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The Hippocratic Fingers of the Asbestotic.

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THAT LEGISLATIVE MEASURES HAVE PROVED GENERALLY EFFECTIVE IN THE CONTROL OF ASBESTOSIS

A THESIS PRESENTED TO THE UNIVERSITY OF GLASGOW FOR THE DEGREE OF DOCTOR OF MEDICINE

by

H. WYERS, M.A., M.B., Ch.B.

1946

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Eorum igitur, qui Fodinis praefecti sunt, nec non & Medicae Artis Professorum, qui in id operam suam locarint, munus erit, Fossorum, quantum licebit, incolumitati prospicere, ac eniti, ut, quando causam occasionalem removere non liceat, Operarii quam minime laedantur.

Ramazzini: De Morbis Artificum.

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CHAPTER 1

INTRODUCTION

The following paper is the result of researches carried out by the writer in answer to a friendly challenge made to him by a prominent industrialist, that, in spite of all the professional skill which had been brought to bear on the asbestos industry, the costs of compensation for asbestosis were higher than ever. A glance at the official figures (20) show this to be only too true, and not only is this so but there has been a progressive numerical increase in the number of persons disabled by the disease.

TABLE 1

YEAR	DEATHS	COMPENSATION	DISABLED	COMPENSATION
1931			1	£36
1932	1	£229	18	£539
1933	2	£162	24	£1136
1934	2	£390	23	£1057
1935	2	£315	43	£1518
1936	3	£748	33	£1219
1937	1	£225	46	£2402
1938	4	£330	54	£2266

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It was immediately obvious that the increase in the cost of living had been reflected in compensation costs, that a numerical increase in cases probably did not represent an upward trend in morbidity rates and that experience of the health of workers convinced one of a more optimistic conclusion than the one implied. The Challenge, if one may call it so, was, however, so serious as to demand a careful inquiry, and measures were adopted to this end. The results were of such an order as would seem wholly to have justified the methods originally laid down by the legislators who inaugurated them. Indeed, any further improvements, short of finding a harmless substitute for asbestos, are likely to come from an intensification of those methods rather than from alternate means.

For the purposes of this investigation facilities were extended to the writer by Dr. Roodhouse Gloyne, Pathologist to the London Chest Hospital and the Silicosis & Asbestosis Board, to make use of clinical and pathological reports. Indeed, it is to the teaching at the bed-side and in the post mortem room of this hospital, together with the valuable discussions with the members of the Board and the Medical Inspectorate, that the writer owes such knowledge as he may have of the elements of diseases of the chest. Thanks are also due in no small measure to Mr. Whittaker, General Manager of the Cape Asbestos Co. and his staff and work-people for much data and helpful co-operation.

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The manufacture of textiles presents problems to the ventilation engineer which are basically different from those which confront the chemical engineer. He cannot, for example, enclose processes with the same exclusiveness as his chemical colleague, for however mechanical the spinning, plaiting or weaving may become, there are physical limitations inherent in asbestos fibre which demand interference in the process by the operator. On the other hand, asbestos dust is not nearly so acutely noxious as many substances used in the chemical industry and it is therefore permissible for standards to be lower. If the ventilation engineer in an asbestos factory can so reduce the concentration of the dust cloud both for operatives and for maintenance personnel that asbestosis cannot develop during a human lifetime, then he has attained almost all that need be asked of him. But what is of hardly less importance is that not only must he invent and apply the best methods of ventilation, he must also see that maintenance is continuously effective.

If the ventilation standards are more difficult of attainment in the asbestos industry, than in certain other trades, the medical precautions must also be more thorough. The chemical engineer may so construct a methyl bromide plant, that, by locating it in the open air and by use of special piping, gasket materials and lubricants, he can prevent all but the most infinitesimal escape and concentration of toxic fumes in the vicinity of the plant, so

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long as it is properly maintained. At such levels he still has safety factors to assist him such as the worker's sense of smell and knowledge of early symptoms of poisoning. Withdrawal from the dangerous area at this stage then effects a cure. Such safety factors do not hold in an asbestos factory. The motes which dance and move in the shaft of sunlight like wisps of pale, thin smoke are seen and recognised by all the workers, but the extent of the danger is dependent upon length of exposure as well as on the concentration of the dust in the air, and therefore largely outside their control. However, once the process of pulmonary fibrosis begins it is irreversible, indeed progressive. The warning signals of asbestosis are not only of danger to come, but of danger actually present. If, however, the early case is removed from the hazard, progress of the disease is much slower. It is of the first importance, therefore, that only those with healthy lungs and hearts should be submitted to the risk, that they should be kept under observation and removed from danger at the first signs of fibrosis.

It may well be asked what the social values of asbestos are which justify a continuance of manufacture. No one would suggest the abolition of coal mining or deep sea fishing on account of their dangers, because they are vital to the welfare of the nation. But before the uses of asbestos are considered it would be well to mention the psychological fact that physical injuries do not impress

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the workers or affect their morale to anything like the same degree as the more mysterious medical diseases of industry. The effects of a railway accident will excite sympathy, but the delayed effects of dimethyl sulphate among half a dozen men will create fear in many more workers, not to mention puzzled enquiries during the night from as many hospitals.

The Romans knew asbestos under the name of amianthus and obtained it from the Italian Alps and the Ural. It is said to have been woven into cremation cloths for the well-to-do and to have been used as lamp wicks by the Vestal Virgins. Another fibrous mineral resembling Italian amianthus was spun and woven into cloth in Siberia in the 13th century. A factory for the manufacture of asbestos articles was opened in Russia in 1760. In 1876 modern industry re-discovered asbestos and extensive fields were soon being exploited.

Textiles fall naturally into first place among the articles from which asbestos can be made. These are worked into safety curtains, fire-fighting suits and mattresses for the conservation of heat in boilers. Yarn can be plaited into rope which may be fortified by metallic wire. It is in the opening and carding of crude asbestos and in the spinning and weaving of textiles that most cases of pulmonary fibrosis occur. A mixture of about 10% asbestos with magnesia and other materials is used for lagging steam pipes and boilers. Pipes made of

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short asbestos fibres bonded with a watery solution of sodium silicate are cut into sections for fitting to steam pipes. The cutting of asbestos pipes etc. by high speed band saws sets free a fine dust containing a proportion of asbestos in a fine state of subdivision more likely to reach the alveoli than to stuff up the bronchioles and therefore less dangerous. A similar mixture is used to line pans in the chemical industry to prevent erosion by acids. Electrodes are coated with an asbestos composition or yarn which also enters into the composition of paints, roofing tiles and filter pads and asbestos itself may be put to its ancient use as a wick, but in petrol lighters. Bulkheads of ships are coated with asbestos fibre to render them fire-proof and to prevent condensation of moisture. The great development of the motor car industry has been, in some part, due to the manufacture of moulded clutch rings and brake linings of asbestos, with urea-formaldehyde resin as a bond. Mixed with cotton, asbestos has been used as filtering material in masks for protection against war gases. The resistance of blue asbestos to sea-water provides many uses for it on piers and ships. Indeed, there is scarcely any industry which does not depend to a more or less degree upon asbestos. To take this valuable mineral away from a nation, therefore, would put back that nation's industrial capacity by half a century, a blow which this country, at any rate, could not survive.

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CHAPTER 2

THE SCHEME

In 1906 the Departmental Committee on compensation for Industrial Diseases (2) heard evidence from various authorities arising out of the inclusion of certain industrial diseases in the Third Schedule to the Workmen's Compensation Act, 1906. In addition to those diseases named, sec. 8 of the Act empowered the Secretary of State to add other industrial diseases from time to time and it was the duty of the Committee to consider what further steps could be taken and to make recommendations. The new Act had also overcome certain serious difficulties of workmen who attempted to prove that the occupational diseases from which they suffered were accidents whose onset could be determined by a point in time like a physical accident and that the employer had been notified of that occurrence within the statutory period of 6 months.

At that enquiry Murray cited the case of an asbestos worker who had died in the Charing Cross Hospital of "typical fibroid phthisis". This man, a card-room worker who had died at the age of 34 in 1900, had stated that nine of his mates in the same card-room had died round about the age of 30. Whilst agreeing that fibroid phthisis was a clinical entity and specific to certain occupations, the Committee felt they could not recommend its inclusion in the list of diseases compensatable under

the Third Schedule owing to certain difficulties, chiefly of certification, and suggested trade insurance schemes as alternatives. Perhaps the prevalent doctrine of the inocuous character of the combined silicates tended subsequently to deter medical investigators and so retarded legislation. Cases were reported, however, notably by Fahr in 1914 (5) and Cooke in 1924 (4); but it was Seiler's case in 1928 (3) which seemed to establish an unequivocal relationship between the inhalation of asbestos dust and pulmonary fibrosis.

It was Seiler's case, also, which precipitated the Home Office enquiry of Merewether & Price in 1928 (6). They made clinical examinations of 363 workers, approximately 16.5% of the population at risk and of these 133 were examined radiologically. The result of their report was a certificate by the Secretary of State under sec. 79 of the Factory & Workshops Act of 1901 to the effect that "the manipulation of asbestos and the manufacture or repair of articles composed wholly or partly of asbestos and processes incidental thereto are dangerous". In 1930 the Workmen's Compensation (Silicosis & Asbestosis) Act (8) extended the provisions of sec. 47 of the Workmen's Compensation Act 1925 (7) "to industries and processes involving exposure to asbestos dust" and gave power to the Secretary of State to "make a general scheme applicable to all industries and processes ..... to which compensation schemes apply, for the purpose of co-ordinating

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the medical arrangements in connection with those compensation schemes". Provision was made by this general scheme for the establishment of a medical expenses fund, to be administered by trustees appointed by the Secretary of State (sec. 1). Fees in respect of examinations and certificates were to be paid into the fund by or on behalf of employers and workmen. It was further provided that employers should not employ in any industry or process a workman who had been suspended in pursuance of the Scheme or had refused or neglected to submit himself to examination. Employers were also to give notice to the medical officer or medical board of the commencement of any process involving exposure to silica or asbestos dust. Different provisions as respecting different industries or processes might become the subject of regulations (sec. 2).

Before such a scheme could be constructed, however, many problems of engineering and ventilation had to be considered. Whilst it was true that sec. 74 of the Factory & Workshops Act 1901 had contained provisions for the suppression of dust generally and that means had already been adopted to this end in card-rooms, the new Act seemed to envisage a much more thorough control of the whole problem of dust removal. Often a dusty process would be carried out in the same room as one which was relatively safe. It was necessary to devise means of extracting dust from the large surfaces of textiles.

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Spinning frames with hundreds of bobbins presented as many sources of dust flung into the atmosphere at high speeds. To consider such matters, a Conference of Employers & Inspectors was called and their report became available in 1931 (9). The Secretary of State now had not only the power, but the technical information to enable him to devise the schemes which are now known as the Silicosis & Asbestos (Medical Arrangements) Scheme 1931 (10), The Asbestos Industry (Asbestosis) Scheme 1931 (11) and the Asbestos Industry Regulations 1931 (12).

The first Scheme is concerned with the constitution of the Medical Board (sec. 3), post mortem examinations (sec. 7), applications to the Board for certificates of disablement (sec. 8) and procedure to be adopted. Sec. 12 deals with the initial examination of all entrants to a scheduled process within two months of employment and the physical standards to be used. These are:-

(1) The chest must be at least of average development and the respiratory passages must be free from obstruction;

(2) There must be no signs of disease of the lungs or heart: and

(3) There must be no tuberculosis of any region.

Besides new entrants to the industry, certain others must also submit to this examination; these persons are those who have been transferred from other work carried on under the same employer but not subject to a Compensation Scheme, provided such persons have not been medically examined under this scheme or any Compensation Scheme

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during the preceding twelve months. Thus, a person engaged in bonding asbestos fibre with sodium silicate solution into slabs is not liable for examination by the Board but yet is exposed in some degree to the inhalation of asbestos dust. If such a person is transferred to the Spinning Department she is treated as a new entrant to the industry. She may, on transfer, be found to be suffering from asbestosis. For such cases, asbestosis is specifically mentioned as a reason for suspension at this examination, as well as tuberculosis; but in such circumstances the other conditions as to physical fitness do not apply. The examinations may be carried out by a "duly qualified medical practitioner especially appointed by the Secretary of State for the purpose", but only the Medical Board can suspend the person. Such a specially appointed medical practitioner is usually the local tuberculosis officer for the area in which the factory is situated.

Sec. 13 prescribes periodic medical examinations and powers of suspension by one or more members of the Medical Board "at the prescribed intervals". As will later appear, the prescribed intervals are of the greatest value for statistical purposes, besides their prime objects, because they assist in making an estimate of the population at risk. The direction of the Secretary of State is as follows:-

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"In pursuance of paragraph 13 (1) of the Silicosis & Asbestosis (Medical Arrangements) Scheme 1931, I hereby prescribe that workmen employed in the industries and processes specified in the First Schedule to the Scheme shall be examined by one or more members of the Medical Board at the following intervals, namely:-

Asbestos Industry:- All persons employed in the processes specified in the said Schedule shall be examined once a year.

Provided that the Medical Board may require the re-examination at shorter intervals of individual workers in suspected cases in any of the scheduled processes.

For the purpose of these examinations, a worker who is only occasionally employed in a process for which the prescribed interval is eighteen months may, if the Medical Board so directs, be examined once every three years."

In practice, the number of First Schedule workers absolved from yearly examinations is minute and not of such an order as to cause any statistical error in calculations of population.

Sec. 14 gives the Board authority to obtain a radiologist's report in any case where they think it necessary. The Medical Expenses Fund & Fees are dealt with in sections 17 to 21. Employers' duties with

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respect to notification to the Board of First Schedule processes, initial examinations of workers, periodic medical examinations, the engagement of workers suspended from First Schedule processes, are set out in sections 21 and 22. A register is to be supplied to each worker containing certain details of his occupational history and of his examinations by the Board.

The First Schedule appears at the end of the Scheme and delineates the processes to which the special regulations apply. Owing to their very great importance, they are quoted here verbatim.

"The following processes in the Asbestos Industry except where such processes are carried on occasionally only:-

(1) breaking, crushing, disintegrating, opening or grinding of asbestos, and the mixing or sieving of asbestos, or any admixture of asbestos, and all processes involving manipulation of asbestos incidental thereto;

(2) all processes in the manufacture of asbestos textiles, including preparatory and finishing processes,

(3) the making of mattresses composed wholly or partly of asbestos, and processes incidental thereto."

The Second Schedule prescribes the forms of the various certificates and the Third Schedule lays down conditions relating to accommodation for medical examinations at the workers' place of employment.

The Asbestos Industry (Asbestosis) Scheme is a scheme designed to set up machinery for the award of compensation

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rather than one aimed at the prevention of asbestosis. On the other hand, the Asbestos Industry Regulations lay down very specific instructions as to exhaust ventilation, the prevention of dust gaining access to the atmosphere of a workroom, the damping of floors and benches where insulating mattresses are made and repaired (Reg. 3), the cleanliness of floors, benches and plant and storage of asbestos (Reg. 6), lighting, containers, ventilating plant, breathing apparatus and the employment of young persons. At least two cases of asbestosis and tuberculosis, following brief exposures to asbestos dust, are known to have occurred in adolescent boys. It is to prevent such tragic occurrences that Reg. 12 provides that no young person shall be employed in certain processes such as the manufacture of insulating mattresses, the hand mixing of asbestos, cleaning of sacks, in dust exhaust apparatus or in the cleaning of cards. Certain duties are placed upon employed persons by Regulations 13 to 17.

The Scheme might be criticised as being too restrictive in that a workman may contract asbestosis and in failing to qualify legally he may lose compensation. A strict interpretation, for example, is placed upon the wording of the Third Schedule in accordance with the decision in Doncaster v. Ludlow (1929) (13) to the effect that technical words used in the schemes should be interpreted in the sense in which they are used in the trades and not in the popular sense. For example, the word "earthenware" does not

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include "Jet & Rockingham" ware, a special branch of the pottery industry to which the term, as used in the trade, is not applied. Or again, a person who left the industry prior to 1st May 1931 has no claim to compensation. These restrictions, however, permit of the placing of liability on the individual employer or employers who, knowing their liabilities, are able to make sufficient provision to meet claims by insurance and so afford greater security to the workman and his dependents.

The Board consists of specially qualified full-time medical officers under the direction of a Chief Medical Officer. The functions of the Chief Medical Officer are to ensure uniformity, to make the final decision where a panel fails to agree and to undertake the ordinary work of the medical officers. He has his headquarters at Sheffield, the centre of the Metal Grinding and Refractories industries. There are also five panels, each of two medical officers disposed throughout the country so as to cover all those areas where silicosis and asbestosis are likely to occur. One is at Sheffield (Metal Grinding Industry), a second is at Stoke-on-Trent (Pottery Industry), a third is at Manchester (Asbestos Industry), and two each are at Cardiff and Swansea (Mining). Scotland is covered by the Manchester panel and England and Wales are split into appropriate territories under the jurisdiction of the other panels. The Coal Mining Industry (Pneumoconiosis) Compensation Scheme, 1943, accounts for the increase in

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medical personnel in South Wales where, in addition to silicosis, dust reticulation has also to be considered. Suspensions from work cannot be decided unless by a panel of two medical officers.

It is of importance to realise that once the Board has issued a certificate it is functus officio. It cannot reconsider or amend a certificate, nor is there any appeal from it. If a workman applies through a County Court for review of a certificate to which a time limit has been attached, the Medical Board are substituted for the Medical Referee. This may appear arbitrary, but is not so in practice. Whilst the members of the Medical Board are now appointed by and act under the instructions of the Ministry of National Insurance, they are not civil servants. They act judicially and without regard to the consequences of certification so far as compensation may be affected. Two medical officers consider each case and may refer it to the Chief Medical Officer. Most applications pass through trades unions and employers' federations, organisations with vast experience of workmen's compensation, and all agree on the smooth working of the Scheme. Moreover, there is nothing to prevent a fresh application being made by a workman at a later date, if he thinks that since his previous application he may have developed the disease. The provisions for radiological and post mortem examinations are additional safeguards.

There is a mistaken notion current that the Scheme was

intended to cover only such cases of tuberculosis as could be shown to follow silicosis or asbestosis and that where there is extensive tuberculosis and only slight silicosis or asbestosis the latter should be regarded as unrelated and fortuitous. On the contrary, the two diseases are only required to be present together in order to come within the Scheme. Tuberculosis is interpreted as active tuberculosis.

Where a workman suffers from asbestosis and also from an unrelated disease, the Board must assess the disability in terms of asbestosis only, although, in practice, a wide interpretation is given, that is to say, in favour of the workman.

Death is regarded as a novus actus interveniens (even if a workman has been awarded a certificate of disablement during life) and a fresh certificate obtained from the Board. This can ordinarily only be issued after post mortem examination. If the deceased had been in receipt of weekly payments under the Scheme, the Board have discretionary powers to dispense with a post mortem examination, but never actually waive their right to such an investigation unless exhumation of the body would be necessary. A pathologist is attached to the Board but it has been found more equitable for coroners to appoint their own pathologists to carry out the post mortems. By arrangement, the lungs are sent to the Board's Pathologist for histological examination. Thus the margin of error or

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the possibility of suspicion that one or other of the contending parties might be favoured, is reduced to a minimum. The post mortem is directed to be made, if possible, in the presence of the Medical Board.

Such, then, are the general terms of the Scheme and their accepted interpretation (14). The statistical survey which follows has been constructed on the basis of entities defined according to the Scheme and this interpretation.

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CHAPTER 3

RESULTS OF THE SCHEME

Merewether, in the Chief Inspector of Factories Report (1945) (21) states: "What we are most anxious to discover is a yardstick by which to test the efficacy of preventive measures in diminishing the risk of contracting silicosis and asbestosis". It is the writer's submission that this yardstick is to hand in the published reports of Periodic Medical Examinations made by the Board.

Actual figures for the population at risk are not available, nor would such figures be of much assistance in attempting to assess the effects of legislation unless morbidity and mortality data were also available for the same group. All workers inhaling asbestos dust are subject to risk, but only according to concentration of dust and length of exposure; for whereas a high concentration may evoke the disease in five years, a low concentration may require 50 years, by which time the person could have succombed to a cause quite unrelated to his occupation. To define the term "risk" is therefore a primary necessity and for present purposes this has been taken to mean the risk involved in First Schedule occupations. Secondly, cases of asbestosis are taken to mean those who have been discovered by the Board in the course of their periodic medical examinations. Incidence rates have been calculated as ratios between cases of asbestosis as so defined to numbers

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of workers in First Schedule occupations, annual figures of periodic medical examinations having been accepted in each case. Three objections might be made to this course.

(1) During the war, much use was made of asbestos, such as the fire-proofing of decks and bulkheads by blowing the fibre on to a prepared surface, as, in the opinion of many, to constitute a risk equally as dangerous as a First Schedule occupation and yet not included in the Scheme.

(2) The labour turnover is disproportionate to the population at risk, so that whilst it may remain numerically constant it does not retain an identity of personnel. As a corollary to this objection, it might be pointed out that a yearly sample of the population is but a crude average.

(3) Cases of asbestosis have become disseminated in the general population, treated as bronchitis, broncho-pneumonia or tuberculosis, and after death, certified as such.

These objections will be dealt with seriatim.

1. The first objection can be met largely by the factor of maturation. Most cases occur after an exposure of five to eight years so that it is only now (1946) that the results of exposure commencing in 1939 are likely to become manifest. To add the numbers of these new workmen, even if known, workmen who have not had the benefit of preventive measures, to the numbers of First Schedule workers, would simply vitiate results.

