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 33, 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.
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> 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 of the discovery of "curious bodies" in the lungs—discussed later—but also, and of more importance generally, be-

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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 the patient's industrial and medical history, no presumptive cause, other than the inhalation of asbestos dust, was found to account for the existence of the fibrosis.

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 of the different processes in relation to the evolution of dust, and the examination, both clinical and radiologic, of workers, was undertaken during the year 1928, commencing

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 great groups—those which 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, if no acute illness has caused a fatal termination,

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.

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	48–53	1.2–9.4	34–44	0.7–6.4	....	0–2.5	...	2–3.8
	Tremolite	57.2	0.9	3.2	22.8	13.4	0.6	0.3	2.4

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 contain less magnesium 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, compiled from various

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 (30).

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 not only 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 of exhaust 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 is general. It increases over the root areas; below 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 according to the state of the lungs before the onset of the fibrosis. Thus,

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 been a source of difficulty in clinical diagnosis is that in which there are extensive bilateral fibrotic changes due 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 again during expiration. This seems to indicate the anchoring of normal apexes by fibrous tissue in the lower portions of the lungs. Some confir-

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, and only 3.4 per

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 irritating effect 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 very poor; in twenty-one (22.1 per cent.), fair; 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." Dr. Tattersall also drew attention to the more or less rounded "whorls" of

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 displacements due to the pull of the fibrosing lesions.
>
> The few tuberculous lesions we have come across have been easily distinguishable.

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 variety of fibrosis, might be added; or it may be that all dust fibrosis will be found to approximate more or less closely one or the other of these two

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 asbestos workers, 2 of whom showed

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 pneumoconiosis, e.g. South African gold miners. . . . . "

Clearly, the maturation of asbestos fibrosis is spread over a 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 363 examined (5.8 per cent.) were so classified:

	No.	*Per Cent.*
Clinical Examinations (363):		
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 this early stage could be detected radiographically, but only 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 unsafe to draw any deductions. For the purposes of this inquiry, therefore, this

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, a female, aged 24, has been employed in asbestos for ten and one-half years—as a card tenter one year,

and as a doffer 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 at the right base. The respiratory murmur is a little weakened generally. No added sounds are detected. A skiagram showed 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.

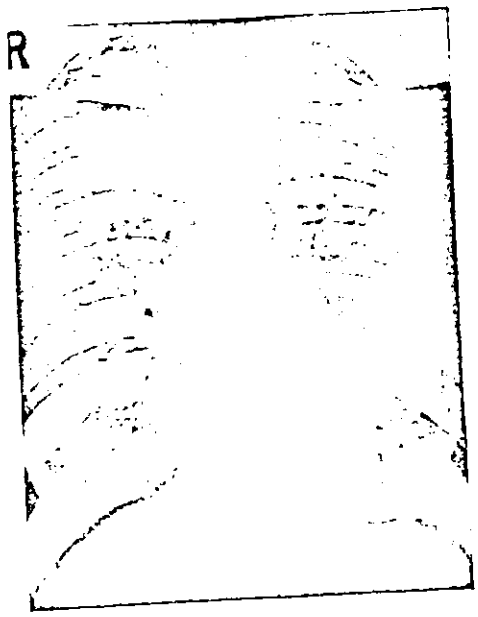

FIG. 1.—Case 4: Moderate fibrosis. Undue degree of striation and fine mottling in central zones of both lungs; calcified glands in roots, with rather coarse striation in upper lobes; ? interlobar pleurisy on right side, between the lower and middle lobes; emphysema at bases.

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 weak, generally, and expiration is prolonged. No added sounds are detected. The skiagram (Fig. 1) shows an undue degree of striation and fine mottling in the central zones of both lungs; calcified glands in the roots, with rather coarse striation in

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

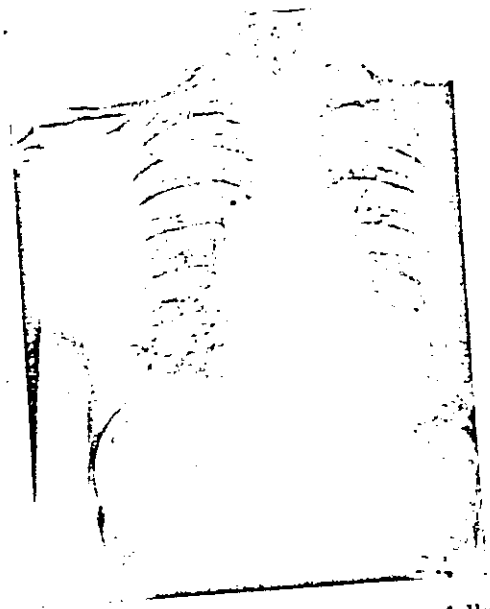

FIG. 2.—Case 6: Moderate, but fully developed, fibrosis. Enlarged glands in both roots; diffuse fine mottling in both lungs, especially the right.

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 râles are noted low down in the right axilla. A skiagram showed diffuse fine striation and fine nodular mottling, with light opacity at the bases. The case was diagnosed as 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.

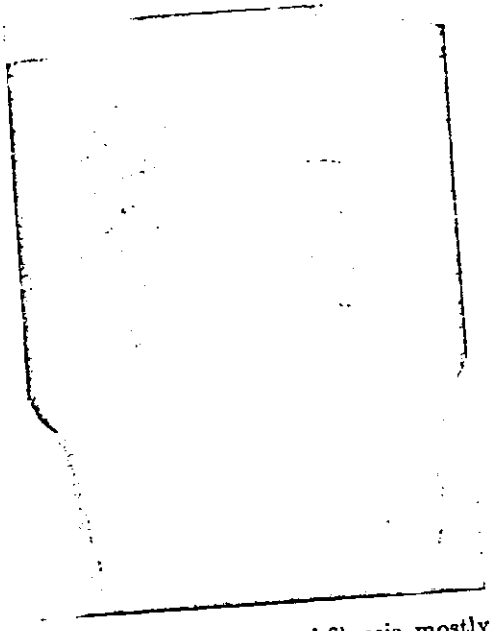

FIG. 3.—Case 7: General fibrosis, mostly linear, but with little mottling of right lung. Old puerile tuberculous scars at apexes.

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 of moderate, but fully developed, fibrosis.

CASE 7.—This woman, aged 48, has been

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 râles are noted at the left base, and a few râles at the right base. A skiagram showed a definite diffuse fibrosis with nodulation. This is a case of fibrosis in the early advanced stage.

CASE 9.—This man, aged 41, has been 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 "boxy," note on percussion, generally, especially over the upper half of the left lung, and over the upper two-thirds of the right lung, behind, and in front. Respiratory murmur is generally weak. Expiration is slightly prolonged, but diminishing toward the bases. Pleural crepitations are noted low down in the right axilla. A skiagram showed a definite fine diffuse fibrosis. Neither lung lights up very well, and throughout both there is a very fine diffuse reticulation and mottling—moderate, but fully developed, fibrosis. The fine diffuse character of the radiologic appearances is particularly noticeable here.

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 disclose nothing of note. His complaints are: shortness of breath on exertion for three months; cough after a few hours' work in dust during the last two or three years, with little or no 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 "boxy" 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. The heart is normal. The skiagram (Fig. 4) shows diffuse striation and fine speckled mottling throughout both lungs—fibrosis, fully developed.

CASE 11.—This worker, a male, aged 40,