ACCIDENT CAUSATION*

E. G. CHAMBERS, M.A.

Investigator for the Industrial Health Research Board, London

THE main purpose of this article is to give some account of the practical and theoretical difficulties encountered in a research into the personal qualities involved in accident causation. This problem is exceedingly important and is, in a sense, one of the main problems of psychology since its solution demands a stringent research into mental integration. The occurrence of an accident, as I shall show later, is not necessarily fortuitous but is often the result of the complicated interaction of various psychologic and physiologic functions; and a research which has as its object the disentanglement of such complex mental situations (using mental in its widest sense) is bound to throw light on the nature of mental life as a whole. The accident situation is one in which everybody finds himself sooner or later, and to which he reacts either by incurring an accident or by avoiding one. If we can find out why some people react in the one way and some in the other, we shall have learned a good deal about the nature of the reactions of mind to its environment.

About the practical importance of this problem to industry there can be little doubt. The cost of accidents is enormous not only in actual money but also in loss of time and efficiency. It is difficult to gauge the full extent of this latter loss, but the financial cost of accidents, as represented by the annual expenditure in insurance premiums, runs into hundreds of thousands of pounds. This insurance, of course, only covers cases where compensation has to be paid, and compensated accidents are actually only 1 or 2 per cent. of the total number of industrial accidents.

An accident is commonly regarded as being due to chance; that is, as an event the causes of which are external

* Received for publication M

tioned for conjugal tuberculosis is an "average" figure, and therefore subject to the usual fallacies of averages. There is, for example, little doubt that the incidence rate among young married couples is higher than 5.6 per cent., and that in middle age it is lower. There are many other factors to be considered, as well as the ages of the infectious and the noninfected spouse. Child bearing, lactation, housing, nutrition, educational and social condition, all have their influence. But when, eventually, all the factors which determine the incidence of conjugal tuberculosis come to be included in one comprehensive equation, we must not be surprised to find that the variable for "silicosis" has dropped out.

Meanwhile, facts are needed. It is in the power of the Tuberculosis Officers working in silicotic areas to pile up these facts, little by little, from the families of silicotic patients who come under their observation.

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 ensheathed 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-

TABLE 6.—SUMMARY OF DETERMINATION OF DUST CONTENT OF ATMOSPHERE OF WORKROOMS AND OTHER PLACES

SAMPLE NO.	PROCESS AND SOURCE OF SAMPLE	LOCAL EXHAUST VENTILATION TO PROCESS OR NOT	OTHER PROCESSES IN SAME ROOM	NO. OF PARTICLES PER C.C.	PERCENTAGE 2μ AND UNDER	PERCENTAGE 7.5μ AND UNDER
1. 1/2	Carding; passageway between cards	Yes	Yes	2,139	81.3	95.1
2. 1/5	Spinning	No	Yes	1,456	95.9	100.0
3. 1/3	Cloth weaving, dry	No	Yes	1,756	84.1	96.7
4. 1/7	Opening	Yes	Yes	2,384	80.9	93.7
5. 1/6	Sieving; shoveling fiber on to sieve	No	Yes	2,829	83.0	97.6
6. 1/8	Plaiting	No	No	1,687	99.0	99.0
7. 1/1	Office	...	...	901	98.8	100.0
8. 2/11	Office	...	...	1,480	98.7	100.0
9. 2/1	Cloth weaving	No	Yes	1,788	88.9	98.6
10. 2/14	Spinning	No	Yes	1,073	96.2	100.0
11. 2/10	Mattress making; filling	Yes	No	985	92.5	98.1
12. 2/8	Mattress making; beating, damp	Yes	No	970	85.9	95.8
13. 2/7	Mattress making; beating, dry	Yes	No	1,629	86.9	96.6
14. 2/9	Mattress making; after filling 2 mattresses	Yes	No	1,204	96.6	99.4
15. 3/1	Band weaving, wet	No	No	808	96.6	100.0
16. 3/4	Opening	Yes	No	2,313	77.7	96.6
17. 3/3	Carding; between cards	Yes	Yes	1,528	82.5	97.4
18. 3/5	Carding; feeding card	Yes	Yes	1,321	82.9	96.4
19. 3/2	Office	...	...	1,049	96.3	100.0
20. 4/1	Mattress making; filling	No	Yes	1,858	60.1	90.1
21. 4/1	Mattress making; filling basket	No	Yes	1,390	....	.....
22. 4/3	Emptying settling chamber	No	Yes	3,401	....	.....
23. 4/4	Same	No	Yes	4,711	....	.....
24. 4/5	Insulating sections; leveling fiber	No	Yes	1,938	....	.....
25. 4/5a	Carding; side of dirty card	Yes	No	1,402	....	.....
26. 4/6	Carding; end of clean card	Yes	No	1,092	....	.....
27. 4/7	Carding; feeding card	Yes	No	1,689	....	.....
28. 4/9	Cloth weaving, very slightly damp	No	Yes	3,033	....	.....
29. 4/10	Cloth weaving, damp	No	Yes	758	....	.....
30. 4/11	Braiding	No	Yes	506	....	.....
31. 4/12	Spinning	No	Yes	954	....	.....
32. 4/13	Office	...	...	689	....	.....
33. 5/1	Band weaving, dry	No	Yes	2,202	....	.....
34. 6/2	Cloth weaving, dry	Yes	No	885	....	.....
35. 6/3	Band weaving, dry	Yes	No	701	....	.....
36. 6/9	Opening	Yes	Yes	643	....	.....
37. 6/6	Carding; feeding card	Yes	Yes	562	....	.....