2. Whilst it is true that the population at risk in First Schedule occupations is not an absolute entity with fixed

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components, nevertheless its components vary at a constant rate, with the exception of the period succeeding the adoption of the Essential Work Order at some date subsequent to April 1941. Using the method of calculation recommended in "Sickness Absence & Labour Wastage" by Major Greenwood and M. Smith (16), in one of the largest asbestos undertakings the labour wastage was found to vary only between 40.8% and 43.5%. So accurate are these figures, that labour requirements for the ensuing year can be planned to a nice degree of accuracy. Further, it compares very favourably with the figures presented in the M.R.C. Emergency Report No.2 "Hours of Work, Lost Time & Labour Wastage" 1942, p.23, Comparison of Ten Factories (17), where the average labour wastage for a year is 42.7%. It is generally conceded that labour wastage is higher where female labour is employed and in one factory it was as high as 84.2%. Considering that the asbestos industry employs a very large proportion of its workers of the female sex, the figure of just over 40% would therefore appear to be small. The authors of the M.R.C. Industrial Health Research Board Report No.85 "The Recording of Sickness absence in Industry" p.10 (18) state that in order to establish the average number of workers employed in any period, it is adequate to add the numbers on the pay-roll at the beginning and end of the period and divide by 2, provided that the population is reasonably stable. This condition would seem to be fulfilled in the present case.

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The yearly sample would only be a crude figure if taken alone, but as it is one of a series of 14 years, and as each annual figure is derived from a number of small samples spread over each year, there is such a period of time and such frequent sampling as to confer a more exact connotation. It is this lapse of time which elevates the investigation into the more exact sphere of calculations for the incidence of the exanthemata in a large and stable population. The retardation in rate of change of labour turnover consequent on the Essential Work Order (19) was a sudden change which would alter a graphic curve, but having once stabilised the population the factors will remain constant until the Order is relaxed or rescinded. Also, labour turnover was not abolished by the Order. To quote again the Emergency Report No.2 of the M.R.C. (v.s.), p.24 "Although the results show that the Essential Work Order reduced labour wastage, there were indications that the workers in some factories were finding ways of evading this Order. Thus, in Factory E, the number discharged as medically unfit before and after the Order came into operation was:-

TABLE No.2

Average number employed	Before		After			
	February	March	April	May	June	July
18,364	0	0	4	65	71	95

There is evidence that these workers obtained medical

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certificates from their doctors to the effect that "their present occupation was detrimental, etc." or "the worker is unfit to continue his present job". A large proportion of these workers were new entrants. The last sentence is significant for the present discussion for it indicates that labour wastage subsequent to 1941 has been, at least in part, due to cases with brief exposure to dust. The effects of the Order were therefor to stabilise the population by a reduction, but not abolition of labour turnover and by that degree diminished the error for present purposes. Finally, the present object is not so much to establish an absolute figure of incidence as to describe a curve for trend, a curve which should be accurate even supposing the rates of incidence are not exact, provided the factors determining those rates remain constant.

3. A true estimate of the number of cases would take into account those also which have not been certified by the Board. A proportion of labour wastage from the industry will inevitably include some who remain well for some years but have had sufficient exposure as to cause the disease after working at a dissimilar occupation in an area remote from the factory where the exposure took place. Such a drift of labour is known to occur among early silicotics from the South Wales coalfields to the lighter portering jobs to be found in Midland mental hospitals. Lack of breathing space has a centrifugal effect. Incredible, as

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it may seem, workers themselves are sometimes ignorant of any casual connection between asbestos and lung disease, although it should be added that such people have always left the industry at some distant date. Two women, sisters-in-law, had worked in the Spinning Department of an asbestos factory and 8 years after leaving the industry presented themselves for re-employment. The writer found them both to be suffering from asbestosis and neither woman seemed in the slightest degree aware of the nature of her illness. No doubt, in some cases, the patient and his relatives wilfully suppress information of a vital kind from the attending practitioner, especially such as left the industry prior to 1931, and are therefore not entitled to compensation under the Act. More than once an old employee has indulged in some grim humour at the writer's expense to the effect that "You'll never perform a post mortem on me; when I feel I've got the "dust" I shall go to some country place where the doctor doesn't know about asbestosis and he'll think I've got bronchitis or pneumonia". Many personal reasons account for this reticence, such as a disinclination on the part of relatives to become involved in a Coroner's Court proceedings and the attendant publicity, the knowledge that no cure exists, and the very human habit of suppressing painful impressions from the conscious memory. It may also happen that medical men neglect to obtain a full history and arrive at a wrong diagnosis so to speak

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by default. This is most likely to occur in those large areas of the country where an asbestos hazard does not occur among the working population. The intrinsic difficulties in diagnosing a case are an additional factor. In assessing the effects upon trend, however, it should be borne in mind that the majority of such cases as escaped recognition did so in the early part of the last decade and are increasingly less likely to do so with better diagnosis and greater awareness on the part of doctor and patient alike of the etiological relationship. Thus, any improvement in trend would only be accentuated if all the older cases were known. Credit attaches particularly to Registrars of Deaths in stopping certificates where the occupational history suggests that as a cause, a practice which is increasing. The vigilance of a Coroner is illustrated by the following case which he referred to competent authority for post mortem:-

Case 1. W.T.E. Male. Age 47. Worked as a tinsmith in asbestos factories on and off from 1916 to 1936. Lived in London until 3 weeks before his death on 15/11/40, when he removed to a Hertfordshire village.

P.M.: Body much wasted. Moderately advanced asbestosis. Asbestosis bodies +. Congestion right base. Several pints of fluid in abdomen. Columnar celled carcinoma of colon. Abdominal viscera matted together. Complete symphysis pleurae on right side due to secondary growths.

Diagnosis: Asbestosis and Cancer of Colon.

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It is fortunate that figures for First Schedule workers can be approximately ascertained from the numbers of periodic medical examinations made in accordance with paragraph 13 of the Silicosis & Asbestosis (Medical Arrangements) Scheme 1931, formerly published by the Home Office. As each worker must be examined annually, it follows that the number of periodic medical examinations recorded each year is a sample number of First Schedule workers at risk for that year, particularly as the sample is not taken at a point in time but is spread over the year. Most of the examinations concern old employees; for others it is their first examination subsequent to the initial medical examination made within two months of engagement in accordance with paragraph 12 of the same Regulations. The writer is indebted to the Ministry of National Insurance for figures relating to the war years of 1939 to 1944 and permission to use them is confined entirely to this paper for the present.

Consideration of Graph 1 indicates a fairly constant increase in the intake of new entrants to the industry until the inception of the Essential Work Order, when there was a rapid fall lasting four years. On the other hand, periodic medical examinations show a similarly fairly constant increase in numbers to a maximum in 1942 before there was a falling off, which even then was neither so regular nor so acute as the curve for initial examinations. This observation seems to confirm the statement made above,

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that the labour turnover rate was less after the inception of the Essential Work Order than before: new entrants became far fewer and wastage from leavers was restricted, producing a more stable population with a larger percentage exposed for longer periods.

Graph 2 indicates a steady increase in the percentage of suspensions until 1938 when the first effects of labour shortage began to be felt and applicants of lower medical standards presented themselves for initial examinations. This lowered physique was apparent as a constant factor for three years of the war until 1944 when there was a sharp rise probably due to a severe combing out of industry for military needs. Nothing could emphasise more the difficulties of personnel managers in securing labour for an industry on which are imposed stringent statutory requirements of health. Even under the direst need of war-time labour shortage the Board were unable to relax these requirements. Nor, perhaps, is it desirable they should be relaxed: the only real solution is to protect the worker by such efficient engineering as is outside his control. Whether people should be killed in battle because of the lack of efficient equipment or whether lives should be lost in producing that equipment only Government can decide. Generally, the tendency is to protect the civilian population and balance decisions with that object in view.

Graph 3 indicates a commendable fall in the morbidity

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rate as calculated under the conditions set out above. So effective, indeed, have the new conditions been, that only three cases within the writer's knowledge developed the disease as a result of employment subsequent to 1931. The explanation as to why cases continue to occur among the older class of employees has a twofold origin: first, there is the factor of maturation and secondly, the factor of chronicity. The first peak in the incidence of morbidity curve coincides with the year 1932 and is mainly due to the bad conditions of previous years, but also to the rush of applications consequent upon the first year's working of the Board. Having "cleared the books", 1933 showed a marked fall which is not maintained for the two succeeding years because of the maturation factor. No cases were discovered at periodic medical examinations in 1940, but 1941 provided a sharp, if small, rise over the preceding four years and the zero figure has not since been maintained. For present purposes, the fact that of four applications for disablement certificates, three were found to have the disease in 1940, has been neglected. Such applications arise from persons who may or may not be engaged in the industry at the time of the application and calculations based on such figures would require a knowledge of the total numbers of persons who have been at risk. For the same reason, mortality rates have not been calculated.

The increased incidence in 1940 may be explained by the severity of the first war winter of 1939-1940, the effect of

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which was to increase common colds among asbestotics, so hastening maturation. The war factors of fatigue and increased incidence of tuberculosis probably accounts for the continued appearance of asbestosis since 1940.

Maturation may be defined as the silent progress of the disease from the beginning of fibrosis to its earliest clinical and radiological manifestations. Whether continued exposure to the dust is necessary during this period is open to discussion; it is the writer's opinion that once the process of fibrosis has begun it continues, probably at a slower rate than when fresh fibres continue to push through at other points along the bronchiolar walls, but nevertheless its progress cannot be arrested. When fibrosis commences in the human being cannot of course, be ascertained. In one experiment by L.U. Gardner and D.E. Cummings (23) on guinea pigs which had been exposed to chrysotile dust for 70 days, a moderate degree of fibrosis was found in 15 months; a subsequent experiment seemed to indicate regression in the absence of dust exposure. They also found that the interjection of the infection factor causes fibrosis to become progressive and they were of the opinion that this may explain the belief that asbestosis is a progressive disease. Fibrosis in the human being has been detected at post mortem after as little as 6 months exposure. It may be added that, in human beings, asbestosis tends mainly to reveal itself after winter colds. E.R.A. Merewether thought that approximately 7 years must

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elapse between the commencement of exposure and the production of a serious degree of asbestosis.

In the factory where the writer is engaged, all employees are examined clinically and radiologically once a year and oftener if necessary; yet fresh cases continue to appear both among those at risk and among those no longer exposed to the hazard. One man (R.W.) who died on 29/3/45 had not been exposed to asbestos dust since 1928 and did not exhibit signs of the disease until 27/3/44. A woman (A.M.) worked as a spinner from 15/1/26 to 23/4/30. On re-employment on 10/11/39 she appeared to have a healthy chest, but on 31/12/41 she was found to be suffering from a well defined degree of asbestosis. Indeed, it sometimes appears as if re-employment lights up a quiescent or slowly progressive lesion. A male employee (J.M.) worked in the Sectional Department from 1926 to 1938 and afterwards as a timekeeper. He was passed Grade 1 for military service in February 1942. On 2/12/43 he was found to be suffering from advanced asbestosis with marked clinical and radiological signs. Another male (W.P.), a myxoedematous patient under treatment with thyroid gland, worked in the Opening Room from 1931 to 1939 when he was transferred to the Impregnating Department where the dust is bonded and reputedly inert. Yet he exhibited a slight degree of the disease on 26/4/45. A man (C.J.S.) worked at various dusty jobs from 1910 to 1930. Subsequently he worked in the Stores where there was practically no exposure to

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asbestos dust. By 16/9/41 signs of early asbestosis were apparent. Another male employee (W.E.W.) worked in the Millboard Department from 1919 to 1930. After that, for all practical purposes, there was no exposure to asbestos dust but asbestosis and pulmonary cancer were diagnosed tentatively on 14/2/44. A woman (R.E.W.) worked in the Disintegrating Department from 1926 to 1932. She left to marry and remained in good health until 1942 when she began to suffer from dry cough and dyspnoea. On 12/7/45 there were clear indications of the disease. Thus 7 cases occurred in which there had been intervals of some years between the exposure and the appearance of the disease.

In these cases there seems good reason to believe that asbestosis is progressive. If this characteristic is due to the interjection of infection, then the greater is the necessity to advise asbestotics to avoid infection so far as that is possible. To avoid infection altogether is almost impossible. Therefore, to all intents and purposes, the disease should be regarded as progressive and the patient withdrawn from further exposure at as early a stage in the disease as possible. The utmost that can be done is to prolong life and if this can be done to the extent that old age or an intercurrent disease overtakes the person, then one might, in that sense, speak of a cure.

Chronicity is the second factor causing the appearance of apparently fresh cases. Evidence of this factor can be found in the changing characters of the disease itself, but

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consideration of these phenomena must be deferred for the discussion on clinical signs. Further evidences are to be found in Graph 4 which gives curves of rates of exposure and of life where the term "life" is taken to mean that period existing between first employment and death. It is at once apparent that the curves closely resemble each other. There appears to be a correlation between the duration of exposure and the life, the number of cases being similar. In the ascending series there are three peaks (numbered 1, 2 and 3) and in the descending series there are two peaks for the life incidence and four for the exposure incidence (numbered 4 and 5, and 4 to 7 respectively). The peaks which correspond numerically also correspond in amplitude. The differences in years between the respective peaks may be expressed thus:-

9 - 3 = 6 years
13 - 5 = 8 "
16 - 8 = 8 "
19 - 11 = 8 "
25 - 15 = 10 "

That is to say, the base of the life curve is wider than the base of the exposure curve. Therefore, the longer the exposure, the longer the life. This apparent absurdity becomes more intelligible if it is expressed as the longer the exposure, the more protracted or chronic is the disease. There can be only one explanation for duration of exposure being associated with longevity, that is to say, lower

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concentrations of the noxious agent. And this is so. Most of the fatalities thus dealt with graphically survived to work for varying periods subsequent to the 1931 Regulations. It may be argued against this that to reverse the life curve would also produce a correlation with the exposure curve, that is to say, the longer the exposure the shorter the life. This cannot be true however: first, because the correlation is only one of enumeration of the peaks and not of the general form, but also because a point is reached where exposure to dust exceeds the life, a true reductio ad absurdum.

The curves are based upon the post mortem findings in 98 fatal cases of asbestosis alone or complicated by other diseases such as tuberculosis and carcinoma. Further observations on the curves reveal that most fatal cases have had exposure of eight years and die 16 years after first employment, although when these figures are compared with those in the table of averages (Table 3) it is found that the average length of exposure was 10.4 years and the average life coincided with the graphic figure, viz. 15.7 years. The other big discrepancy is shown in the duration of the disease. Whilst the curve indicates that most die after the disease has been in progress two years, the average duration of the disease is 6.3 years. Once again, chronicity seems to be the likely explanation. Some survive much longer exposures than others and whereas most cases have been acute (2 years), some have lived with the disease in a chronic form for many years.

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That Legislative Measures Have Proved Generally Effective in the Control of Asbestosis (1946)
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TABLE No. 3

Figures represent years. Compiled from writer's Series

	EXPOSURE			POST EMPLOYMENT SURVIVAL			DURATION OF DISEASE			LIFE			AGE AT DEATH			MATURATION		
	O x	O +	Average	O x	O +	Average	O x	O +	Average	O x	O +	Average	O x	O +	Average	O x	O +	Average
Asbestosis alone	13.7	7.4	9.4	2.8	9.7	7.4	5.9	6.1	6.0	16.0	17.2	16.4	49.4	36.8	42.3	8.8	9.2	9.1
Asbestosis with Tuberculosis	10.5	6.4	7.5	2.5	9.0	7.8	5.8	6.0	5.9	12.2	15.5	14.4	34.4	34.9	34.9	8.1	8.7	8.5
Asbestosis with Cancer	25.4	5.9	17.6	1.5	13.5	6.3	7.4	10.0 ✓	7.9	26.7	19.2	24.0	52.1	46.1	49.8	19.0	10.7	14.3
All cases minus Tuberculosis	18.5	7.1	11.4	2.3	10.3	7.1	6.5	6.4	6.5	20.7	17.5	18:6	50.5	38.5	43.6	11.9	9.5	10.3
All cases	16.2	7.3	10.4	2.3	9.8	7.1	6.4	6.3	6.3	17.9	16.8	15.7	46.5	37.5	40.8	10.1	9.3	9.8

✓ Figures available for 2 cases only.

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In the Chief Inspector of Factories' Report for 1945 (figures relate to 1944) Merewether does not segregate cancer cases for the obvious reason that a causal relationship between asbestosis and cancer cannot be established. His figures for asbestosis alone should therefore be compared with the group entitled "all cases minus tuberculosis" in the present series. In that event, there is still general agreement, the writer's figures being some three years less for average ages at death. Notwithstanding, the inferences are the same, viz., asbestosis appears to kill at an earlier age than silicosis, after a briefer employment history and irrespective of whether tuberculosis is present or not. Merewether's figures are given below in Table No.4 for comparison.

TABLE No. 4

Disease	Number of Deaths	Average Age at Death	Duration of Employment in years		
			Longest	Shortest	Average
Silicosis	844	57.2	62.0	1.5	34.0
Silicosis with Tuberculosis	906	53.3	67.0	0.7	31.3
Asbestosis	125	46.6	48.0	0.5	15.1
Asbestosis with Tuberculosis	65	38.8	29.0	0.8	10.4

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Any discussion of this character which only takes into account the average age at death is misleading as referecne to Graph No.5 will indicate. These average figures do indeed show asbestosis to be a disease which proves fatal early in life, but they seem to indicate also that most die a decade later than the facts justify. It is in the first half of the fourth decade of life in which asbestosis takes its greatest toll and is therefore so much the more a social and personal disaster. Other points of social and economic importance are to be found in Table No.3 where it is noteworthy that post employment survival is markedly greater in females than in males, indeed 7.5 years longer. Whilst many factors undoubtedly contribute to this result, it is mainly due to the tendency of the woman to retreat to the protection of home when she begins to feel unwell and also the economic necessity for the man to continue at work even when he is unfit. Perhaps, also, each experiences a greater buoyancy of morale, the woman in the home environment and the man among his fellows, and certainly the psychological outlook plays a large part in this disease. Diagnosis has probably lagged behind symptomatology partly from neglect or absence of radiographic facilities and partly from too close an application to clinical evidences of pulmonary disease which may be absent for long periods at a time and failure to appreciate cardiovascular signs.

The disease also seems to affect the sexes differently. Females would appear to be more susceptible to the complaint

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than males (8-9 years difference in average exposure). It also seems to be fatal at an earlier age in women than in men. Cancer occurs after a briefer employment history, but in the more chronic case than in men. The "life" of the female asbestos worker with asbestosis or asbestosis and tuberculosis is longer than the male worker's, probably because of the retreat to the protective influences of home already mentioned. In both sexes cancer supervenes after more protracted exposure, in the more chronic case, after a long maturation period and in the higher age groups. Tuberculosis has not the profoundly modifying effect upon the disease as would have been expected. It is true that the average age at death is less than with other cases, alone or complicated, but most die in the first half of the fourth decade, that is to say, in the same quinquennium as cases of asbestosis without tuberculosis (Graph 5). When compared with uncomplicated asbestosis, rather more cases in the latter group survive to the first half of the fifth decade. The duration of the disease is not very appreciably lessened, but it occurs in cases which have had a briefer exposure. The industrial "life" is a little shortened, by comparison. The fact that the period of exposure and the maturation period is shorter in tubercular cases may be explained by postulating an increased vulnerability to respiratory infections generally and the effects of interjected infection. But undoubtedly the nature of the tubercular infection itself plays a great

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part in producing these somewhat complex phenomena. Gloyne has pointed out that obsolescent tuberculosis is not re-activated by asbestosis and also that the infection is of the acute caseous type. It is therefore more by way of being a terminal infection like bronchopneumonia, occurring towards the end of fibrosis, rather than in symbiosis with it. The disease is not therefore of much less duration, but the tuberculous infection behaves in other respects in the usual way by attacking the young and reducing the expectation of life. This question will be referred to later on in the discussion on tuberculosis as a complication of asbestosis. Like bronchopneumonia, the tubercular infection attacks in the 30-35 quinquennium, but unlike bronchopneumonia which is fatal in a matter of days or weeks, tuberculosis runs a course of months or years.

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Graph 1.

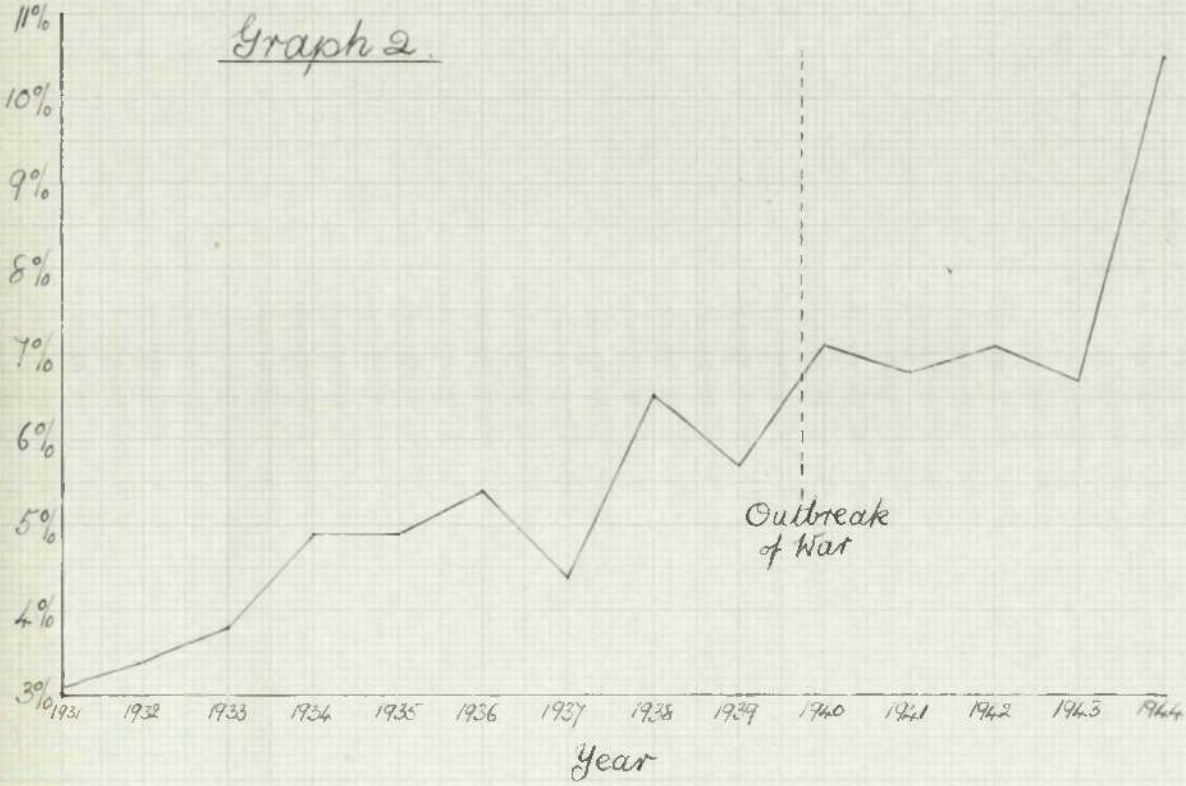


Year	Initial Exams	P.M.Es
1931	32	782
1932	295	665
1933	258	1157
1934	266	530
1935	488	1099
1936	420	1223
1937	848	863
1938	352	1463
1939	831	1547
1940	1331	1695
1941	1045	1425
1942	842	1991
1943	501	1368
1944	416	1680

Initial Exams

Periodic Medical Examinations

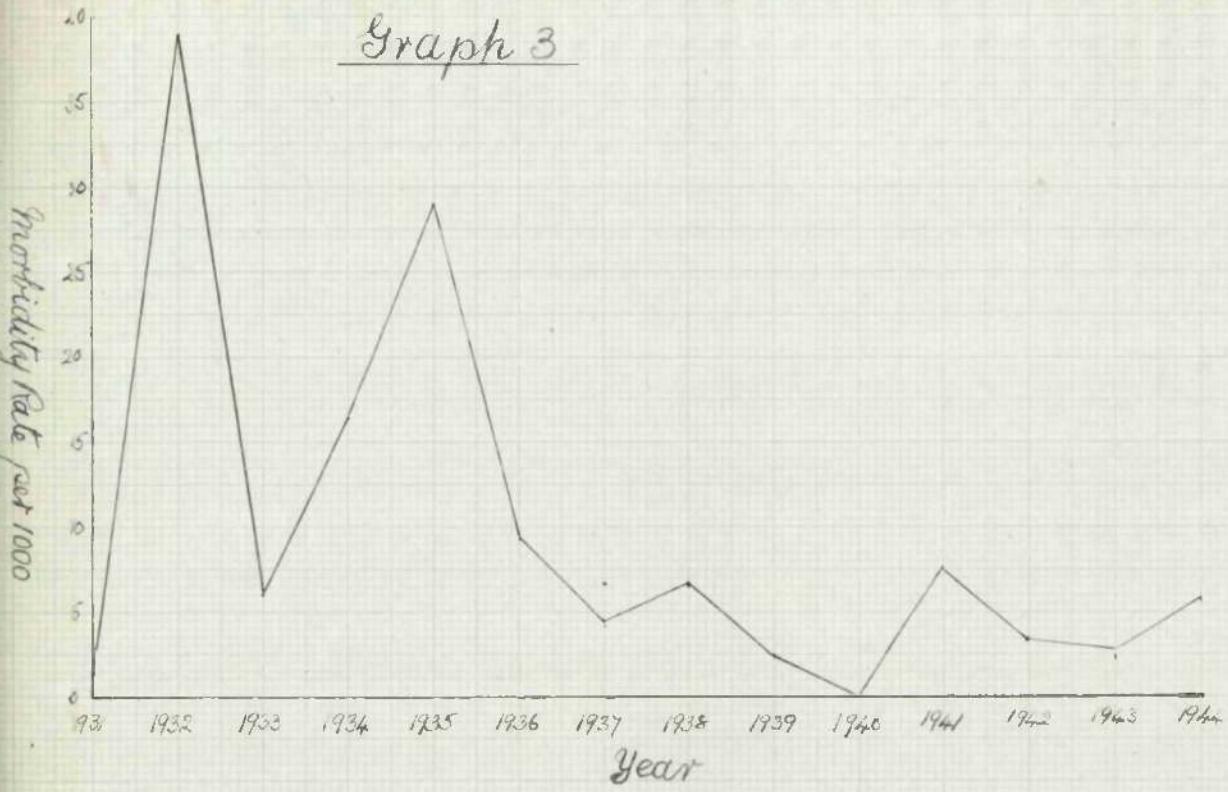
Graph 2.



Year	Initial Exams	Rejects	% Rejected
1931	32	1	3·1
1932	295	9	3·4
1933	258	10	3·8
1934	266	13	4·9
1935	488	24	4·9
1936	420	23	5·4
1937	848	37	4·4
1938	352	23	6·5
1939	831	48	5·7
1940	1331	95	7·1
1941	1045	72	6·8
1942	842	60	7·1
1943	501	34	6·7
1944	416	44	10·5

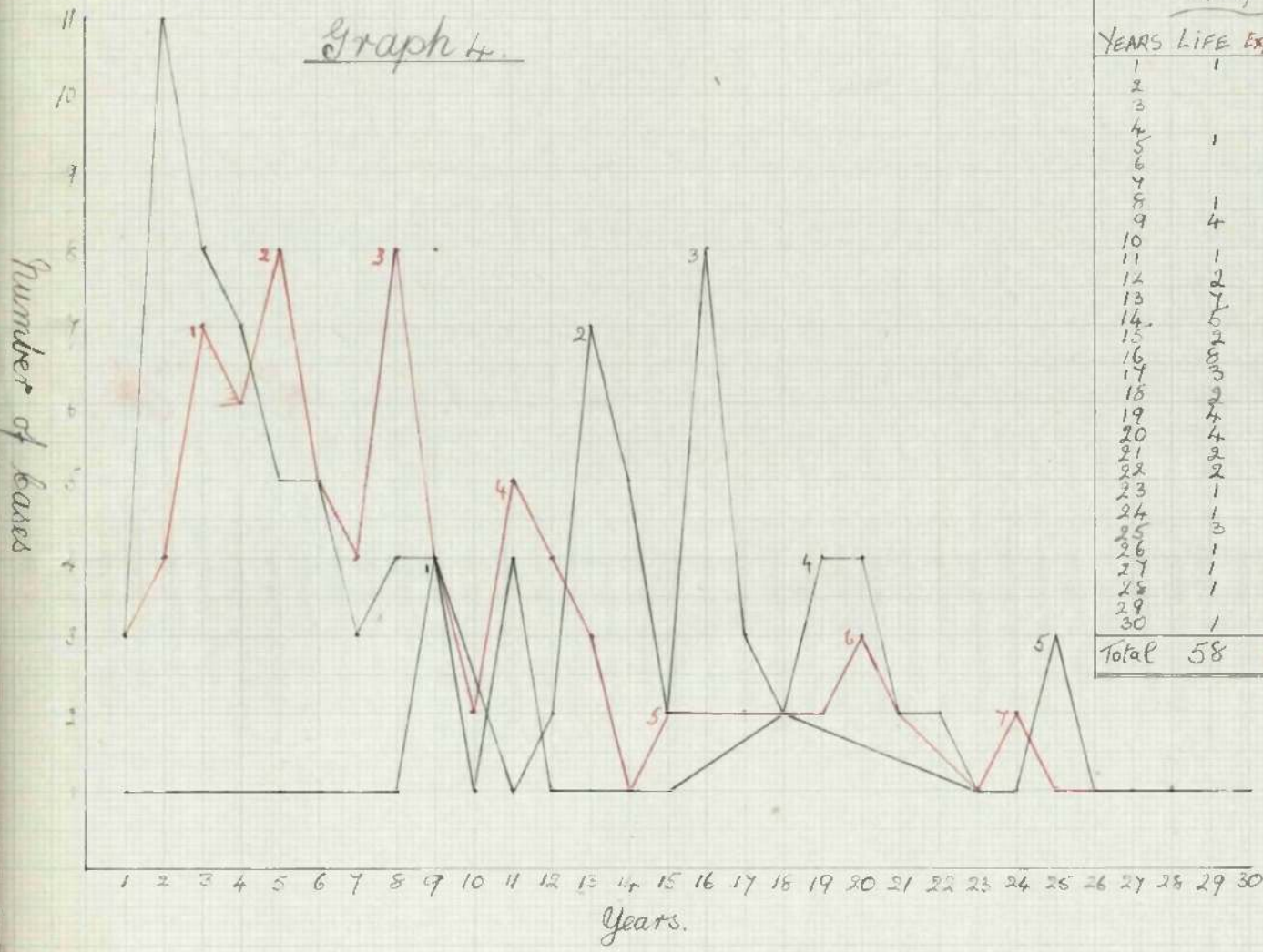
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Graph 3



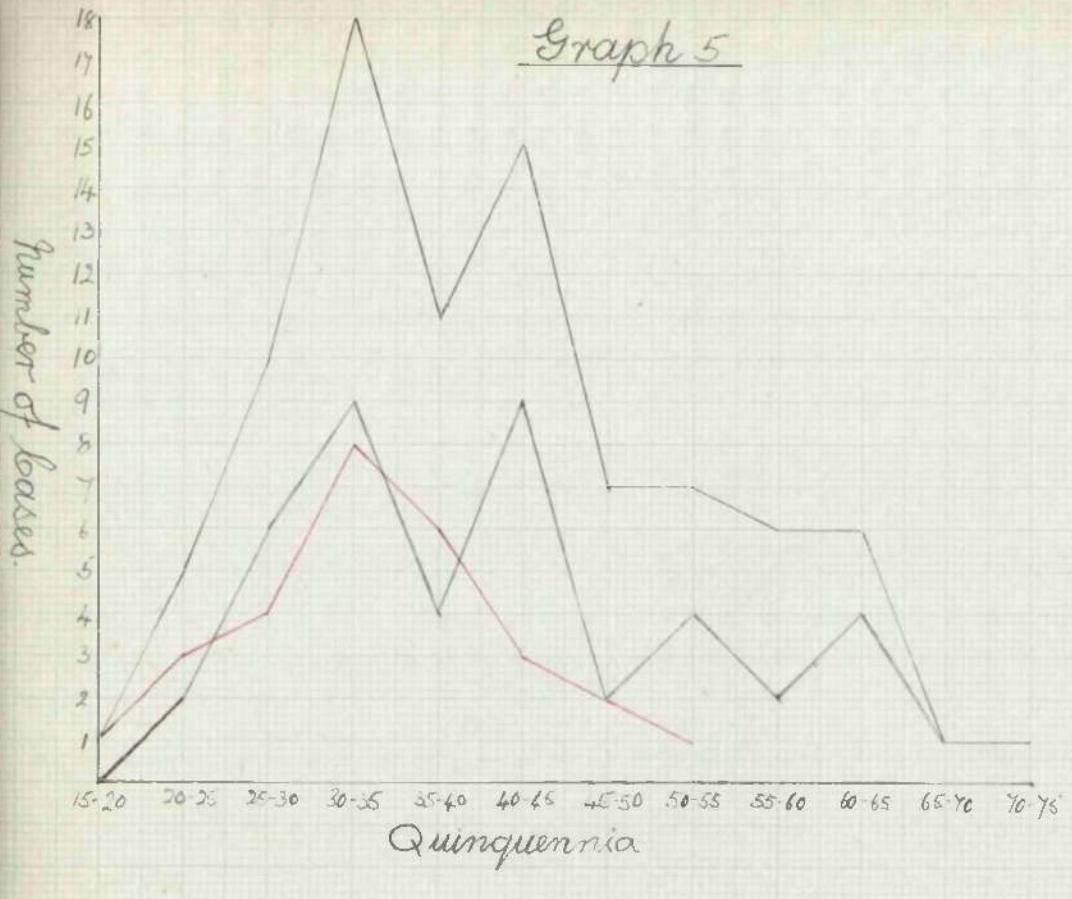
Year	Morbidity Rate per 1000.
1931	1·5
1932	39·1
1933	6·0
1934	16·9
1935	29·1
1936	9·8
1937	4·6
1938	6·8
1939	2·5
1940	0·0
1941	7·7
1942	3·5
1943	2·9
1944	5·9

Graph 4.



Years	Nº of Cases: Life	Exposure	Duration of Disease
1	1	3	3
2		4	11
3		7	8
4		6	7
5	1	8	5
6		5	5
7		4	3
8	1	8	4
9	4	4	4
10		2	1
11	1	5	4
12	2	4	1
13	1	3	1
14	5	1	1
15	2	2	1
16	8		
17	3	2	
18	2	2	2
19	4	2	
20	4	3	
21	2	2	
22	2		
23	1	1	1
24	1	2	
25	3	1	
26	1		
27	1		
28	1		
29			
30	1	1	
Total	58	82	62

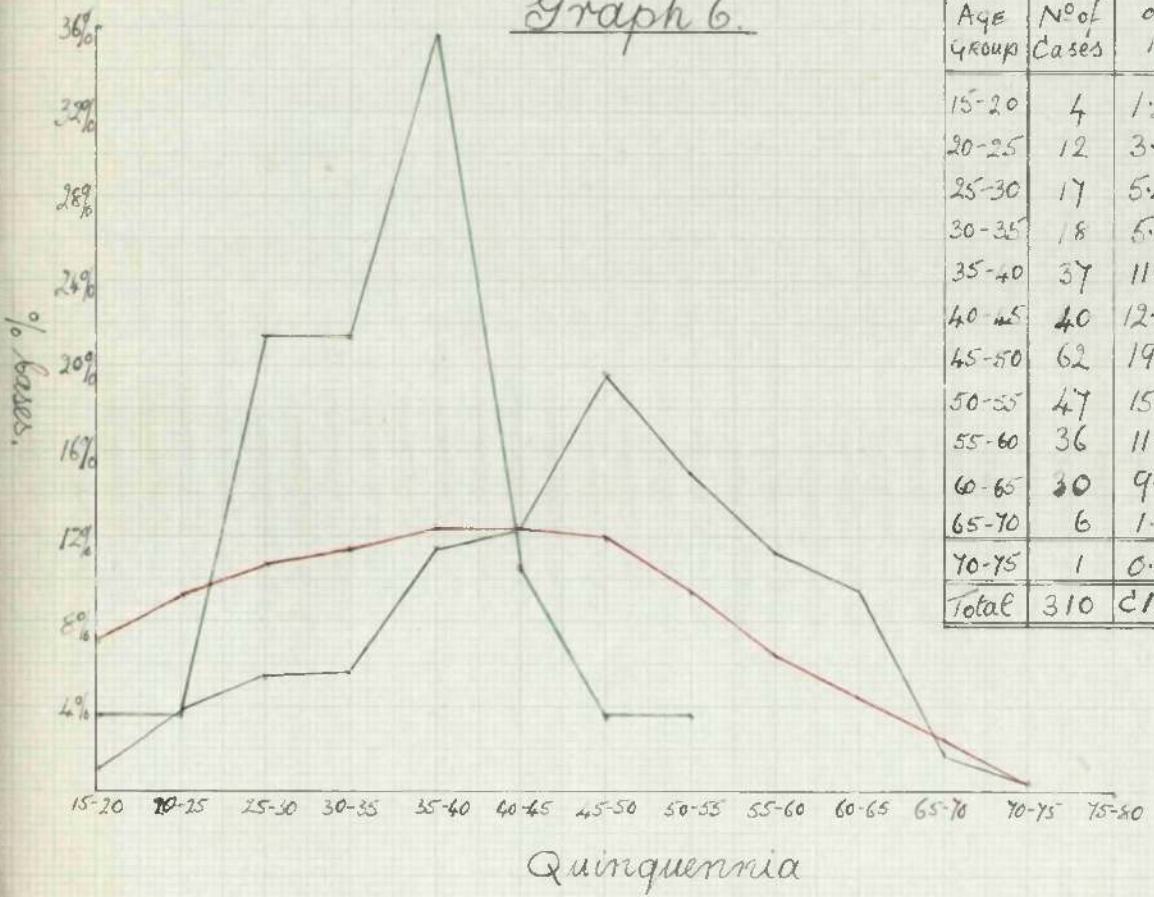
Graph 5



Quinquennia	All cases Asbestosis	A & T.B.	A alone
15-20	1	1	0
20-25	5	3	2
25-30	10	4	6
30-35	18	8	9
35-40	11	6	4
40-45	15	3	9
45-50	7	2	2
50-55	7	1	4
55-60	5	0	2
60-65	5	0	4
65-70	1	0	1
70-75	1	0	0
Total	86	28	43

Asbestosis, all cases
Asbestosis & Tubercle
Asbestosis alone.

Graph 6.



	Grinders		Controls		Asbestos	
Age Group	No of Cases	%	No of Cases	%	No of Cases	%
15-20	4	1·28	99	7·23	1	3·5
20-25	12	3·84	129	9·42	1	3·5
25-30	17	5·44	147	10·73	6	21·4
30-35	18	5·76	157	11·45	6	21·4
35-40	37	11·84	169	12·34	9	35·7
40-45	40	12·80	169	12·34	3	10·7
45-50	62	19·84	167	12·19	1	3·5
50-55	47	15·04	163	9·71	1	3·5
55-60	36	11·52	89	6·50		
60-65	30	9·60	61	4·45		
65-70	6	1·92	33	2·41		
70-75	1	0·32	8	0·58		
Total	310	c100	1361	c100	28	c100

Grinders
Controls
Asbestos Workers

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CHAPTER 4

CLINICAL, RADIOLOGICAL & PATHOLOGICAL SIGNS OF ASBESTOSIS

It has been well said that the clinical picture of a disease is a composite picture. The so-called typical, classical text-book case is generally a rara avis; and it is better that it is so, for if all examples of a disease were identical, then human beings themselves might also retreat beneath their carapaces of deadly uniformity. The device of a composite picture is necessary, however, for descriptive purposes and will be adopted here, but only in the sense that Galton superimposed portraits in order to discover common family or other characteristics of groups of people.

It is environment which chiefly determines asbestosis and although the factory may only be a part of that environment and although, as an observation of common experience, the individual has free will to move in and to mould his environment, his free will is occasional and limited. In the investigation of every industrial disease the factor of environment is paramount. Social and climatic influences have their bearings upon all questions of deviations from the healthy norm, but they seldom determine the onset, course and termination with the precision that the industrial environment determines an occupational disease. Just as it was shown in the last chapter that legal qualification for compensation depended

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among other things, upon a strict interpretation of First Schedule processes, so the diagnosis of asbestosis or any other occupational disease depends upon the strict interpretation of the medical history as respects the hazard to be considered.

It seems trite to say that plumbism cannot exist without exposure to lead and expressed thus simply may even appear ridiculous. The writer was once asked by a workman to consider an alleged case of lead poisoning for certification under the Workmen's Compensation Act in which exposure to lead was found to consist in the fall of a lead ingot on the workman's foot! But such cases are rare and the real difficulties arise when substances are toxic through an unusual and unsuspected channel of entry because, under industrial conditions, that may be the only available channel. Books on pharmacology are full of data relating to the toxicity of metals when injected beneath the integument, but for the workman they are generally inert. When the integument is attacked by a metal the results may be different from what the pharmacologist may anticipate, as when men engaged in turning articles of magnesium on a lathe begin to suffer from emphysema of the loose tissues of the arms from gas, evolved by the interaction of minute spicules of the metal embedded in the skin, with tissue fluids. Or, again, a substance may only become toxic under certain meteorological conditions. Dichloraniline is a solid at ordinary temperatures and

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will cause methaemoglobinuria by absorption through the skin. On a warm summer's day, however, it will volatilise and cause the same condition through the respiratory system. One hot day the writer was walking through a factory when two women were observed sitting outside a shed where spirit aether.nit. was being bottled. Both presented the leaden, grey-blue appearance of methaemaglobinaemia and this was confirmed by spectroscopic examination of the blood. Except for slight headache, neither felt ill, but was rather euphoric as such cases usually are. Both recovered completely in 48 hours. Infants have been known to suffer from the condition after ingestion of the substance by the gastro-intestinal tract. Although the particular job on which these women were engaged had been done frequently for years no previous trouble had been encountered. It appeared that under the conditions of high temperature prevailing that day and the large quantities of material being handled, sufficient sodium nitrite was absorbed to affect the blood changes described. Neither woman would admit to drinking the substance.

Contamination by unexpected substances also presents baffling problems in industrial toxicology. Minute quantities of arsenic in sulphuric acid, present in the nascent state, have been known to combine with hydrogen in the manufacture of that substance and to form arsine with disastrous results. The evolution of chlorine from hypochlorite and acid effluents has also been responsible

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for fatalities. Compounds may produce effects entirely different from the clinical syndromes produced by their component elements. Thus Hunter, Bomford & Russell (1940) (25) recorded four cases of poisoning by methyl mercuric iodide in the form of dust. Inhalation by the men in the course of manufacturing this fungicide caused severe generalised ataxia, dysarthria and gross constriction of the visual fields; the only resemblance to poisoning by metallic mercury was tremour. The observations are of particular interest in comparing the effects of silicon dioxide with the effects of such combined silicates as the asbestiform minerals, sericite and mica.

On the other hand, pathological conditions occur which are not specific to a particular substance. Aplastic anaemia should suggest to the practitioner's mind the possibility of benzene anaemia, but adequate investigation of the working conditions is an essential prerequisite to an accurate diagnosis. A catarrhal bronchitis may be due to other causes than the inhalation of vanadium pentoxide. Parkinsonism occurs apart from manganese. But in such cases the words of Lord Justice Goddard should be borne in mind: "We should approach questions under the Workmen's Compensation Act with a tendency to give what is sometimes called a liberal interpretation to the Act, that is to say, 'liberal' in favour of the workman, remembering that the Act was

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created to extend the remedies and right to compensation of the workman, and not to limit them in any way". It should be added, however, that the doubt must be real and not imagined.