TABLE 6.—*Continued*

SAMPLE NO.	PROCESS AND SOURCE OF SAMPLE	LOCAL EXHAUST VENTILATION TO PROCESS OR NOT	OTHER PROCESSES IN SAME ROOM	NO. OF PARTICLES PER C.C.	PERCENTAGE 2μ AND UNDER	PERCENTAGE 7.5μ AND UNDER
38. 6/4	Carding; center of room	Yes	Yes	620	....	.....
39. 6/5	Carding; grinding rollers	Yes	Yes	540	....	.....
40. 6/7	Spinning	No	Yes	620	....	.....
41. 6/8	Office	...	...	666	....	.....
42. 7/2	Cloth weaving, dry	Yes	Yes	4,668	....	.....
43. 7/1	Opening; shoveling fiber into box	Yes	Yes	4,880	....	.....
44. 7/3	Opening	Yes	Yes	6,324	....	.....
45. 7/4	Band weaving, wet	No	Yes	5,353	....	.....
46. 7/5	Spinning	No	Yes	6,044	....	.....
47. 7/6	Carding; between cards	Yes	Yes	4,607	....	.....
48. 7/7	Office	...	...	3,413	....	.....
49. 8/2	Cloth weaving, dry	No	Yes	1,287	88.6	94.9
50. 8/3	Band weaving, dry	No	Yes	1,233	78.2	93.7
51. 8/5	Carding	Yes	Yes	1,195	67.2	94.9

tent was determined by means of Owens' jet apparatus. Here also the undetermined effect of dust from neighboring processes must be noted. Nevertheless some information of practical value was obtained.

Reference to Table 6 shows that the counts ranged from 506 to 6,324 particles per cubic centimeter in the air of workrooms, and from 666 to 3,413 per cubic centimeter in the air of offices associated with the factories. In some cases there was a smaller count in a workroom than in an office of the same factory; this was due either to there being much fewer smoke particles in the air of the workroom, or to adherence of the smoke particles to the inorganic dust in the workroom.

The character of the dust in the two cases is, however, entirely different. In the air of the office almost all the particles are black, or brownish, and amorphous, evidently smoke; in the air of the workroom, the majority of the particles are, just as clearly, crystalline particles of asbestos, together with, frequently, some cotton fibers and numbers of black and brownish particles, some adherent to asbestos spicules and some free. Most of the latter are iron containing and are derived from the asbestos, and there are relatively few smoke particles. Isolated golden-brown scales or plaques, also derived from the asbestos, are scattered about the field.

The asbestos fiber first fragments longitudinally, and apparently this process can go on indefinitely, since there is no ultimate fiber comparable to a vegetable fiber such as cotton. The dust collected in the Owens apparatus contains numbers of these very fine fibers—some about 0.5 micron in transverse diameter, many less—which have fragmented transversely. Thus spicules of very diverse length—up to

about 80 microns—but about 0.5 micron broad, are found in numbers on the sample, together with the black, brownish, and yellow particles already mentioned. The great majority of the spicules are found broken down into particles of the order of 2 microns and less, many being about 0.5 micron and more or less rounded. The proportion of elongated to rounded particles seems to vary in the different processes; for example, a greater proportion of elongated particles is found in the air near dry cloth weaving than near a spinning frame. Weaving damp reduces the proportion of elongated particles.

This variety of microscopic spicules, particles, and frequently some cotton fibers, found in the samples of dust collected in the Owens apparatus, results in a tendency to clumping on the cover slip, making counting, especially of the higher concentrations, a matter of extreme difficulty. Occasionally, also, part of the slot of the apparatus choked up with dust. The effect of the clumping is to reduce the counts of all except the low concentrations.

Furthermore, this apparatus is much influenced by local air eddies and currents. It takes a grab sample, which is accurate enough as an indicator of the local conditions at the moment of sampling, but does not give a picture of the average state of the atmosphere during a period of time, as does a gravimetric apparatus. Moreover, with high concentrations of dust, accurate counting becomes impossible.

The apparatus has, however, many advantages, including those of convenience, quickness in operation, and direct examination of the nature of the dust. By serial observations, too, temporary and local large increases in the dust content of the atmosphere—such as that produced by shoveling asbestos fiber into a sack—can be detected and estimated. This is of great importance since short, but repeated, exposure to massive concentrations of dust is agreed to be very harmful. Counts Nos. 5, 22, 23, and 43 in Table 6 are examples. Much useful work can be done by means of serial dust counts in various dusty processes in the same factory, and by comparison of the results.

The counts set out in Table 6 are derived from eight different factories, and it is a matter of interest to consider whether counts from different factories can be directly compared with one another. Strictly they cannot, since the content of smoke particles varies in each group. The difficulty might be obviated by counting only the particles derived from asbestos. The method has been applied successfully to counts of crystalline silica dusts; but it is not so easily applicable to counts of asbestos dust, which contains numbers of black and brownish iron containing particles, many of which are not quickly distinguishable from smoke and tarry particles.

To classify the processes accurately on the basis of amount of dust evolved, it is necessary to take serial samples at intervals corresponding to the details and complexity of the processes. With some straightforward processes, such as spinning, few samples will be necessary; but in others, such as mattress making, mixing, grading and opening—especially with old-fashioned plant—in which the evolution of dust rises and falls rapidly in a comparatively short space of time, many more observations are necessary throughout

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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