Examples of failure to relate scientifically environmental history and pathology could be multiplied many times over and could be taken from the daily experience of every industrialist. Workmen habitually double the weights they have to carry by neglecting to mention the men who assist them; but generally the history a workman gives of his job should be treated like the history a mother gives of her sick child - it may be exaggerated but is seldom untrue and should never be treated lightly.

Granted that exposure to asbestos dust of sufficient concentration for an adequate period of time seems fairly established from the history, there remains a laboratory test which gives reasonable proof of exposure to the dust, namely the finding of asbestosis bodies in the sputum. For the practitioner who has not access to independent records of employment, this investigation is essential to diagnosis. That asbestosis bodies do not necessarily indicate pulmonary fibrosis is so well known as not to require elaboration here, but their importance in establishing exposure is unique.

It may be no more than accident that among the relatively small number of asbestotics as yet known, cancer

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has occurred chiefly in those situations where the asbestosis body is found: the lungs, pleura, hilar glands and intestinal tract. They have not been observed in asbestos warts. These "curious bodies" may be regarded as "an expression of a tissue reaction to a foreign body acting as a benign irritant". Their value is, to some extent, limited by the fact that they do not give any indication of concentration of the dust cloud or duration of exposure except within wide limits. Animals vary very much in their reactions to asbestos fibre, formation of asbestosis bodies having been variously recorded in guinea pigs after periods of seventy days to six months' exposure to dust. They have been observed by Simson in a human being after two months' exposure. Clumping of the bodies is generally held to signify break-down of tissues. Tubercle bacilli may, of course, also be found in the sputum since asbestosis is so frequently accompanied by tuberculosis.

If the pathologist exercises his craft chiefly on the end-results of disease, it is only because the early beginnings are usually inaccessible and not because they lack interest or importance. Indeed, it is the pathological process which occurs first and determines the clinical and radiological signs which follow. The interpretation of clinical signs by pathological appearances after death is perhaps fraught with many errors, chief of which is the necessity of arguing a

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posteriori. Nevertheless, it is the chief method of elucidating the mechanics of a disease. The mechanics of asbestosis as seen in the post mortem room will therefore be discussed first.

Dense, sessile adhesions of the pleura are common and the interlobar fissure may be obliterated; the pedunculated adhesions of tuberculosis are rare. The pleura loses its transparent character as the disease progresses and assumes a ground glass appearance. Yellow, horn-like plaques (sometimes 2" or 3" in diameter) may involve the membrane. Recent patches of pleurisy may also be found. The cut surface of the lung exhibits a typical picture of blue-black polygonal areas, each $\frac{1}{8}$" to $\frac{1}{4}$" in diameter. They are the homologues of the silicotic nodule, are roughly lobular in distribution, discrete at the apex and tend to confluence at the base. As with the pneumoconioses in general, the lesions exhibit the "pleural drift". The extent to which confluence occurs is an indication of the concentration of the dust cloud to which the respiratory organs were exposed. Hence it was more extensive in the earlier cases than in those seen to-day. The characteristic pigmentation is unaffected by the type of asbestos fibre which has been inhaled, whether it was white, blue or brown or a mixture. Bronchopneumonia frequently gives a red background to the blue areas. The vessels and bronchi are seen to stand out from the specimen because of their fibrosed walls and may be dilated. Bronchiectasis

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is rare. Emphysema is common and chiefly marginal; large bullae are commoner in silicosis than in asbestosis or indeed than in any other industrial disease of the lungs and when present are generally found in the apex. The middle mediastinal group of lymph glands are intensely pigmented and fibrotic. The right heart is generally enlarged and there may be the usual signs of back pressure. The spleen is generally septic.

Before describing the histological appearances of asbestosis, it is necessary to consider briefly the nature of the attacking substance, for what bacteria are to the general pathologist, dusts are to the special pathologist. The legal definition (Asbestos Industry Regulations 1931) runs as follows: "Asbestos means any fibrous silicate mineral, and any admixture containing any such mineral, whether crude, crushed or opened". As used in the trade, asbestos is a collective term applied to a group of silicate minerals which differ from one another in chemical composition, and to a less degree in physical properties, but which resemble each other in being finely fibrous and flexible. Unlike the vegetable fibre, they have no ultimate element beyond which splitting cannot be continued without loss of identity. Their value depends on the facility with which they can be split into long and flexible fibres for spinning, on their resistance to heat, acids and sea-water and on their insulating properties with respect to heat and electricity. Chemically they

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consist of silicates with metallic bases, mainly magnesium or iron and, to a less extent, calcium, sodium and aluminium.

Generally speaking, the asbestiform minerals occur in the banded ironstones and the deposits are classed as cross fibre, slip fibre and mass fibre. In cross fibre the delicate straight fibres stretch from wall to wall of the vein, whilst in slip fibre the orientation is in the direction of the vein. In mass fibre the fibres are scattered, stellate or radially axial. The countries of origin are Quebec, South Africa, Australia, the U.S.S.R. and Italy. Hall (31) describes three main groups:-

1. The Serpentine Group, e.g. Chrysotile, generally white in colour, characterised by a high percentage of magnesia and water and with the composition

$$2\,H_2\,O,\ 3\,Mg\,O,\ 2\,SiO_2.$$

2. The Rhombic Amphiboles, e.g. Amosite, contain a high percentage of iron with variable amounts of aluminium, magnesium and calcium. If soda is present, it imparts an amber colour. The general formula is

$$(Mg\ Fe)\,O,\ Si\,O_2.$$

3. The Monoclinic Amphiboles, e.g. crocidolite, are mainly silicates of calcium, magnesium, iron and sodium. In these cases, the soda imparts a blue colour and is invariably present. Crocidolite is represented by Dana thus:-

$$Na\ Fe,\ (Si\,O_3)_2,\ Fe\ Si\,O_3.$$

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White asbestos is regarded with most favour by the workers because it is reputed to be least dangerous, spins and weaves easiest and causes least wear on steel parts. Blue asbestos is regarded less favourably because it is said to be more dangerous, weavers are continually adjusting broken ends, necessitating constant standing at the loom and machine parts wear more quickly. The dirty white, or brownish yellow amosite is friable, dusty, difficult to work, so hard that steel parts which normally last weeks or months are worn away in two or three days and its reputation as regards health utterly pernicious; it is seldom now used. Blue asbestos resists acids and sea-water much better than white asbestos but does not resist heat so well; it therefore has considerable vogue in the chemical industry and for marine structures.

Chemical analysis explains the varying properties of these three groups of asbestiform minerals:-

	Canadian Chrysotile %	Blue Crocidolite %	Amosite %.
Silica	41	50	47
Alumina	3	1	6
Ferric Oxide	2	35	37
Magnesia	40	2	6
Alkalines	--	8	1
Water	14	4	3
	100	100	100

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Crocidolite and amosite contain high percentages of ferric oxide and low water content, providing a harsh and elastic texture which encloses the maximum amount of air in the minimum quantity of incombustible matter and endows them with special efficiency as heat insulators. It is the ferric oxide content of blue crocidolite which encourages slag formation when it is used as a wrapping for electrodes. On the other hand, the high magnesia content of white chrysotile provides a natural lubricant and renders this variety most suitable for stuffing boxes and glands and accounts for lessened wear and tear on steel parts in weaving and spinning.

Dust is inseparable from every industrial process. Indeed it is inseparable from life on this planet; it is the dispersed phase of the time-space continuum. It may be defined as any substance which is so divided that the particles are small enough to be blown about by the wind. Particle size varies up to 200 microns. The most dangerous size of asbestos particle is sixty microns in length, and the most dangerous concentrations are those in excess of 5,000,000 particles per cubic foot. The shortest fibre recorded is Grand Canon chrysotile of the order of 0.00075 m.m. in length whilst the longest is exhibited in the Maritzburg Museum and is 43" in length. The latter fibre came from Umsinga, Natal, and is also probably chrysotile. The best length of fibre for

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spinning and weaving is $\frac{1}{4}$" to $\frac{3}{4}$" long.

The lobular bronchioles are 200 microns in diameter and it is in these that the asbestos fibres with their frayed ends, tend to be arrested and set up irritation. Asbestos belongs to the class of non-toxic inorganic dusts commonly thought of as dust. Other classes are (1) systemic poisons such as lead, arsenic, manganese and trinitrololuene, (2) irritant or corrosive dusts, as lime, arsenic, chromic acid and the bichromates, (3) allergic dusts such as pollen grains, (4) carcinogenic dusts, as for example, pitch and radio-active materials and (5) infective dusts carrying anthrax, monilia, actinomyces and other fungi. Among the dusts are smokes and fumes with particle sizes down to 0.5 micron and 0.1 micron respectively. Both types tend to flocculate readily, so increasing particle size and reducing penetration, as when the cobweb of metallic oxides sink to the ground during the process of arc-welding owing to the low vapour pressures of this phase of matter. Mists result from the atomisation of liquids or the volatilisation of solids and from the condensation of vapours when the barometric pressure falls. Animal experiments with chrysotile asbestos ground to less than three microns in length tend to suggest that the action here is more like that of an inert dust; instead of being held on the irregular surfaces of the respiratory bronchioles, the particles pass into the terminal air spaces where they are phagocyted.

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It is in this last respect that silicon dioxide differs from asbestos. Silica is transported from the alveoli through the lymph channels by means of dust cells to the lymph nodes where the particles slowly dissolve and, perhaps by local toxic and chemical action set up a fibrosing action which results in the silicotic nodule. On the other hand, the desquammated epithelial cells of the bronchioles do not phagocyte the insoluble asbestos fibre. Macrophages, the homologues of the dust cells, attempt to engulf the fibres which are generally too long to be enclosed. Asbestosis giant cells, probably collections of partially degenerated phagocytes which have lost their envelopes, are uniformly present. The fibroblasts are found around bronchioles and alveoli and in the interlobular septa and sub-pleural connective tissue. There is little leucocytic reaction. Gloyne (26) has noted whorls of connective tissue resembling discrete silicotic nodules near the point where the interlobular septum joined the pleura, i.e. the point at which the pulmonary deep lymphatic vessels in the septum anastomose with the sub-pleural lymphatics.

The process of fibrosis seems, from animal experimentation, to proceed by a cuffing of the bronchioles. This fibrosis does not appear to cause atalectasis of the peripheral air spaces by contraction as might be expected. This seems to be due to the fact that the inner surfaces of the bronchioles become smoother and inelastic and permit

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the passage of further fibres to more distal parts. Eventually the alveolar ducts and atria are also involved forming a reticular pattern. Fibrosis of the alveolar sacs has not been observed experimentally but sections showing advanced disease indicate that they are crushed, sometimes out of recognition. The physiological effect is to impair gaseous interchange, contributing towards the most characteristic symptom of the disease, dyspnoea. The respiratory excursion is also diminished from replacement of elastic by fibrous tissue to the extent that is easily detectable by the eye as well as by cyrtometric measurements. When the thorax is opened at post mortem the lungs do not collapse as would normally be expected. The devotee of hyper-aeration in the treatment of pneumonia and tuberculosis might find here an explanation of the proclivity of asbestosis for these diseases.

There is an acute as well as a chronic type of asbestosis, the one sometimes merging into the other, the former due to heavy concentrations of dust and of the type described by Merewether, the latter due to lower concentrations spread over prolonged periods and exhibiting the stigmata of chronic disease. Asbestosis differs from silicosis in being a clinical, rather than a radiological disease, that is to say that whereas gross radiological evidences of silicosis may be present in the absence of any clinical signs, the reverse may be found in asbestosis. As Amor (27) has remarked of silicosis: "It is important

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to realise that even when the most extensive degree of pulmonary involvement is present the physical signs may be relatively slight" and "radiological examination of the lungs provides the most reliable single method of diagnosing silicosis during life". Neither of these statements is true of asbestosis. Indeed, it is the almost monotonous experience of the post mortem room that one year's exposure to the old, unregulated conditions will produce asbestosis of a degree sufficient to be fatal when insufficient to be convincing from the skiagram alone.

Case 2. R.C.N. Female. Age 31.

History: Mattress Department 28/7/21 to 3/12/29.
Developed a dry cough in 1925 which never left her and eventually she gave up work owing to dyspnœa.
Died 14/3/34.

Clinical Signs: Slight clubbing of the fingers. Basal crackles. Poor chest expansion. Emaciation.

X-Ray: No definite abnormality seen.

P.M.: Recent and old pleurisy. Plaques. Advanced asbestosis. Asbestosis bodies +. Fibres ++. Small cellular reaction, but extensive fibrosis suggestive of a long standing condition. Amount of fibrosis seen in sections out of proportion to naked eye appearances. Fatty degeneration of heart and liver. Septic spleen. Terminal pulmonary congestion. Emphysematous bullae along lower free margins. Azygos lobe right upper lobe probably due to abnormal course of azygos vein.

Diagnosis: Asbestosis.

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The importance of skilled histological examination of specimens is also evident from this case.

Attention in the past has tended to be focussed too much upon signs in the lungs themselves to the neglect of cardiovascular effects. This is unfortunate, as adventitious sounds in the chest are apt to be evanescent, whereas the cardiovascular signs, once they appear, are permanent. As Pendergrass (28) has remarked: "The heart and pericardium also suffer in asbestosis, much more so in this occupational disease, in fact, than in any dust hazard with which I am acquainted". Whilst it would be erroneous to assume that the two systems can be thought of as anything but interdependent, there are broad, general considerations in which symptomatology may be referred to one or other system.

1. Respiratory System

Within three or four weeks of employment in a First Schedule process, a few workers develop catarrhal sounds in the chest and sometimes bronchial spasm may be noted. This is not specific to asbestos and should be clearly differentiated in the medical examiner's mind. Such phenomena are common whenever dust of a non-toxic, non-fibrosing type such as graphite is inhaled. Removal from the dusty environment results in complete recovery in the course of one or two weeks. Should this course not be adopted, however, the catarrhal condition will continue as long as exposure continues. Whether such

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persons are more liable to pulmonary fibrosis or not cannot be answered empirically; the writer has always acted, in such cases, as if they were more susceptible and had them withdrawn from the hazard. Normally, the vibrissœ of the nose trap a proportion of the dust if the person breathes through that organ; but if the nose is not well equipped with a straining mechanism, or the person is a mouth breather, much larger quantities of the grosser fibres will penetrate into the bronchioles. This is the most likely explanation of the clinical phenomena and it seems a reasonable conclusion that such people should be excluded from the industry.

Burton Wood's statement 11 years ago that asbestosis is almost a monosymptomatic disease remains true. Dyspnœa is the chief complaint of the patient. It is first noticed in the performance of the less frequent acts of the day, such as the exertion of getting to work or of going upstairs to bed. Once at work, the day's routine is managed with little discomfort. Later, the patient may take to sleeping downstairs and eventually use a bath chair for progression. There are several possible causes of dyspnoea, all of which act at the same time: (1) The crushing of alveoli and bronchioles by fibrous tissue. Starling (29) calculates that it is possible for 6,000 cc. of oxygen to pass through the 90 square metres of alveolar surface in the normal lung in one minute, but in the resting state only 250 cc. are actually used. There is,

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therefore, an enormous reserve which would require almost complete fibrosis of the lungs to produce dyspnoea from this feature alone. (2) Defective gaseous interchange consequent upon fibrosis. It is necessary for the blood to contain 6.7 cc. of O_2 unsaturation per 100 cc. or 5 gm. of reduced haemoglobin per 100 gm. to produce cyanosis, and cyanosis is not commonly seen. (3) Defective circulation in the lungs, as occurs in mitral stenosis. As will later appear, there seems to be some evidence to support this observation. (4) The almost invariable presence of emphysema which would contribute to the reduction in alveolar surface. (5) Cardiac failure might be a factor but only in the later stages of the disease. (6) Meiklejohn has put forward the interesting hypothesis, in the case of silicosis, that the fibrosis causes the lungs to expand (as seen at post mortem) so that they come to occupy, ultimately, a mid position between inspiration and expiration, thus reducing the volume of tidal air. (7) Fibrotic bands between the pleura and pericardium tend to anchor the heart and impede its functions.

Cough is variable, may be troublesome, but is usually present on rising in a morning. Expectoration is slight and rarely blood stained; it is generally thick and mucoid. Loss of weight is a constant symptom. Fear of the "dust" produces a somewhat melancholic appearance and psychological outlook. A dull ache, rarely sharp pains, may be complained of in the chest. The so-called earthy

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cyanosis may be observed when clubbing of the fingers is present, but in itself presents great difficulties of assessment and is open to gross inaccuracies. The normal person may appear cyanosed and the pathological case may seem to be within normal limits. Not only the cheeks, but the lobes of the ears and the colour of the nails should be examined.

On inspection, the chest is seen to be poorly clothed and one is impressed by the low amplitude of the respiratory excursion, often not more than an inch in circumferential measurement and this even when dyspnœa is present from the mere act of divesting the clothes. The breathing is chiefly of the abdomino-thoracic type with indrawing of the epigastrium on inspiration. Litton's sign (32) is not usually observed probably because of adhesions in the costo-phrenic space. Both sides of the chest move equally and there is no flattening. On palpation, the apex beat and trachea are normally placed and the trachelo-mastoid sign (30) is absent. Pleural friction may be felt. Percussion may indicate patchy dullness. Over these areas the experience through the plexímeter finger is sharp and definite. The patches, if found, remain constant and permanent at all subsequent examinations and are probably due to those plaques in the pleura found at post mortem. Merewether has described the sensation of "felting" on percussion, but the writer's experience is that one's tactile and auditory perceptions

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are in inverse ratio of acuity; at any rate, he feels more confident of the latter subjective phenomena in the assessment of clinical findings.

Friction may be heard over the patches of dullness, which may persist for weeks, is painful but never becomes moist. Numerous adventitious sounds may be heard in all areas if the patient has a "cold", but may be completely absent in the same person a few weeks later. In more advanced cases, the adventitious sounds persist. Generally, dry fibrotic crackles and marginal emphysematous crepitations predominate. A high pitched, slightly musical rhonchus with a somewhat woody timbre, lagging behind the act of expiration, is sometimes and characteristically present.

2. Cardiovascular System

Clubbing of the fingers is a sign of considerable value in this disease, but as there seems to be some confusion as to what is meant by the phenomenon a brief consideration of the subject may not be out of place. Such loose terms as "clubbism" and "curving and parrot-beaking of the nails" are frequently mentioned, terms which leave much to the imagination and would be better qualified by reference to the angle. In other words, it is essential that the potter whose fingers have become bulbous from kneeding clay should not mislead the examiner into diagnosing clubbed fingers as a confirmatory sign where other evidences of silicosis are perhaps equivocal.

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To quote Lovibond (34), "It is manifestly a malady of the nail beds, or, more correctly, of the soft tissues between the nail bed and the bone. Although the true pathology is still in dispute it seems likely that chronic passive congestion of the capillaries of the nail bed brings about an oedema of the tissues deep to it, which in turn is followed by trophic changes resulting in clubbing. An essential contributory factor that must be present before the characteristic reaction will occur is a chronic anoxaemia of the blood in the terminal arterioles, such as obtains in many states of pulmonary and cardiac disease. It is thought that all venous blood contains toxic substances tending to provoke the typical changes in the fingers. Normally these toxins are removed from the blood in its passage through the lungs, hence pulmonary disease predisposes to clubbing". He goes on to describe the condition as a definite, firm, transverse ridge at the root of the nail which causes an increase in the normal angle of 160° subtended by the nail on that part of the finger which is proximal to the nail root. When 180° is reached, slight clubbing is present; in gross clubbing, not only does the angle exceed 180° but the nail may be rocked as if it were floating. The sluggish circulation can be demonstrated by pressure on the nail. The pathology and signs are similar in clubbing of the toes. The sign is well known and sometimes accurately described by asbestos workers themselves who regard it as of ominous

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portent. In 29 consecutive cases of asbestosis the writer found 14 to be showing clubbing of the fingers, and in 2 of the cases the toes also were affected (48.2%). The condition is said to be 8 times more common in men than in women. In this series 11 were men and 3 were women, but this is not remarkable owing to the preponderance of female labour in the industry. Lovibond does not mention asbestosis in his list of diseases exhibiting the phenomenon. Amor (27) says of silicosis: "Clubbing of the fingers is extremely rare, but sometimes occurs late in the disease".

Clubbing is of biological as well as clinical significance, for it was not considered as of great importance 15 years ago. Ellman (1933) (40) considered that clubbing was found in the more advanced cases, in which bronchiectasis was usually present. Burton Wood's view (1934) (33) was that "Early clubbing of the fingers may occur but is seldom well marked, and when seen is usually represented by slight swelling of the skin surrounding the proximal ends of the nails". Through the kindness of Dr. Roodhouse Gloyne, the writer obtained access to the late Dr. Burton Wood's notes on 89 cases collected up to 1931. He described 2 as exhibiting pre-clubbing, 9 with curving of the nails, 6 with slight clubbing and one with marked clubbing. In 11 cases he made the definite statement that no clubbing was present and did not mention the feature in 51 cases. His

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recorded incidence was therefore 20.4%, although at least some of his cases would not come within Lovibond's definition. Lanza (1938) says that cyanosis and clubbing of the fingers are common late in the disease as is loss of weight and emaciation. The writer's experience is that the sign occurs relatively early in the disease. That the incidence of the sign has more than doubled since 1931 seems to indicate the existence of a more chronic type of the disease.

A feature which seems to have escaped observation altogether is an accentuation or reduplication of the second pulmonic sound, often of a loud slapping character as heard in cases of patent ductus arteriosus and some cases of pulmonary stenosis. In the 29 consecutive cases already mentioned, it was present in 22 (75.5%). The sign would appear to be due to the increased intra-pulmonary pressure consequent upon the fibrosis. Peripheral blood pressures tend also to be raised, more particularly the diastolic readings. Of the 29 cases 6 had average systolic pressures of 160 m.m. or more (20.7%) and 16 exhibited diastolic pressures of 90 m.m. or more (55.1%).

It is possible that once fibrosis has commenced in one part of the vascular tree, namely the pulmonary arborisation, the condition may spread, perhaps even in the absence of local stimuli to the other parts. Lynch and Smith (1930) (37) have reported asbestosis bodies in

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the thrombi of veins. A more likely explanation would seem to be found in the physico-chemical effects of pulmonary fibrosis on the circulation. (1) There is likely to be an increase in the pressure in the pulmonary artery which is reflexed in the usual way upon the systemic veins and capillary bed. (2) The diminished pulmonary ventilation lowers the suction effect of inspiration upon the great veins, auricles and right ventricle, still further contributing to a raised venous pressure, capillary congestion and depletion of oxygen from the blood, the pulmonary fibrosis aiding the latter by defective oxidation. (3) So far, the mechanics are much as in mitral disease, but in asbestosis the left heart is not diseased. It therefore responds to the raised peripheral resistance and the tissue demands for oxygen by increased force. Much of systole is absorbed by the elasticity of healthy arteries, but the basal, diastolic pressure remains elevated. Should, however, the arterial system develop a slight degree of sclerosis, the fact is betrayed more readily by the sphygmomanometer than if the other factors were not present. Hence also toxic substances are concentrated in the stagnant capillaries of the nail bed so as to cause clubbing of the fingers. That these phenomena occur before, and often apart from, other evidences of back pressure such as oedematous viscera and extremities is no new observation since enlargement of the liver may often be

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found with or without oedema of the dependent parts and vice versa. It is of interest that clubbing of the fingers and accentuation of the pulmonic 2nd sound only occur in those varieties of morbus cordis in which cyanosis is present and in which the right heart is involved, namely the tetralogy of Fallot, pulmonary stenosis and pulmonary atresia.

The heart is generally of normal size, the sounds closed and of normal rhythm, but as the disease progresses the right heart may enlarge, to be followed later by the signs and symptoms of congestive heart failure already mentioned.

The three cardiovascular signs of asbestosis, an accentuated 2nd pulmonic sound, a raised diastolic blood pressure and clubbing of the fingers are set out in the Table No. 5. It will be apparent from the table that there is no apparent correlation of the signs, a fact which serves to give point to the opening sentence of this chapter that the classical clinical picture of a disease is a composite picture.

TABLE No. 5

Case	Sex	Age	Clubbing	Blood Pressure Systolic	Blood Pressure Diastolic	Accentuated 2nd Pulmonic
E.T.	F	37	-	180	130	-
A.W.	M	57	+	142	90	-
F.B.	F	38	-	154	100	+ +
R.B.	F	42	-	140	98	+

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TABLE No. 5 (Contd.)

Case	Sex	Age	Clubbing	Blood Pressure Systolic	Diastolic	Accentuated 2nd Pulmonic
A.L.D.	M	52	-	164	84	-
E.E.	F	42	+ +	140	80	+ +
F.J.F.	M	63	+ +	150	88	+
J.G.	M	54	-	144	96	-
F.M.H.	F	46	-	126	80	+
J.H.	M	32	-	140	100	+
M.M.	F	39	-	110	90	+ +
V.M.	F	40	+	150	98	+
T.McK.	M	54	Fingers & Toes +++	166	84	+ +
A.M.	M	56	-	180	94	+
A.M.	F	35	-	96	64	+ +
F.N.	F	39	-	124	70	-
J.N.	M	40	+ +	140	98	+
A.O.	M	55	+ +	160	110	+
J.P.	M	45	+	150	86	+
W.P.	M	42	Fingers & Toes +++	122	80	+
A.R.	M	59	+	140	100	-
C.J.S.	M	60	-	110	76	+
G.S.	M	42	+	154	96	+
G.T.	F	40	+	144	84	+
W.E.W.	M	49	-	150	80	+
C.W.	M	42	+	168	128	-
R.W.	M	50	+ +	150	104	+
R.E.W.	F	38	-	130	80	+
A.W.	M	51	-	136	90	+

Figures are averages. Readings ascertained by means of mercurial sphygmomanometer.

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The radiological appearances are important and of great value as confirmatory aids to diagnosis; but whereas it is possible (if inadvisable) to make a diagnosis of asbestosis on clinical grounds alone, the skiagram can, at best, only suggest or confirm the diagnosis. It is a matter for discussion as to whether the radiologist should merely describe what he sees and leave the diagnosis to the clinician, or express his opinion. This much, however, is certain: unless the radiologist is in possession of the employment history, at least, he will fall into serious error should he attempt to make a diagnosis. The radiological appearances in the chests of arc welders are similar to those seen in asbestosis and yet there is no evidence that the former produces symptoms, disability or shortens life. Miliary tubercle may closely simulate silicosis except that whereas the tubercle has an opaque centre and clear corona, the reverse is the case in the silicotic nodule; but much careful searching of the film is necessary to reach a convincing conclusion. As Merewether pointed out in his Annual Report for 1943: "There is, however, already sufficient evidence that abnormal X-ray appearances which may be discovered in workers who may be exposed to dust or fumes but which are not caused by non-occupational diseases, do not always signify any present or impending disturbance of health; still less should they be regarded as <u>necessarily</u> signifying the existence of a definite

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occupational disease. They may, and often do, indicate exposure to an industrial dust, but that does not necessarily imply the existence of a pneumoconiosis, or disease, or present or future disablement, any more than the discovery of asbestosis bodies in the sputum by itself determines anything but exposure to asbestos dust, and that not necessarily to a significant degree".

Where radiological signs are admittedly difficult to detect and even more difficult to interpret, consideration of the various structures likely to cause shadows is important. The integument can generally be ignored except in so far as prolonged inunction with mercurial ointments or other radio-opaque substances may be concerned. The female breast shadows in the lower fields and the male pectorals in the mid fields may simulate (or conceal) asbestosis. The elliptical shadows delimited by the coincidence of the female breast shadows and the cupolae of the diaphragm have been mistaken for echinococcus cysts. The pleura becomes thickened, opaque and of a ground-glass appearance to the naked eye in asbestosis and may be expected to modify the radiological appearances of underlying tissue. Beneath the pleura is usually an agglomeration of the polymorphic areas of fibrosis already described, so that what appears upon the plain surface of the film as central may actually be peripheral.

Iron which has been inspired into the lungs as part

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of the siliceous material or obtained from the tissues in the formation of asbestosis bodies, may also add its quota to the film. As Sutherland remarked (38): "Definite X-ray changes are described as a ground-glass appearance of the lung parenchyma with a fine stippling; but such changes usually occur when physical signs are even more definite. In the production of this stippling there is a possibility of the density of the shadows being increased by iron either from the fibre or deposited from the body fluids (asbestosis bodies)".

Finally, the basal congestion accompanying a failing heart presents appearances closely resembling asbestosis. The heart shadow and enlarged hilar shadows together with the more homogeneous appearance of oedematous lungs will serve to differentiate the condition from asbestosis provided the patient has never been exposed to asbestos; but it is in precisely such a case where it is necessary to know whether asbestosis is or is not concealed beneath the oedema. Generally, this would be impossible on radiological appearances only. History and clinical examination would be required to supplement findings based on a film. Fortunately, such cases do not often arise, because, as was pointed out in Chapter 2, there is a statutory obligation on the Medical Board to exclude cardiac cases from First Schedule occupations. Periodic medical examinations enable the Board to discover cases of carditis developing during employment and also to

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anticipate just such a film by identifying asbestotics before the heart fails either from rheumatic endocarditis or right heart failure. Employees who leave the industry have usually passed the acute rheumatic age.

There is no place here for an elaboration of the classical descriptions of the X-ray film in asbestosis. Such descriptions have been adequately dealt with elsewhere, but briefly they are described variously as like a cobweb, like ground-glass, curtained with butter muslin, and as having a dusty appearance. But these appearances were more characteristic of gross disease consequent upon massive innocula of the dust. To-day, the radiological characters tend to be coarser and more granular and the dusty appearance seems to have gone. Some reticulation is often present and, in addition, the film indicates innumerable small opaque areas, roughly circular and measuring up to 2 or 3 m.m. in diameter, on a 14" x 17" film, with fine radiating threads. It is this latter appearance of stippling which is so marked a feature of the radiological appearances. The hilar shadows show little enlargement in the uncomplicated case and certainly not to the same degree as in silicosis. Where there is most lung tissue in the antero-posterior view, i.e. where the heart is situated and above the diaphragm, fibrosis appears densest, giving the flared out, shaggy outlines to the cardiac silhouette and cupolae of the diaphragm. The costo-phrenic and cardiaco-phrenic angles are blunted from

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old exudate. Generally the fibrosis is seen in the lower two thirds of the lung fields, but variations do occur. For example, it may be observed in the lower third of one lung and the middle third of the other lung, in the same patient as in J.H's film. In two cases in the present series the fibrosis was seen to be in the upper halves of both lung fields. The interlobar fissure sometimes shows a tendency to wander in an anticlockwise direction. The right heart is sometimes seen to be enlarged. The trachea is placed centrally. A lateral view of the chest is sometimes useful, but screening is hardly ever necessary and then only to observe the amplitude of the diaphragmatic movements which are diminished in asbestosis. Serial films are only of use if radiographic technique is standardised.

Emphysema can usually be demonstrated in the upper lung fields when the fibrosis is basal and vice versa in upper lobe fibrosis. Tuberculosis, when present, is generally apical and then presents no more unusual differences than elsewhere. On the other hand it may be impossible to diagnose the condition if it is basal. A diagnosis of cancer and asbestosis has not, so far as the writer is aware, been made radiologically, a feature which is perhaps not very surprising considering the small cancers which have occasionally been found in asbestotic lungs at post mortem. The balance in favour of a fatal termination seems determined, in these cases, before the growth has involved much lung tissue.

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Case 3. A.O. Male. Age 45.

History: Weaving Department 8/8/32 to 31/12/42.

Managed to evade examination in 1942 because he was losing weight and understood this to be the first sign of asbestosis. He admitted to dyspnoea in June 1943 and dry cough in September 1943. In December of the same year he had an attack of pneumonia.

He died on 13th November 1944.

Clinical signs: Marked clubbing of fingers. Anterior chest hyper-resonant. Coarse basal crepitations. Accentuated 2nd pulmonic sound. B.P. 160/110.

X-Ray: Typical. Mottling in lower two thirds of lung fields, emphysema in upper zones, shagginess of left cardiac border, blunting of cardiophrenic and costophrenic angles.

P.M.: Asbestosis and Emphysema.

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CHAPTER 5

COMPLICATIONS

(a) Respiratory Infections

Most important of the complications of asbestosis are pulmonary tuberculosis and bronchopneumonia; indeed, few cases reach the post mortem room without exhibiting some phase of the pneumonic process. Of 98 fatal cases 30 (30.5%) were found to be complicated by pulmonary tuberculosis. But serious as this risk of tuberculosis is, however, the incidence is not so high as in silicosis. The following table has been compiled with the assistance of the Annual Report of the Chief Inspector of Factories for the year 1943:-

TABLE No. 6

	Number of Deaths	T.B. %	Average age at Death	Duration of Employment in years		
				Longest	Shortest	Average
Merewether's Report:- Silicosis	801		57.2	62.0	1.5	34.1
Silicosis with T.B.	859	51.7	53.3	67.0	0.7	31.4
Asbestosis	118		46.4	48.0	0.5	15.1
Asbestosis with T.B.	62	34.5	30.0	29.0	0.8	10.4
Writer's Series:- Asbestosis	68		43.6	42.0	0.5	11.5
Asbestosis with T.B.	30	30.5	34.9	24.0	0.75	7.6

It is probable that many cases in the two series are identical and as Merewether's series is much the greater, his figures for the percentage of tubercular cases and average ages are probably the more accurate. In any case; the conclusions are not materially altered, viz: (1) There are about even chances that a silicotic will develop tubercle, whereas in the asbestotic the chances are two to one against. (2) Asbestosis kills at an earlier age than silicosis. (3) When tuberculosis complicates asbestosis it seems to cause a more rapidly fatal issue than when it complicates silicosis. (4) The average exposure is less in the asbestotic than in the silicotic.

In his examination of 310 cases of silico-tuberculosis in Sheffield metal grinders, Meiklejohn (1) was able to show that there are two types of tuberculous infection in silicotics. One, which he called the "Natural Group" included those who would have died from tuberculosis by reason of factors of heredity and contact and irrespective of occupation and a second group, which he called the "Industrial Group" who contracted tuberculosis on account of occupational pulmonary fibrosis. The first were of the age group 40-45 and the second of the age group 45-65. The latter belonged to Brownlee's middle age type. He was also able to show that the course of tuberculosis was similar when it complicated silicosis and when it occurred alone. On the other hand, tuberculosis attacks the asbestotics of the 30-35 age group and does so after a

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briefer exposure and shorter maturation period than the uncomplicated cases of asbestosis (Graph 6). We are therefore dealing here with cases likely to show a much more acute and adolescent type of tuberculosis and one more likely to act as a terminal infection. It is doubtful if any asbestotics with tubercular complications can ever be considered as falling into Meiklejohn's "Industrial Group" and in that respect also differ from silicotics.

Case 4. Miss M.C. Aged 35. Carding Department 11/1/26 to 1/10/30.

History: Always well until 1930 when she began to suffer from chest pains and dry cough. Attended the London Chest Hospital in October 1930 and continued to do so for a year. She complained of pains in the back and front of the chest of 6 months' duration, dry cough for nearly a year and thought she had been breathless and losing weight for 2½ years.

Clinical Exam: Healthy appearance. No cyanosis or clubbing. Basal dry crackles.

X-Ray: Slight haze at left base.

Pathological: Asbestosis bodies present in sputum. No tubercle bacilli seen.

It was concluded she was a very doubtful clinical case. She did not work again and was admitted to Ilford Sanatorium on 1/1/45 where she died on 7/9/45.

Family History: 2 brothers and 2 sisters, all well.

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Clinical Appearances: Looked toxic and wasted, on admission, but was cheerful. Tachycardia. Intermittent temperature.

Heart: N.A.D. Lungs: dullness over whole of left lung anterior and posterior; also middle lobe. Adhesions left base. Rales widely disseminated in both lungs. Tubular breathing over middle lobe and pleural friction. On 16/5/45 dullness and post tussive creps were noted at both apices. No clubbing.

P.M.: Body emaciated. Left lung firmly adherent throughout. Cavities in left apex. Bilateral tuberculosis. Some dilatation of bronchi. Advanced asbestosis, tending to confluence at bases. Septic spleen. Liver normal. Heart, brain, kidneys, bowel: N.A.D.

Diagnosis: Asbestosis and Tuberculosis.

Case 5. G.H.S. Male. Aged 42.

History: Exposure commenced in Mattress Department on 1/4/19 where he remained for 3½ years. Transferred to Boiler Covering (outside contracts) for 5½ years. Returned to Mattress Department as foreman for 3 years. Was then diagnosed as suffering from asbestosis and tuberculosis (1931) at the London Chest Hospital. Became Outside Contracts Manager for boat work and remained in that position until August 1943. His wife was said to be suffering from pulmonary tuberculosis also. In December 1931 he gave a history of having had a "cold" 10 months

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before followed by persistent cough with some sputum in the morning. Six weeks before the sputum was blood-streaked for one day.

Clinical Exam: December 1931. Dyspnoea. No clubbing. Obvious loss of weight. Cyanosed. Chest movements poor on both sides, especially right. Impaired percussion note over both upper lobes in front and behind. Bronchial breathing right infraclavicular area. He was admitted to the National Sanatorium, Bournemouth for 6 months in 1933. On 1/3/43 clubbing of fingers and accentuation of 2nd pulmonic sound noted. B.P. 154/96. Empyema scar left side. Consolidation and creps right apex. Trachea displaced to right. Sputum +ve for tubercle bacilli and asbestosis bodies.

X-Ray, March 1943: Fibrotic, nodular T.B. Extensive fibrosis right apex with very marked traction of trachea to the same side. Cavitation left lower lobe. Calcified gland next to trachea. Right lung below apex emphysematous with some fibrosis (? asbestosis). Left apex showed air content but no lung structure. From clavicle downwards marked fibrosis and patchy infiltration diminishing towards the base. Heart much displaced to right and left border showed fine fibrotic bands.

He died on 2/2/44.

P.M.: A moderate degree of asbestosis rather more diffuse than usual. Massive fibrosis with some whorled nodules of silicotic type in right upper lobe. Congestive heart

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failure. Dilatation right heart. Ascites (2 pints).
Diagnosis: Pulmonary Asbestosis and Tuberculosis.

Case 6. R.W. Female. Age 41.

History: Spinner 25/7/18 to 15/6/21 and 8/8/21 to 10/7/24. Left her work to marry. Child born 1927 and was alive and well when patient died. A sister died of asbestosis in 1935. Patient had had a dry cough for several years but did not complain until, after an attack of mumps in 1939, she failed to recover completely. Dyspnoea became very troublesome and in 1940 she took to her bed. She was admitted to Black Notley Sanatorium in January 1941 and died 13/11/41.

Clinical and radiological data not available.

P.M.: Extensive, diffuse fibrosis. Asbestosis bodies ++ Bronchopneumonia. Thickened pleurae. No T.B. Healed T.B. mesenteric glands. Septic spleen.

Diagnosis: Asbestosis and Bronchopneumonia.

(b) Emphysema

Emphysema is so frequently a complication of asbestosis as in all pneumoconioses as to be almost a manifestation of the disease, although actually it is compensatory in character. It certainly plays a large part in persuading the patient to visit his doctor and with equal certainty assists the doctor to detect some pulmonary abnormality. It occurs mainly along the margins of the lungs where honeycombing and bullae may form, but the bullae never attain the size sometimes

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exhibited by silicotic lungs. As Merewether (39) has pointed out one of these bullae may burst and produce a spontaneous pneumothorax. No doubt the emphysema also contributes in the effects of back pressure. It was noted in 28 (30.1%) of the present series, but this is almost certainly an understatement.

Case 7. S.F. Male. Aged 42.

History: Jointing foreman 1921 to November 1929. In 1926 he suffered from a dry pleurisy. He returned to work but was "off colour", short of breath and had a dry cough. A second attack of dry pleurisy occurred in 1927. He gave up work in 1929 owing to weakness and dyspnoea. He was admitted to the London Chest Hospital on 30/8/30 with a right spontaneous pneumothorax and died on 13/10/30.

P.M.: Emaciated. Extensive asbestosis. Asbestosis bodies ++. Pleurae thick and adherent. Several emphysematous bullae. Honeycomb appearance at both apices, the fibrous septa being left with vacant spaces between them. In the right apex, immediately below the pleura there was a cavity ½" in diameter with smooth glistening walls ? due to coalescence of emphysematous bullae. Calcareo-caseous mass 1" in diameter behind gall-bladder; no giant cell systems or T.B.

Diagnosis: Asbestosis and Pneumothorax.

(c) Bronchiectasis

The complication of bronchiectasis seems to have been unduly prominent in the writings of the earlier workers.

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Ellman (40) states that he found clubbing of the fingers only in the more advanced cases of asbestosis "in which bronchiectasis was usually present". This is contrary to the writer's experience and seems to indicate a higher proportion of cases of bronchiectasis than was encountered in the present series. Merewether's opinion is that "in the absence of intercurrent infections the fibrosis may progress to an extreme degree; bronchiectasis, non-tubercular cavitation and spontaneous pneumothorax may occur". Elsewhere, Merewether (39) makes the statement that "in advanced asbestosis, bronchiectasis due to dragging and distortion by fibrous tissue is quite a common concomitant .....". Gloyne (48) seems to give the most accurate account when he says that "dilated bronchi are met with, but bronchiectasis with fusiform or cavity-like dilatations containing foul pus have not so far been encountered by the writer". In the present series of 98 post mortems, 4 cases with dilated bronchi were found (4.08%) and two cases exhibiting early bronchiectasis (2.04%). Curiously enough, clubbing of the fingers was not found in either case of bronchiectasis and only one case with dilatation of the bronchi had even slight curving of the nails.

Case 8. M.A.S.R. Female. Married. Aged 33.

History: Worked as a spinner from 20/3/22 to 21/9/28. During this period she had an illness resembling chorea. She married in 1929 and gave birth to a child the

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following year; the child was alive and well at the time of her death on 26/4/39. She was reputed to have suffered from cough and ill-health for some time but did not consult a doctor until February 1939 when she was referred to the London Hospital and admitted there for one month. The last phase of the illness began on 22/4/39 when she retired to bed with a temperature and an exacerbation of her cough.

Clinical findings: No clubbing. Basal bronchitis. Apex beat in 6th interspace 4½" from mid sternal line. Heart sounds closed. Lower edge of liver palpable two fingers' breadth below costal margin. Spleen moderately enlarged. Slight oedema of ankles.

P.M.: No wasting. Parietal and basal pleural adhesions both sides. Advanced asbestosis, reticular in upper lobes, generalised and diffuse in lower lobes. Some bronchiectasis in lower lobes. Asbestos bodies and fibres ++. Terminal bronchopneumonia. Myocarditis. Heart enlarged: weight 12 ozs. Thickening of mitral cusps. Liver: back pressure effects. Spleen: enlarged and septic. Kidneys: engorged. Small cyst left ovary. Carditis appeared insufficient to have caused death, but was no doubt a contributory factor.

Diagnosis: Asbestosis and early Bronchiectasis with Mitral Disease.

Case 9. B.R.B. Female. Married. Aged 30.

History: Worked as a spinner from 4/2/26 to 25/3/29. When examined on 15/3/34 she said she had had a dry cough

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since childhood and had been breathless on slight exertion for 2 years. She died on 3/12/36.

Clinical signs: Nails slightly curved. Fine squeaky rales and crackles both lungs. No wasting. Could only walk with comfort by "taking it steady".

X-Ray: Typical fine diffuse mottling in both lower zones with shagginess of the cardiac border. Blunting of costophrenic and cardiophrenic angles.

P.M.: No definite wasting. Adhesions and plaques both sides. Advanced, diffuse fibrosis with dilated bronchioles. No T.B. Pneumonia represented by red background to blue polygonal areas of fibrosis. Slight fatty degeneration of heart.

Diagnosis: Asbestosis and Pneumonia.

(d) Cardiovascular Complications

These are of two varieties: those such as are discussed under clinical signs in Chapter 4 and are consequent upon the disease and those which are not known to be related aetiologically to asbestosis. It is these latter cases to which reference will be made in this section.

In the post mortem series, one case, a woman (L.M.B.) aged 37 was found to have had endocarditis. There was no rheumatic history. A man (T.H.B.) aged 63 exhibited atheroma of the mitral valve, aorta and right coronary artery. He also suffered from asthma and bronchitis. Another man (W.W.C.) aged 68 showed advanced aortic

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disease and atheroma. The asbestosis was slight and was not the cause of death. His dyspnoea was probably referable to the aortic disease rather than to asbestosis. A mother and daughter appear in this group. The former (Mrs. M.M.F.) was aged 62 and her case history is given below. The daughter (Mrs. L.C.) was aged 38 and in addition to asbestosis had also a dilated, pale and flabby heart, fatty degeneration of the liver and pancreas and chronic interstitial pancreatitis.

Case 10. Mrs. M.M.F. Aged 62.

History: Employed in two periods 19/7/16 to 15/6/21 and 20/8/21 to 16/8/23 as a Mattress Maker except for a few weeks spent in the Weaving Department during the first period. She was found dead on her bedroom floor.

P.M.: Stout woman. Old dense adhesions on left side. Moderate asbestosis. Asbestosis bodies small. Chronic bronchitis. Myocardial degeneration. Right heart dilated. Marked fatty infiltration between muscle bundles. Infarct in spleen. Dilated oesophageal vessels at cardiac orifice probably accounted for blood stained fluid in stomach.

Diagnosis: Asbestosis, chronic bronchitis and myocarditis.

(e) Pregnancy

Questions as to the effects of pregnancy on the course of asbestosis or the effects of asbestosis on the woman in labour have not so far received any attention in the literature. Nor is information on these matters easily

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obtained. No doubt this is due in no small measure to the relative insignificance of the problem so far as the vast proportions of obstretricians is concerned. Also, women do not usually bear children during their active industrial lives, when they are in touch with the Factory Medical Officer, but after leaving employment. It probably occurs to few obstretricians to take an industrial history, especially during an emergency. The case is far different with tuberculosis which is widespread in all parts of the world and has been known to many generations. The evil results of pregnancy in association with this disease are so well known as almost to have become part of the folk-lore. Brief histories of 20 asbestotics who became pregnant at least once and later died are set out in Table No. 7.

The most striking feature in these histories is the large incidence of complications. Only two cases presented no complications. Five cases exhibited pulmonary tuberculosis, six cases had heart lesions and three had neoplasms. In five cases pregnancy coincided with a turn for the worse which led to a fatal issue.

Whatever metabolic changes may take place in the patient with asbestosis who becomes pregnant or in whatever way asbestosis resembles or does not resemble tuberculosis in this respect, there can be no doubt that a disease which is characterised by dyspnœa and sometimes cyanosis must be adversely affected by the strain of labour. The following

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patients are still alive and were questioned with regard to their pregnancies:-

F.B. Aet. 41. First employment 1921. Asbestosis. One child aged 17. Long labour due to cessation of pains. Otherwise no abnormality.

A.M. Aet. 36. First employment 1926. Advanced asbestosis. 2 children: normal confinements.

G.T. Aet. 41. First employment 1922. Recovered from pneumonia following normal confinement 1938. Moderate asbestosis.

R.E.W. Aet. 39. First employment 1927. Moderate asbestosis. 1 child: normal confinement.

F.M.H. Aet. 46. First employment 1917. Father died of pneumonia. She recovered from pneumonia 1945. 4 children: all normal confinements. Two died at ages 3 months and 16 years of septicaemia. Moderate asbestosis. Very dyspnoeic.

The suggestion is put forward that when the fibrosis is not far advanced and the right heart unembarrassed, labour may progress normally, but as the fibrosis increases and the strain on the right heart begins to be apparent the danger from depletion of cardiac reserve may be very great.

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TABLE No. 7

ASBESTOSIS & PREGNANCY

CASE	AGE	EXPOSURE	PREGNANCIES	DEATH	COMPLICATIONS FOUND POST MORTEM	REMARKS
A.E.A.	32	Spinning 28/8/23 to Jan./30	One living child. 4/12 pregnant.	1937	R. heart dilatation & failure. Nutmeg liver. Oedema dependent parts.	"I think she actually died of heart failure precipitated by the pregnancy, the whole being due to the fibrosis" - Pathologist's evidence at Inquest.
S.B.	27	Spinning 30/5/28 to 13/3/29 & 25/6/29 to 2/7/30	One, a son aet 10 at patient's death.	1938	T.B. cavitation both lower lobes. Terminal bronchopneumonia. Fatty degeneration of liver.	Left work for confinement which occurred 2/12 later. Attended T.O. as contact of her son who had T.B. adenitis.
F.B.	37	Weaving & Sectional. 5/1/19 to 28/1/21.	One child aet 6 at patient's death.	1935	Fibro-caseous T.B. Cavitation L. upper lobe.	Married 1920. Well until she became pregnant 1929 when she began to complain of cough & expectoration. T.B. found 1932.
K.B.	41	Viewer 1917 to 1919.	3 children born between 1919 & 1935.	1935	Abdominal cancer.	Onset of illness August 1935 with pains left leg & back. Died in December 1935.

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CASE	AGE	EXPOSURE	PREGNANCIES	DEATH	COMPLICATIONS FOUND POST MORTEM	REMARKS
L.C.	38	Weaving 24/11/14 to 28/4/21.	One daughter aet 15 at patient's death. Healthy.	1939	Heart dilated, pale and flabby. Fatty degeneration of liver and pancreas. Chronic interstitial pancreatitis. Oedema of legs. Ascites.	Patient's mother died of asbestosis, chronic bronchitis & marked fatty degeneration of the heart.
I.D.	42	Weaving 11/5/22 to 30/10/25.	6 children, including twins, born subsequent to 1925.	1942	Congestive heart failure. Myocarditis. No T.B.	1927 in hospital with pneumonia. 1941 in hospital with. wet pleurisy.
S.A.L.	37	Mattress 7/5/14 to 4/2/16 & 19/5/30 to 1/11/30.	One child aet 10 at patient's death.	1933	R.lung: extensive fibro-caseous T.B. upper lobe with cavitation. L.lung: T.B. cavity at apex with surrounding dense fibrosis.	Worked on munitions 1916 & married. Husband had T.B. laryngitis. Probably marital infection from him.
L.M.L.	41	Doubling 7/3/21 to 15/6/21 & 11/2/24 to 7/4/25.	1925 Caesarean Section for twins born dead.	1945	Glandular carcinoma R. ovary.	Married 1943.
M.E.M.	39	Opening 12/10/26 to 20/11/26 Mattress 2/6/27 to 28/12/29.	One child aet 4½	1945	Heart 13 ozs., chiefly hypertrophy. Coronaries normal.	Became dyspnoeic after her confinement. Acute respiratory illness 1943 left her with a persistent cough.

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CASE	AGE	EXPOSURE	PREGNANCIES	DEATH	COMPLICATIONS FOUND POST MORTEM	REMARKS
E.E.O.	32	Carding 26/1/26 to July 1930.	One child aet 4/12.	1939	Oat cell carcinoma super mediastinal glands. Ovaries enlarged & cystic.	
F.E.P.	33	Spinning 31/5/23 to 28/8/28.	Two children aet 7 yrs. and 5/12.	1939	Enlarged heart.	First became ill 1934 but not seriously until 1936. Remained in bed after last confinement until her death.
I.L.P.	40	Doubling 1923 to 1925.	5 children.	1945	Fibro-caseous T.B.	Onset of illness March 1938.
F.P.	33	Disintegrating 7/1/24 to 29/10/24.	3 children	1934	R.lung: large, multilocular cavity upper lobe with 2 or 3 ozs. of pus. A few patches of caseous T.B. apex R.lower lobe & lower part L.lower lobe.	Married December 1924. Pleuropneumonia Oct. 1929; several weeks in hospital. Admitted sanatorium early 1931.
A.M.R.	30	Spinning 13/5/24 to 28/11/28 & 27/4/29 to 15/3/30.	One child aet 7.	1939	Heart dilated and pale.	Onset of cough 1927. Married 1930. Confined 1932.
M.A.S.R.	33	Spinning 20/3/22 to 21/9/28	One child aet 9.	1939	Heart 12 ozs. Mitral cusps thickened. Liver: back pressure. Lungs: early bronchiectasis.	? history of chorea. Carditis insufficient to cause death, but was a contributory factor.
L.M.S.	35	Spinning 25/9/24 to 10/7/29.	One child aet 12.	1944	Right heart dilatation. Subacute nephritis.	No oedema.

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CASE	AGE	EXPOSURE	PREGNANCIES	DEATH	COMPLICATIONS FOUND POST MORTEM	REMARKS
G.E.S.	31	12/3/17 to 19/5/21 & Oct.1921 to Dec.1930. Slabs 2 yrs. Backing Asbestos shts. 2 yrs. Sack Sorter 5 yrs. Cloth trimmer 18/12.	One child aet 12.	1933	Heart 10 ozs.	Left work owing to "illness" and married same year. Onset of cough 1926 and dyspnoea 1928.
L.E.S.	35	Spinning 31/12/28 to 1933.	One child aet 4.	1944	Squamous carcinoma base of right lung.	Poor health since 1937, but had been very ill since birth of baby in 1940.
C.S.	29	Spinning 17/3/24 to 5/1/30 & 14/7/32 to 3/3/34.	One child aet 18/12. Early pregnancy.	1937	Pyonephrosis. Pneumonia. Renal calculi. Large cyst R. ovary.	Asbestosis found during first pregnancy at 4/12. Allowed to continue and Caesarean section successfully performed. Second pregnancy terminated aurgically and she seemed to go downhill very rapidly afterwards.
R.W.	41	Spinning 25/7/18 to 15/6/21 & 8/8/21 to 10/7/24.	One child aet 14.	1941	Nil	A sister died of asbestosis in 1935. Cough for several years but not severe until 1939. Unable to get about owing to dyspnoea for a year before her death.

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Case No. 11. C.S. Aged 29. Married.

History: Spinner 17/3/24 to 5/1/30 and 14/7/32 to 3/3/34. First seen by the writer privately. Attended London Chest Hospital 9/4/35 and admitted three days later. In-patient 12/4/35 to 16/6/35. Found to be suffering from asbestosis and four months pregnant. Latter allowed to continue and Caesarean Section carried out at St. Mary's Hospital, Stratford, London and living child delivered. Very protracted convalescence. Resumed attendance at London Chest Hospital D.P.D. 13/10/36 when Friedman pregnancy test found positive. Transferred to St. Mary's Hospital for interruption of pregnancy. Was now very dyspnoeic and emaciated and appeared much worse. After second operation she seemed to go downhill very rapidly and died 7/4/37.

P.M.: Poorly nourished. Pleural adhesions, thickening and plaques contained asbestosis bodies. Extensive reticular asbestosis. Asbestosis bodies ++. No T.B. Pneumonia at bright red stage. Patches of emphysema. Renal calculi and pyo-nephrosis. Right heart dilated and muscle thin. Spleen hard and fibrotic. Uterus healthy; no remains of pregnancy. Small cyst left ovary. Large globular cyst 3" diameter right ovary filled with liquid blood.

Diagnosis: Asbestosis, Pyo-nephrosis and Pneumonia.

Case No. 12. F.E.P. Aged 33. Married.

History: Spinner 31/5/23 to 28/8/28. Husband stated at

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inquest that his wife ceased work in the asbestos factory "because it wasn't doing her any good" and she went to a job as a margarine packer. First became ill 1934, but not seriously until 1936 and then diagnosed as suffering from asbestosis. First child was born in 1932 and a second child 5 months before the mother's death on 17/11/39. She kept to her bed during these last 5 months and was orthopnoeic. She had been admitted to a sanatorium, but as no signs of tuberculosis were found she was discharged. P.M.: Advanced asbestosis. Asbestosis bodies ++. Enlarged heart. Septic spleen. Pleura slightly thickened. Terminal bronchopneumonia.
Diagnosis: Asbestosis accelerated by bronchopneumonia.

(f) <u>New Growths</u>

Of the complications so far considered there is no great divergence of opinion as to the causal relationship, but the problem of asbestosis in the etiology of pulmonary cancer is highly controversial. Many years must elapse before cases are sufficient in number to be so statistically significant as to fulfill Bridge & Henry's conditions (41), viz. (1) Pulmonary carcinoma in the industrial environment under review must exceed the rate in the general population to a recognisable degree and (2) there must be sufficient exposure of the workers to an agent proved experimentally to be carcinogenetic. As will be seen, however, from the cases about to be cited, evidence already accumulated seems to favour a causal

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connection between asbestosis and pulmonary cancer and humanitarian motives may decide the public conscience not to wait for scientific proof before insisting on more stringent safeguards against dust inhalation.

The role of silicon dioxide in this respect enjoys a much better position in that much larger series of cases are available for investigation and, in spite of fallacies inherent in enquiries based upon death certificates and the variations in conclusions which can be drawn from them, the general concensus of opinion would seem to exonerate this substance from blame as a carcinogenetic agent. Thus Kennaway & Kennaway (1936) (43), whilst noting that Sheffield Metal Grinders show an excess of lung cancer amounting to two and a quarter times the standard rate, conclude: "The general indication of these results is that the factors which lead to silicosis are not very active in producing cancer of the lung and larynx". On the subject of a Sheffield analysis Turner & Grace (1938) (44) conclude: "Significant excessive mortality from cancer of the respiratory tract is marked in engineers, foundry workers and grinders and is seen in no other occupational group. With regard to lung cancer it is worth while to consider that the cancer excess in grinders may be dependent more on the less obvious iron component of the dust, than the more obvious silica content". Craw (42) has found pulmonary cancer in 5 out of 85 fatal cases of silicosis in haematite miners (5.9%). The vast statistical knowledge and

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experience of silicosis accumulated and accurately treated by the South African Medical Phthisis Bureau is especially valuable. Strachan, Pathologist to the Bureau, says that "Primary cancer of the lung does occur among miners both European and native but its relationship to silicosis is extremely ill-defined". The Schneeberg & Joachimstal mines have earned an unenviable reputation for lung cancer, but although silicosis is a hazard in those mines it is important also to realise that known carcinogenetic agents such as radioactive materials and arsenical dusts also operate. Finally, if the survey is spread cartographically, it is found that maps of silicosis and lung cancer here or abroad, are not superimposable.

In considering actual figures in this country, Bridge in the Annual Report of the Chief Inspector of Factories for the year 1938 gives some impressive data. Among 943 fatal cases of silicosis or silicosis with tuberculosis, lung cancer was found in 23 cases (2.4%). Of 347 post mortems carried out during the same period on persons who had been exposed to a silica hazard, silicosis was not confirmed but 17 cases (4.9%) revealed lung cancer. Similar incidence rates are found in a private communication for which the writer is indebted to Dr. Meiklejohn (45) of the Stoke Panel of the Silicosis Board. During the period 1st June 1931 to May 1939, 730 post mortems were personally investigated for alleged silicosis or silicosis with tuberculosis. In 546 cases silicosis

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was found and no evidence of the disease in 184 cases. Among the 546 silicotics were 12 cases of primary lung cancer (2.2%) and of the 184 cases without silicosis 11 cases of primary lung cancer (5.9%). As to age groups, approximately 80% of the cancer cases were 50 years or over in both groups whilst at 60 years and over the respective figures were 65% and 53% for silicotics and non-silicotics. Secondary growths were noted in four only of the 23 cases. Dr. Meiklejohn's conclusions are that "in the present state of knowledge there is no convincing evidence of any scientific kind to justify the assertion that silica plays any part in the etiology and development of primary lung cancer. When silicosis and primary lung cancer occur together they should be considered coincidental and unrelated. At the same time we should recognise that it may be necessary at a later date to alter this view".

Even apart from difficulties arising from paucity of statistical details in the asbestos industry, there are other factors common to all such enquiries, not least among these being the generally recognised increased incidence of pulmonary cancer both in Europe and in America among the general population. The striking upward trend displayed in Kikuth's graph of the incidence of primary bronchial carcinoma taken from the autopsy figures at the Eppendorf Hospital, Hamburg, between the years 1889 and 1923 is evidence of a very persuasive character.

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Similarly, the International Cancer Conference held in London in 1928 published figures of 0.94% of total cancer cases during the period 1903 to 1907 rising to 1.69% for the period 1923 to 1927. Percival Bailey (America) in 1923 placed the proportion as high as 7% to 10% and the age incidence between 40 and 60.

A further difficulty which is peculiar to asbestos and seems to have been insufficiently appreciated is an inherent chemical fallacy. Asbestos is not, like silicon dioxide, a precise chemical substance but a group better described as the asbestiform minerals. Because this group in which the silica is in the combined form constantly produces a specific fibrotic response in the pulmonary tissues under given circumstances and therein exhibits a similar quality to substances containing silica in the free or unchanged state, namely $Si\ O_2$, it is not possible to argue that all members of that group will or will not produce other effects on the tissues in the same degree or even at all. Even in so far as the fibrosing effects are concerned, it is well known that the short, dusty fibres of the amber coloured amosite are the most pernicious of all, that blue asbestos occupies an intermediate position both as regards wear on machine parts and effects on the lungs and that white asbestos is least harmful both to steel and to the human respiratory mechanism. If these effects are mainly due to physical factors, the chemical considerations in regard to cancer are much more diverse.

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Which, if any, is the carcinogenetic property or substance? Is it physical or chemical? Is it the siliceous portion or not? If it is the siliceous material, one would have expected a more pronounced carcinogenetic effect with silicon dioxide which is said to be slowly dissolved and to form highly toxic compounds with tissue proteins. On the other hand, the asbestiform minerals, although to some extent causing fibrosis of the mediastinal glands, do not choke and fibrose the lymph nodes but exert their baneful effects around the smaller bronchioles. It is obvious therefore that whoever undertakes the experimental proof of the carcinogenetic properties of the asbestiform minerals in accordance with Bridge & Henry's desiderata must be prepared to investigate not one but many substances.

But if statistical certainty is a matter for the future such evidence as exists can be supported by observed clinical and radiological factors. The result is a plain warning to use to the full such engineering wit as is available to suppress dust, whereby asbestosis certainly will be abolished and perhaps some cases of cancer prevented. Of all deaths in England and Wales between 1925 and 1935, 0.65% were due to pulmonary cancer as calculated from the Registrar General's returns (47). On the other hand, in the present series of 98 post mortems, 20 cases (20.4%) exhibited cancer in some organ or another; of these, 15 (15.3%) were primary intrathoracic growths. The extrathoracic cancers involved primarily the colon

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(2 cases), the pancreas (1 case), the ovary (1 case), and the other was described as "abdominal". That 4 of the 5 extrapulmonary cancers involved the digestive tract is perhaps not surprising seeing that they form the preponderance of neoplasms in the general population; but it may not be without significance that Gloyne identified asbestosis bodies in the faeces (48). Bridge (1938) (21) records 12 cases of pulmonary cancer in a series of 103 fatal cases of asbestosis or asbestosis and tuberculosis (11.6%), a rate which is not so high as the present series but sufficiently high as to call for comment. It may also indicate an increased cancer rate as the writer's figures include cases up to 1945.

The so-called aniline worker's papilloma of the bladder, the mule spinner's cancer, the chimney sweep's scrotal carcinoma, the pitch worker's atrophic skin, comedones, warty excrescences and rodent ulcers, the clay-pipe smoker's cancer of the lip, the asbestos worker's corns of the fingers and other evidence from occupation and otherwise, all lend weight to the belief that chronic irritation is an important factor in the etiology of new growths. The fibrosing activity of asbestos in the lungs, mediastinal glands and cardiovascular system has already been discussed and it may be of some importance to consider in more detail some special characteristics of the fibrosing process which precedes cancerous states. Industrial exposure to asbestos dust in the pulmonary

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cancers of this series was generally protracted; in one case it was as much as 42 years, although the shortest exposure was only 19 months. The average exposure, however, was 16.4 years as compared with an average exposure of only 10.6 years in 76 fatal cases of asbestosis without cancer. The primary growth was found in the left lung in 9 of the 15 cases, in the right lung in 3 cases, in the superior mediastinal glands in 2 cases and in the pleura in 1 case. This reverses the general experience of lung cancers and might be accounted for by the observation made by some workers, notably Pendergrass's earlier opinion, that asbestosis usually begins in and develops a greater extent in the left lung than in the right. (Later, Pendergrass refused to commit himself on the question, a position with which the writer is in complete sympathy). The case seems clearer, however, when the position in the lung of the primary site is considered. It was discovered in the lower lobe in 6 cases, in the middle lobe in 1 case and in the upper lobe in 3 cases. Again, this is contrary to general experience but may be related to the more intense fibrosis at the bases. The average age at death for all cancers in the series was 50.9 years and 52.5 years for intrathoracic growths. The extreme ages were 32 and 71 for all cancers. Whilst it is true that most of the cases might fall naturally into the cancer age groups of the general population, the mere fact of

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age affords longer exposure to dust. Co-existent tubercle was found in 1 case (M.M.S.).

In any discussion of this kind, the co-existence or otherwise of tuberculosis with lung cancer should be indicated in the statistical survey. Maurice Davidson's series of 107 autopsies on cases of lung cancer (46) showed an incidence of co-existent tuberculosis in 6.5%, a ratio which he points out is higher than usual. Nevertheless, deaths due to pulmonary tuberculosis are very inadequately investigated. Post mortems are exceptional with the result that the final pathological phase is not precisely shown and the association of pulmonary cancer and overt tuberculosis insufficiently explored. In some circumstances tuberculosis and cancer are indistinguishable or again the cancer may lie in some remote bronchus masquerading as a small blood clot. The growing use of the bronchoscope has tended to suggest (inaccurately) that bronchial cancer is related only to the bifurcation of the trachea or its main divisions as the sites of election. Lung abscess, also, has concealed many a cancer from the unwary. In the last investigation, a mere sweep of the pathologist's knife through each lung is insufficient: each available bronchus should be slit up and the search is still incomplete without competent histological examination.

One case, 7.1% of the series, showed secondaries in the cerebellum. The comments of Carnegie Dickson &

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Worster-Drought in their communications to the Neurological Section of the Royal Society of Medicine 1934-5 on 6 cases of lung cancer with cerebral metastoses form an interesting parallel with the present series. Their first case, that of a man aged 55, was found to have a tumour measuring 30 mm. x 30 mm. in the left tempero-sphenoidal lobe together with secondaries in other parts of the brain. "On palpating the root of the right lung, there was a firm nodulated mass suggestive of a possible tumour, but found to be due mainly to an old fibrotic condition spreading outwards into the lung tissue from the root. On dissection, the eparterial bronchus was found to be the seat of a stenosing carcinoma, and the surrounding lung tissue showed a mixture of fibrosis resembling a silicosis (the patient's occupation had been that of a plasterer) and the infiltrating malignant tumour". The case is reminiscent of Ramazzini's maidservant, the employee of a plasterer, who coughed up a lump of lime, but may nevertheless have as much value as the observation that calcified tuberculous foci are sometimes expectorated by others besides plasterers' maidservants. The chances of a plasterer contracting silicosis are remote but the significance of one type of tissue proliferation passing into another type may be considerable.

All their cases were males, their ages varied between 43 and 64 years, the average being 53 a figure

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almost exactly the same as in the writer's series. The usual proportion in the sexes is 4 or 5 males to 1 female. (In the writer's series the proportion of females was much higher, namely 8 to 6. This difference is easily accounted for by the fact that the asbestos industry very largely employs female labour). Chest symptoms found were inconspicuous or practically absent in all but two cases. On symptomatology, they quote Simpson in calling attention to general wasting, dyspnoea, bronchitis, haemoptysis, hoarseness, offensive breath (bronchiectasis) and leucocytosis. It is plain that if such symptoms are imposed upon those of asbestosis the quality of the disease is little altered and only the momentum undergoes a grave change. It is the writer's experience that where, in a case of asbestosis, the wasting and dyspnœa suddenly become worse and out of proportion to the degree of fibrosis and if clubbing of the fingers is marked, carcinoma has almost certainly imposed itself notwithstanding negative evidence from the X-Ray film.

They consider that lung cancers have a marked tendency to produce metastases in the brain and quote figures of 37%, 41%, and 47%, these secondaries being found mainly in the frontal and temporosphenoidal lobes, the cerebellum and in the choroid plexuses. The suprarenal medulla is also a frequent site. (Noted in 1 case in the present series, Case E.E.O. in the table

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below). Blood pressures were uniformly low, the highest systolic and diastolic pressures occurring in one man aged 47 with readings of 130/96. The contrary was the writer's experience.

Two types of intrathoracic carcinoma are ordinarily described as being found in association with silicosis or asbestosis. These are (1) Growths involving the mediastinal glands originally considered by Adler as lymphosarcomata. They are white and rapidly growing, occurring in the glands at the bifurcation of the trachea and spreading along the two main bronchi. The cells are described as of the oat cell type and the origin is unknown, but may be from undifferentiated bronchial basal epithelium. It grows into the substance of the lung as a massive tumour. About one half of new growths of the lung are said to be of this type. (2) The other variety is the columnar cell type which appears to arise from the bronchial epithelium and extends along the bronchi pushing the pigment aside and sometimes enclosing asbestosis bodies in its substance. Necrosis and abscess formation frequently occur with this type thus obscuring the diagnosis. (3) A third type is also described, the squamous cell variety which seems to accompany tuberculosis where the bronchial epithelium has undergone this type of metaplasia. An endothelioma of the pleura is described in the present series, the first to be recorded in association with asbestosis, so far as

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the writer is aware. Of the remainder, 6 were squamous carcinomata, 5 oat cell, 1 columnar cell and 1 unknown. The case with re-activated tuberculosis (M.M.S.) showed also a squamous carcinoma. Metastases were present in 6 of the cases, 3 from primary oat cell growths and 3 from primary squamous cell growths and involved the pericardium, abdominal viscera, a dorsal vertebra, breast and brain.

Two cases will serve to illustrate the co-existence of pulmonary cancer and asbestosis, in neither of which cases was cancer suspected before death. Indeed, in no case of this kind known to the writer has the cancer been diagnosed before death, although it was suspected in two cases. In one of these latter cases the neoplasm attacked the hepatic flexure of the colon (Case R.W.) and therefore should not perhaps have presented the same difficulties of diagnosis as a pulmonary neoplasm. Prolonged, competent and thorough investigations, however, failed to explain all the patient's symptoms during life. The fibrosis attacked the upper lung fields and was accompanied by emphysema at the bases, depicted as such in the skiagram and shown to be so at post mortem.

Case 13. L.E.S. Female. (Tables 7 and 8).

History: Was transferred from Spinning Department to Sectional Office in 1933 because of dry cough and dyspnoea. Left to marry in 1936. A baby was born in 1940 and "afterwards she was very ill". In November

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1936 she had chest pains ascribed to bronchitis. In February 1944 she had an attack of pyelitis (symptoms referable mainly to left side) with coliform organisms in the urine. She was admitted to hospital, treated with sulphonamides and appeared to recover. On 10/8/44, whilst walking in her garden, she collapsed with a severe pain in the right side and was admitted to another hospital. She had been losing weight rapidly. Rib resection was performed on 4/9/44 and a lung abscess opened and satisfactorily drained on 11/9/44. Her condition, however, appeared to be degenerating in spite of apparently successful treatment and she was transferred to the London Chest Hospital where she died.

X-Ray: Typical asbestosis. Much thickening around base of abscess cavity.

Clinical: Clubbing of fingers and toes +++. Marked emaciation. Adventitious sounds in chest consistent with asbestosis.

P.M.: A squamous carcinoma of the bronchus occupied the floor of the lung abscess cavity. There were secondaries in the left kidney and pus in the pelvis of the kidney, as well as a secondary in the liver.

Diagnosis: Pulmonary Asbestosis and Carcinoma.

Case 14. C.V.W. Male. (Table 8).

History: Was off work for 3 or 4 weeks in 1933, certified pleurodynia, and for a similar period in 1934 certified as bronchial influenza. First complained of dry cough and

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dyspnoea in 1941. Died 23/8/43.

Clinical: 21/4/41. No abnormality found in chest. B.P. 168/128.

X-Ray: Mild asbestosis 1/6/42: pain left chest and shoulder and "chilliness" of 1 week's duration. T. 99.0°. Wide area pleural friction left base. 15/6/42: returned to work on own doctor's instructions. T. 97.0°. Pain easier. Dry cough. Pleural friction still present. 31/12/42: dry cough, dyspnoea and weakness of the legs. Obvious loss of weight from 10 st. 10 lbs. to 8 st. 6 lbs. Clubbing +++. Impaired resonance left base. 21/1/43: pain under left clavicle and over left scapula worse on breathing. Impaired resonance left base, no adventitious sounds. Pleural effusion aspirated on admission to London Chest Hospital and found to show high lymphocyte count. Injected into guinea pigs produced no effect. Pneumoperitoneum seemed to indicate a mass below the diaphragm associated with the spleen. Blood count: microcytic anaemia with leucocytosis. Sputum: asbestosis bodies +.

X-Ray: Fine fibrosis right lower zone and some lesion in left lower zone. Right diaphragm indistinct and left costophrenic angle obliterated. Later films showed a large effusion at the left base.

P.M.: Asbestosis not very advanced. Endothelioma of pleura.

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The case with cancer of the pancreas (J.G.B. Table 8) would seem to call for comment in view of Turner & Grace's observations. They found an excess of cancer of the pancreas in two groups of workers, the engineers and those engaged in precious and non-ferrous metals. They suggest that iron is the common factor in the first group and copper in the second group. They further point out that the reticulo-endothelial system is richly represented in the liver and pancreas and that this system is intimately associated with iron metabolism. In support of their theory they cite the rare disease haemochromatosis in which iron accumulates mainly in the liver and pancreas due to a failure in the excretory mechanism and that symptomatology springs from the severe cirrhosis of the liver and pancreas caused by the accumulation of iron. Primary cancer of the liver is said to occur more frequently in haemochromatosis than in simple cirrhosis. Mallory believes (perhaps on inadequate grounds) that haemochromatosis is the result of chronic copper poisoning extending over a period of years. Their conclusions are that there are grounds for suggesting that the high incidence of cancer of the pancreas in engineers is intimately connected with excessive absorption of iron and in workers in non-ferrous metals copper is responsible, notwithstanding the Registrar-General's classification of the pancreas as an "inaccessible" site.

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The Serpentine Group of asbestos minerals as shown above is composed mainly of silicates of magnesia, whilst the Rhombic and Monoclinic Amphiboles contain large quantities of iron. Exposure of the respiratory tract to these last two groups in the form of dust might therefore cause the ingestion of iron in excess of the normal daily intake of 10 - 30 mg. But a further factor may operate in asbestosis because of the formation of asbestosis bodies. The deposition of iron on fibres in this process may well upset the iron metabolism of the pancreas.

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TABLE No. 8

P.M. Reports on Cases of Asbestosis & Carcinoma.

CASE	AGE	SEX	EXPOSURE	DEATH	HISTOLOGY	SECONDARIES
P.B.	49	M	Disintegrating & Sectional 30/6/24 to 1932. Ovens 1932 to 16/12/36.	27/6/37	Squamous carcinoma of bronchus with keratinisation involving lower part left upper lobe.	Pericardium Liver, Left kidney.
J.G.B.	50	M	1906 - 1931 Mixing boiler composition: 5% asbestos + fossil meal + China clay. Fiberising plant on same floor.	11/6/31	Acinous cancer of abdomen primary probably in head of pancreas where yellowish nodule size of walnut.	Peritoneum, omentum, mesentery & viscera in a dense mass. A few small deposits in pleura.
K.B.	41	F	1917-1919. Viewer.	Dec. 1935	Abdominal cancer.	
E.C.	69	F	Mattress Dept. 8/8/21 to 21/9/33.	18/8/45	Oat cell carcinoma 3" x 2" lower lobe right lung. No invasion by mucus.	One only, in liver.
W.T.E.	47	M	Tinsmith 1916-1936	15/11/40	Columnar celled carcinoma of colon.	Abdominal viscera matted. Complete symphysis pleura due to secondary growths.

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CASE	AGE	SEX	EXPOSURE	DEATH	HISTOLOGY	SECONDARIES
A.C.G.	59	M	Packer. Stores Foreman 29/7/12 to 7/3/33.	14/9/33	L.lower lobe peripheral half.Pinkish white,soft & fairly well marked off oat cell carcinoma of racemose distribution.	Nil found.
J.G.	55	M.	Stores 1930 - 1938.	1945	Squamous Carcinoma Lt. upper lobe.	Nil.
A.C.J.	58	M	2/1/1894 to 1931. Works Manager.	July 1936	Squamous Carcinoma left lung.	Records destroyed by enemy action.
L.M.M.	41	F	Doubling 7/3/21 to 15/6/21 & 11/2/24 to 7/4/25.	10/12/45	Glandular Carcinoma Rt. ovary.	Entire abdomen filled with growth, intestines running through its depth.
G.A.M.	45	M	Lagger 1904 to 1934.	26/4/34	Squamous Carcinoma size of tangerine & circum-scribed in base of R. lower lobe. Necrotic centre.	In 9th dorsal vertebra & in muscles & fascia on R. side. None in cord. 2 to 3 months old, causing retention of urine and chronic bladder infection.
R.M.	59	F	1912 to 1921.	Aug. 1942	Greenish white glandular cancer of columnar cell type in R. middle lobe involving pericardium & wall R.auricle. Surrounds R. middle lobe bronchus.	Nil seen.

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CASE	AGE	SEX	EXPOSURE	DEATH	HISTOLOGY	SECONDARIES
E.E.O.	32	F	Carding 26/1/26 to July 1930.	5/5/39	Large mass of oat cell carcinoma in supra-mediastinal glands & nodules in both bases.	Left breast diaphragmatic pleura. Liver Spleen L. suprarenal. Both kidneys mesenteric glands.
J.P.	62	M	Opening & Disintegrating 1917 - 1940.	1940	Oat cell carcinoma of left lung.	Nil found
M.M.S.	71	F	Mattress 14/11/15 to 9/5/16. Opening 21/1/18 to 14/2/19.	12/12/34	Emphysematous cavity in apex of L.lung.Rest of lobe necrotic with grey irregular masses breaking down into small cavities.Squamous carcinoma.Chronic T.B. mediastinal glands - an old infection re-activated.	Nil found
L.E.S.	35	F	Spinning 31/12/28 to 1933. Sectional (Office) 1933 to 1936.	Oct. 1944	Squamous carcinoma of bronchus in base of L. lung.	Left kidney. Liver.
E.H.T.	55	M	Crude Stores 17/8/21 to 1936. Yard 1936 to 27/10/39.	30/12/40	Squamous carcinoma L.lower lobe, still small.	Nil found.
C.V.W.	42	M	Greaser & Beltman 2/8/21 to. June 1942.	23/8/43	Endothelioma of pleura on L. side.	Nil found.

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CASE	AGE	SEX	EXPOSURE	DEATH	HISTOLOGY	SECONDARIES
R.W.	50	M	Plaiting Foreman 3/4/22 to 1934. Brakelining Foreman 1934 to 2/6/42. Asst. Plant Manager 2/6/42 to 29/3/45.	29/3/45	Acinous carcinoma of hepatic flexure of colon.	Nil found.
L.W.	39	F	Sectional 29/7/21 to 9/9/32	Apr. 1939	Small malignant growth plugging the bronchus. Squamous carcinoma of left lower bronchus.	Nil found.
A.W.	58	M	Boiler Coverer 1896 to 1938.	1938	Oat cell carcinoma mediastinal glands of L. bronchus extending along pulmonary vein to base of pericardium.	Lumbar glands. Spleen. Cerebellum (numerous).

If it be granted that there is a causal relationship between asbestosis and cancer, the factors present may be summarised as follows. (1) There is a general, but not a special tendency for cancer to occur most in those sites where fibrosis is most likely to occur or to which asbestosis bodies are accessible. (2) Pulmonary fibrosis due to asbestosis is a progressive tissue proliferation even after withdrawal from the causative dust, but interjected infection is probably necessary. (3) Cancer cannot be shown to have any causal relationship to the inhalation of silicon dioxide

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alone. (4) There are excess cancer rates in those exposed to iron dust, silicon dioxide mixed with iron oxide in haematite miners and in workers in silicates combined with metals, of which iron is one of the chief. (5) There is some evidence to the effect that cancer of the pancreas may be due to a defect in the iron metabolism of the reticulo-endothelial system.

The theory is therefore, put forward that irritation of the lung parenchyma with silicous material whether by physical or chemical means is only sufficient to cause fibrous tissue proliferation and does not of itself cause neoplastic changes. Except for the one case of endothelioma of the pleura (? carcinoma) all new growths so far noted have been carcinomata. It therefore seems necessary to postulate a metabolic factor which, although it does not accelerate the fibrous tissue proliferation, finds a suitable nidus for attacking epithelial cells in those regions which are affected by fibrosis. It is possible that fibrosis may not be necessary to the atavistic process because cancers are known to accompany asbestosis when the two processes occur in different organs and also cancer may accompany excessive iron or copper ingestion in cases where no pulmonary fibrosis is present, but it is an accelerating factor.

(g) <u>Other Complications</u>

Bright's Disease has been associated with Silicosis by Collis, but it is difficult to see how the association can

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be anything but fortuitous. There are, however, three cases in the present series, one of subacute nephritis and the other two of interstitial nephritis, one of which also exhibited fatty degeneration of the liver. There are three cases of lithiasis, two of the gall-bladder and the other renal. One case showed chronic pancreatitis, another gastric ulcer and there are two cases of asbestosis and silicosis existing together in the same persons.

Case 15. L.M.S. Aet 35. Female. Married.

History: Spinner 25/9/24 to 10/7/29. Healthy child born 1932, 3 years after asbestosis diagnosed. On 29/3/44 complained of dyspnoea and palpitation and was sent home with instructions to stay in bed. The cough became more troublesome and was non-productive. During the next 10 days, the dyspnoea increased and before she became unconscious her main complaint was of weakness. Drowsiness developed into semi-coma but she was able to take fluids almost to her death which occurred on 27/4/44.

Clinical Signs: Much wasting. No clubbing. Few abnormal signs in chest. Urine: albumen +++.

X-Ray: Bilateral basal fibrosis.

P.M.: Advanced asbestosis. Heart small, muscle thin and right side dilated. Early bronchopneumonia. Spleen septic. Kidneys: sub-acute nephritis.

Diagnosis: Asbestosis and Sub-acute Nephritis.

Case 16. E.G.A. Aet 51. Female. Married.

History: Opening Room 28/3/18 to 7/8/18. Mattress Dept.

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5/4/27 to 4/5/27 and (?) one month in 1926.

Cough commenced 1931. Died 15/11/40.

P.M.: Advanced asbestosis. Dilated right auricle. Pale, fatty heart muscle. Fatty degeneration of liver. Interstitial nephritis. Bronchopneumonia.

Diagnosis: Asbestosis & Bronchopneumonia.

The case showing renal calculi has already been described in the section on pregnancy (Mrs. C.S.).

Case 17. H.L. Male. Aet 54.

History: Cardroom 1920 to 1929. Cough and loss of weight 1925. Cough became worse 1929. Pleural effusion right side 17 months before death on 13/2/33.

Clinical Signs: Poor chest movement. Right side collapsed. Creps right base.

P.M.: Left side: tough adhesions. Right: tubercular empyema with 1 pint of pus. Diaphragmatic pleura adherent. Asbestosis bodies ++ and fragmented. Streps, B. Proteus and T.B. bacilli +. Small caseous deposits in peripheral parts of right lower lobe and left apex: peribronchial and perivascular. Surrounded by connective tissue but very little round celled infiltration. Typical asbestosis. Old healed calcareous T.B. mesenteric glands. Heart soft and flabby. 50 to 100 stones in gall-bladder.

Diagnosis: Asbestosis and terminal Tubercular Empyema.

Case 18. L.C. Female. Aet 38. Married.

History: Weaver 24/11/14 to 28/4/21. Child born 1924 and healthy at mother's decease on 24/1/39. Patient's mother

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worked in same department and died of asbestosis in 1936.
P.M.: Oedema of legs. Adherent pleura and pericardium. Moderate ascites. Asbestosis bodies +++. Reticular fibrosis in upper lobes, diffuse in lower. Heart dilated, pale and flabby. Fatty degeneration of liver and pancreas. Chronic interstitial pancreatitis. Small septic foci kidneys (terminal). Small uterine fibroids.
Diagnosis: Cardiac failure and Myocarditis due to Asbestosis.

Case 19. E.H.T. Male. Aet 55.
History: Crude Stores 17/8/21 to 1936. Yard: 1936 to 27/10/39. Sister stated at inquest he had had "a terrible cough since 1933". Certified asbestosis 1936. Died 30/12/40.
P.M.: Wasted. Diffuse reticular fibrosis. Thickened pleura with collagenous fibres. Asbestosis bodies +. No T.B. Terminal bronchopneumonia. Squamous carcinoma left lower lobe. Still small and no secondaries. Heart dilated, right side thin and pale. Ulcer ½" diameter pyloric end of stomach, not malignant.
Diagnosis: Asbestosis and Carcinoma.

Case 20. H. Male.
History: 16 years quarry hone cutter. 14⅓ years bag carrier and general labourer in an asbestos works. Died 1934. Dr. Merewether in a private communication describes a similar case he examined in 1928 who had been exposed to a silica hazard as well as 8 or 9 years' exposure to asbestos. The X-Ray appearances showed lesions intermediate between

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silicosis and asbestosis.

P.M.: (1) Silicotic whorls. (2) Calcareous nodules. (3) Caseous tubercles with giant cells and enormous numbers of acid fast bacilli. (4) Reticular fibrosis and asbestosis bodies. The first was the oldest lesion.

Diagnosis: Silicosis, Asbestosis & Tuberculosis.

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CHAPTER 6

MANAGEMENT

Prevention of the disease having failed, the patient certified by the Board and excluded from employment in the scheduled processes, there still remain the problems of management of the case. Prevention is still the keynote, but now it is the prevention of complications, the respiratory infections especially. For if the experimental evidence is of value, there is reason to suppose that the disease will not progress except in the presence of infection. It is not possible under ordinary conditions of civilised life so to isolate a person that he runs no risk of infection from his fellows: most would prefer the death of the body to the killing of the soul. The patient should therefore be instructed to retire to bed immediately on the first signs of a "cold" and to remain there until he is considered out of danger. He should take steps to isolate himself from those of his family and friends who may be suffering from such a condition. Generally, he should avoid crowds. Often the history will reveal a permanent deterioration in the general condition following an attack of "bronchial catarrh" or "influenza". A patient will state that breathlessness dated from such an event, cough became really troublesome or he took to a bath chair. Such a complication should therefore be avoided, not only on account of its danger to life; but because of its crippling effect upon such of those who recover.

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Pneumonia is the proximate cause of death in the majority of cases of asbestosis. This fact should not, however, encourage an attitude of despair in the attendants. Six cases of recovery from pneumonia in asbestotics are known to the writer, four of them before the use of sulphonamides became general.

Case 21. I.D. Female. Aet 42. Weaver 11/5/22 to 30/10/25. Pneumonia 1927. Death from asbestosis and congestive heart failure 23/1/42 (P.M.).

Case 22. W.F.H. Male. Aet 23. 1927-30 mixing asbestos and kieselguhr by machinery. August 1930 to October 1930 carding. Off work with pneumonia November and December 1930. January 1931 to September 1931 despatch warehouse. Then left the industry and worked in building trade until December 1934. From 20/12/34 to 21/2/35 returned to mixing asbestos compound. On that day he was taken ill whilst on his way home from work with what appeared to his doctor to be influenza. On 7/3/35 he was admitted to hospital delusional and suffering from bronchopneumonia. He died on 9/3/35. Diagnosis: Asbestosis and bronchopneumonia (P.M.).

Case 23. F.M.H. Female. Aet 46. Mattress Department 1917 to 1920. Father died of pneumonia 1917. She was treated for lobar pneumonia at left base with sulphathiazole in January 1945. Long convalescence and has been very dyspnoeic ever since. Before that, she had only been troubled with a dry morning cough and breathlessness on exertion. Now (a year later) cannot walk more than a few yards.

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Case 24. I.H. Female. Aet 26. Spinner 1918 to 1923. Treated in London Chest Hospital for pneumonia in 1920. Died of typical advanced asbestosis in December 1935.

Case 25. F.P. Female. Aet 33. Disintegrating Department 7/1/24 to 29/10/24. Treated in hospital for pneumonia October 1929. Admitted to sanatorium early 1931. Died 20/1/34.

Diagnosis: Moderate asbestosis and extensive bilateral T.B. (P.M.).

Case 26. A.O. Male. Aet 55. Loom tackler in Weaving Department. Certified asbestosis by Board 1/5/44. Pneumonia January 1944 treated at home with sulphapyridine. Died of asbestosis 13/11/44.

The type of pneumococcus and nature of other causative organisms determine, to some extent, resistance to chemotherapy. Organisms other than the pneumococcus such as micrococcus catarrhalis, bacillus proteus, Pfeiffer's bacillus, pneumobacillus of Friedlander, staphylococci and streptococci have been isolated. As in uncomplicated cases of pneumonia, it can be expected that certain strains of pneumococci and streptococci will respond to sulphonamides, the Gram negative cocci will be lysed by penicillin and b. proteus and the viruses will remain resistant to both types of therapy. Serious though the complication of pneumonia was and is, it seems reasonable to hope that the newer chemotherapeutic substances will improve the prognosis generally. That being so, full therapeutic doses should be

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administered.

The optimistic theory of some years ago that there is a tendency for occupational pulmonary fibrosis to overwhelm a tubercular process in the lungs has proved to be quite without foundation. Treatment of silicotuberculosis is, generally speaking, the treatment of tuberculosis except that the prognosis is in all cases hopeless. Sanatorium treatment is therefore not justified. Open cases must be instructed in methods of sputum disposal and other precautions against infecting others and perhaps nursed in a hospital for chronic cases of tuberculosis. The sulphones have given equivocal results in the treatment of tuberculosis and they are not without danger. Similar results may be anticipated in silicotuberculosis.

On the difficult question as to whether artificial pneumothorax should ever be carried out on the asbestotic with a tubercular infection, experience is limited to one case which terminated unhappily. Pleural adhesions are very common in asbestosis and the advice also of Maurice Davidson (46) on the selection of suitable cases for the induction of artificial pneumothorax would appear to be applicable to many cases of this disease. "Complications such as cardiac failure, asthma or renal disease, or in fact any condition in which severe dyspnoea is present or is likely to occur, should be regarded as definite contra-indications". Burton Wood's statement, already quoted, emphasising the monosymptomatic character of asbestosis

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(dyspnoea) serves to give point to the argument against this form of treatment. Right heart embarrassment and emphysema to a more or less degree can safely be assumed in many cases and cause the intending operator to give the matter more than ordinary consideration.

Case 27. W.A. Male. Aet 18.

History: Van boy 18/6/28 to June 1931. On days the van did not go out he mixed an asbestos and magnesia composition with a shovel and filled it into bags. He was admitted to the London Chest Hospital on 23/6/31 with 4 month's history of cough and dyspnoea. Tubercle bacilli and asbestosis bodies present in sputum. Artificial pneumothorax and adhesion cutting carried out on right lung. The collapse was unsuccessful. Pyopneumothorax intervened and he died on 5/7/32.

P.M.: Right pleural sac loculated; several ounces of pus. Sinuses present. Ragged, multilocular cavity in upper lobe right lung. Peribronchial asbestosis deposits in the lower lobe and numerous caseating peribronchial tubercular deposits. In the left lung there were similar deposits and a few larger patches of caseation in the upper lobe. Myocarditis. Amyloid liver and kidneys and a few tubercles in these organs. Asbestosis bodies thin and crenated as if iron deposit small.

Diagnosis: Asbestosis and Tuberculosis.

Dusting workers with aluminium powder has recently received much attention in connection with silicosis.

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Whatever may prove to be its merits in that disease it is hardly likely that it will be of any use in asbestosis. In the former case it is contended that deposition of an insoluble, non-toxic aluminium silicate on the surface of the silica particle may inhibit the effects of silica. This is dependent upon the theory already stated that the particles enter the lymph stream and choke the lymph nodules where gradual solution stimulates fibrosis. The position is quite different with the asbestos fibre where no chemical action is postulated. It is unlikely that aluminium dust would interfere with the mechanical action of the frayed ends of asbestos fibres on the bronchioles and no such claim has yet been advanced. On the other hand in vitro experiments have shown that aluminium depresses the solubility of asbestos.

In the treatment of cardiac complications distinction must be made between those failing right hearts and other consequences of fibrosis of the lungs and conditions not so associated etiologically. Rest in bed and perhaps venesection are the only measures likely to do any good in the first type of case. Should, however, fibrillation be present or some other cardiac condition unrelated to the fibrosis, then digitalis or other appropriate measures may be taken.

Some ante-natal clinics already make inquiries regarding exposure of mothers to benzene. It would be useful if complete inudstrial histories were taken. This is the more

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important in the asbestos industry where most of the workers are females, and also in view of the large influx into industry generally of female labour. Lead, mercury, radio-active materials, chlorinated hydrocarbons and others besides asbestos may have a bearing upon the pregnancy and course of labour. In the case of asbestosis, it is the writer's opinion that the obstretrician should think rather of the heart than the lungs and according to the capacity of that organ as judged by dyspnoea, cyanosis, blood pressure, shape of the cardiac silhouette and hilar shadows as well as the usual clinical signs of failure, decide whether to allow the pregnancy to continue and terminate by forceps or Caesarean section or carry out induction of premature labour. The condition of the chest will decide the type of anaesthetic, but generally ether should be avoided. Sterilisation is probably advisable. There are no indications of asbestos having a toxic effect upon the liver.

The psychological aspect of asbestosis is of the greatest importance. It is only in the pneumoconioses that patients are given an official certificate to inform them of what is, in most cases, a warning of dissolution in the not very distant future. Every effort should be made, consistent with the truth, to soften the blow and one of the best means of doing so is to rehabilitate the injured workman. If possible, he should be put to work among his fellows so as to convince him that at least his working, useful days are not over. He does not then depend only

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upon compensation eked out by the charity of a sympathetic Management, but preserves his self respect and an element of independence which is strengthened by such allowances as those to which he is entitled by statute. He cannot, of course, be permitted to work at an occupation which exposes him to asbestos dust. It may be argued that the presence of such patients in an asbestos factory has a depressant effect on the other workers and that therefore they should be suitably compensated and retired. The argument is somewhat reminiscent of the ecclesiastical error of simony, in that it recommends, in effect, that a moral obligation can be remitted on payment of money. That there is a moral obligation is the modern view and would seem to be the correct view.

The only other alternative is to place the person in another factory altogether. But industry is not organised on such a national scale as to permit this dispersal of asbestotics. The quotas which seem to be envisaged by the Disabled Persons (Rehabilitation) Act (52) are too small to cope with such a situation. Most employers will have little difficulty at present in making up their quotas by disabled persons they know and have chosen and will strive to avoid cases of incurably, progressive and fatal disease. Moreover, dispersal takes the asbetotic away from the clinic supervised by the industrial medical officer at the asbestos factory so depriving him of the knowledge and experience of a follow-up system and consigns the patient to those with

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little or no special experience of the disease. Factories in which a large proportion of pneumoconiotics are employed have already started. Asbestotics do not do well in sanatoria and run an unnecessary risk both there and at tuberculosis clinics. Ideally asbestotics should be treated at a hospital which has an industrial medical department to care for these patients.

The cynic has said that the treatment of Koch's infection is financial. To a less degree, but in something of the same manner, this is also true of industrial pulmonary fibrosis.

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CHAPTER 7

CONCLUSIONS AND SUMMARY

The preventive measures as laid down by the Regulations of 1931 seem to have been effective, but only up to a point. Three cases are known to have occurred in employees who commenced work in the industry subsequent to that date. In other cases present conditions have contributed to the development of the disease. As a result of legislation, biological changes have occurred in the disease itself. The original type of acute disease which killed in a year or two after an exposure of a few months, has given place to a disease which is protracted and consequent on many years of exposure. Hence the stigmata of chronicity have increased together with the evil genius of chronic irritation. If the acute disease has merged into the chronic, is the chronic disease merging into the neoplastic? No answer can be given at present.

The question arises as to whether more legislation is necessary. Encouraged by its present success, there is a temptation to add to the already existing corpus of law. This would probably be as useless as it would be unwise. Unpopular legislation cannot be imposed. G.M. Trevelyan (54) has pointed out that the prohibition, instead of regulation, of usury in the Middle Ages did not lead to its abolition but to such high rates of interest as 50%, because it was an illegal act. The prohibition of alcoholic

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beverages in America in our own day led to methyl alcohol becoming the most important poison in that country. It may here be pointed out that neither was factory legislation in the 19th century popular; indeed it was far from being the case. Even among the parents of those children who were employed in the factories, bitter opposition was aroused to the Factory Act of 1833 because it threw some 40,000 children out of work, thereby rendering their parents poorer and less able to pay school fees. The hordes of children thus let loose upon the streets made a system of free education necessary. In all these cases, the intentions were good, but only the last remain and are generally accepted.

The reason is not far to seek. Factory legislation may have been unpopular in some quarters, but generally it was acceptable to many industrialists whose factories were administered on lines which were often in advance of legislation. Moreover, the Victorian public conscience was not difficult to arouse by such able men as Charles Dickens. If laissez-faire has become anathema to the 20th century mind, it must not be forgotten that in the hands of its best practitioners, it was humanitarian and philanthropic.

The history of labour passes from the family handicrafts of the rural cottage to the unhygienic conglomeration of riparian hovels in some isolated spot with no amenities but with plentiful water-power, to the factories where steam was employed as the motive power. Machines became more

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expensive, changed so quickly as to become rapidly obsolete and placed their purchase far beyond the reach of workmen. At first the merchants employed isolated out-workers, then the workers gathered in factories to rent machines and finally toiled for wages or the "truck" which was forced upon them in lieu of money. They brought with them the evil conditions of child labour, long hours and bad ventilation from the cottage; intensified them by urbanisation and lost self respect by becoming tied to the factory and to the town. "Docile labour" in a district was an attraction to the prospective factory owner. Many strove and some succeeded, alone or in groups, to own the machines. Money became the golden key to unlock factory gates. Hence behind the humanitarian and philanthropic industrialists was a monstrous horde of fiercely competitive and ruthless employers. It was for these that the legal minima were largely necessary.

Meanwhile, scientific advances have effected another revolution. Modern industry has become so technical that only supervision by a new class of persons, scientifically trained and often with no financial interest in the business, will permit it to survive. The shareholders have generally retired, so far as management is concerned, and made way for the professional scientist and administrator, a very different person indeed from the Victorian industrialist. This has been termed by Burnham (53) the "managerial revolution".

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To the scientific study of machines and materials has been added the scientific study of personnel. Men work up to a maximum of efficiency with a 60 hour week, after which their labour is uneconomic and so on. By S.R. & O. 1940, No.1325, Emergency Powers (Defence) Factories (Medical & Welfare Services) Order (15), the Minister of Labour & National Service made the following compulsory:-

"The occupier of any factory in which is carried on the manufacture or repair of any munitions of war or of any materials, parts or tools required for such manufacture or repair, or any work on behalf of the Crown shall, if so directed on behalf of the Minister by the Chief Inspector of Factories or by any other Inspector of Factories authorised by the Minister to give directions under this order, make arrangements to the satisfaction of the Inspector by way of the whole or part-time employment of such numbers of medical practitioners, nurses and supervisory officers as the Inspector may specify, for one or more of the following services, namely:-

(a) medical supervision of persons employed in the factory in the aforesaid manufacture, repair or work,

(b) nursing and first-aid services for such persons,

(c) supervision of the welfare of such persons."

It would seem but a short step to creating codes of ethical conduct among all those scientifically trained persons who are engaged in management. Chemists, physicists and engineers should be taught the elements of

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industrial hygiene. Medical men should be taught the elements of machine and building design. It should be against the professional etiquette of an engineer to design a plant without consideration of the physiological requirements of the men who will work it, for the least which will happen will be a loss of efficiency. Certain legal minima will still be required, but they should remain minima.

What has been said of industry in general can, in some measure, be applied to the manufacture of asbestos materials. The technicians who "manage" the industry have, as their object, the maximum productive capacity of a factory to supply a demand, with the profit motive as a secondary consideration. In order to achieve that end, efficient machines must be matched by healthy, contented workers. Any means which contributes to these things is held to be desirable, whether or not there are legal sanctions. Legal minima have already been established; further legislation should only be in the nature of a guide. The evolution of industrial organisation itself with proper guidance will solve more problems than Acts of Parliament.

Ideally, every case of industrial disease is preventable and asbestosis is no exception. But it is also true that every war injury is preventable. Practical difficulties in finding a substitute for asbestos which is at the same time harmless to human beings and also fulfills the same useful purposes to the same degree of efficiency

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have so far proved insurmountable. The effect of legislation has been, however, to concentrate the handling of the crude material into two large organisations where mass production methods have more chance of success in dust elimination than was the case in smaller factories. This result is also important in that better control by the Inspectorate is possible.

It follows, therefore, that scientific dust suppression and extraction are the only methods which have a reasonable hope of success. Construction of suitable ventilation plant is of prime importance and the full-time services of an engineer engaged on this work are well worth while. Maintenance of ventilation plant in an efficient state is almost as important. It is in this sphere that the co-operation of workers actually employed on the protected machines is of most value in reporting wear. Good house-keeping shows as good results in the asbestos industry as in any other. Every industrial hygienist is familiar with the process which begins humbly as an experimental venture with little or no safety precautions and unobtrusively grows into an important unit in the productive effort of the factory, but retaining its original primitive health safeguards.

Hand-fed hoppers on cards and crushers and old fashioned cupboards for the collection of opened asbestos should be eliminated. The spindles which continue to revolve after the yarn has broken, whirling frayed ends at high speeds in the atmosphere, should have automatic

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breaking devices. Closer study should be made of ducting: a bell-shaped exhaust point is useless because it rapidly becomes choked where it is narrowest at its junction with the duct. To apply draught to each end of a duct running the whole length of a spinning frame, a duct perforated by a multitude of exhaust points, simply means that no draught at all is exerted over the middle spindles of the frame. The principle of down-draught ventilation should scarcely ever be varied; there are few processes in which it is necessary to extract dust past the operative's face. All exhaust ventilation should be planned with the idea in mind that masks will not be worn by 1% of the workers, no matter how comfortable, efficient and plentiful they are.

For the future, the duties of hygienists will include an insistence not only upon an irreducible concentration of the dust cloud but also in earlier diagnosis. As Burton Wood (33) remarked in 1934, "We now diagnose incipient phthisis with confidence at a stage when the patient is healthy in appearance, well nourished, and little troubled by symptoms. In the diagnosis of asbestosis we have still to depend upon the symptoms and signs of more or less gross disease". Some years of experience of the disease has added to knowledge of it and some familiarity with its vagaries have led to earlier recognition than heretofore, but Burton Wood's ideal of early diagnosis, comparable to the early diagnosis of tuberculosis, has not been reached yet. Moreover, as the engineer develops the technique of

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ventilation, the disease, which has already exhibited flexibility and elusiveness, will tend to become more insidious and demand higher diagnostic skill than ever.

The social consequences of setting a time limit for exposure to an asbestos hazard are considerable, but not insuperable. It is probably true to say that no one should be permitted to work more than five years in such circumstances. The chief difficulties are the labour shortage, the additional cost of training which would have to be added to the price of the finished article, the psychological effects of a blind alley occupation and the time taken to effect the social changes necessitated by the provision. Labour shortage will largely be remedied by demobilisation from the armed forces, but will require the assistance of vocational guidance. Cost of training should be a direct charge upon the community instead of the indirect way of charging disablement to production and thence to the consumer. It is better, moreover, to spend the money on healthy men and women rather than to provide them with elegant funerals. Men are probably more resistant to the disease than women and where possible should be employed instead of women. The association with pregnancy has not proved a happy one and, in slight measure, might be a contributing factor in limiting the population. As Dean Inge has pointed out, it is useless to destroy the male population and leave the young women if the object is to exterminate a race; but no war leader has yet proved

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himself so ruthless as to adopt this measure. Neither is industry agreeable to adopting the measure.

The alternation of jobs in the same factory has many advantages, but is not a solution and there is also the sobering reflection that re-entry into a dusty occupation after an interval of rest seems to light up a quiescent lesion. Once a man has left an asbestos hazard, he should do so for good. The real answer to the problem is the organisation of industry on the basis of related crafts. There are many motions in the wool, silk, cotton and asbestos industries which are similar and transfer from one to the other would add variety to life with opportunities for enhancing skill and experience, so avoiding the objections of a short term, blind alley occupation. Some slowing up in production and added costs by way of training would, of course, be inevitable. Under a voluntary system the only inducement to work in the asbestos industry should be shorter hours; the bribery of high wages known as "danger money" or "dirty money" is a device which should be severely restricted to temporary operations where no other alternative is available.

The aggregation of similar or related industries might therefore be necessary and would require to be correlated with the general plans for dispersal of factories and diversity of employment which, it is thought, will lead to less unwieldy groups and a more stable mode of life. It must be conceded that such plans will take many years to

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develop, but unless such problems as asbestosis are related to the larger whole, complete solution of many difficulties will remain unattainable. Industrial medical research laboratories can do a very great deal in discovering toxic properties of substances before manufacture on a large scale is attempted. They can also indicate how such toxic properties can be avoided. They should be encouraged and developed. But a negation or prohibition, in peace or war, may well prove fatal to a nation in competition with others and alternate plans which involve the structure of society are necessary to give it that resilience as will stand the shock of competition. It is the application of the principle enunciated by Ramazzini nearly 250 years ago and which seemed appropriate both to begin and to end this paper.

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SUMMARY

1. Costs of asbestosis to the industry have steadily risen since the introduction of legislation.

2. Events leading up to a compensation scheme for asbestosis in 1931 are considered. The Orders which became law in that year were:-

The Silicosis & Asbestosis (Medical Arrangements) Scheme.

The Asbestos Industry (Asbestosis) Scheme and

The Asbestos Industry Regulations.

These are briefly considered, together with their interpretation in the light of experience. The population at risk is defined as the same number as the number of annual medical examinations of First Schedule workers carried out by the Board.

3. The population in the industry is shown to be comparatively static. Curves indicate a dramatic fall in morbidity rates. There is statistical evidence that the disease is becoming more chronic in character. Tuberculosis attacks 1/3 of the cases but only shortens the actual duration of the disease (asbestosis) by 0.1 years on the average.

4. A full and accurate history is essential to the diagnosis of any industrial disease. In the case of asbestosis, a laboratory test designed to find asbestosis bodies in the sputum, is a valuable aid to establishing the fact of exposure.

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5. The pathological features of asbestosis are discussed. Confluent types are becoming rarer. Right heart dilatation is common.
6. A classification of the asbestiform minerals is given, together with some chemical and physical data illustrating properties which are common to all groups and others by which they differ. The dusts, generally, are considered. The actions of silicious dusts on the lungs are compared and contrasted. The physiological results of fibrosis are examined.
7. Experience shows that asbestosis is a clinical rather than a radiological disease. Broadly speaking, there are acute and chronic types, the latter increasing in frequency as the dust cloud has become less concentrated. There are non-specific and specific reactions to the dust. It is a mono-symptomatic disease. Emphasis is laid upon the cardiovascular effects, which, once begun, remain, as contrasted with pulmonary signs which are often not apparent at all times. These signs are a raised diastolic blood pressure, an accentuated second pulmonic sound and clubbing of the fingers.
8. The radiological features are described. The gross changes seen 15 years ago are now rarely encountered.
9. Bronchopneumonia is the commonest complication of asbestosis. Tuberculosis is of the acute, caseous type, generally behaves like bronchopneumonia as a terminal infection and the cases fall into Meiklejohn's Natural Group.

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10. Emphysema is commonly found and may cause spontaneous pneumothorax if a superficial bulla ruptures. It contributes to the effects of back pressure.

11. Bronchiectasis is a rare complication, a finding at variance with most writers.

12. Two types of cardiovascular complications are described:

    (a) those due directly to the disease and described as clinically part of the disease

and

    (b) those such as rheumatic endocarditis and atheroma which are concurrent with it.

13. Pregnancy probably has a deleterious effect on the asbestotic because of its calls upon the cardiac reserve rather than by any effect it may have on the pulmonary fibrosis.

14. Cancer of the lungs complicated 15 of the 98 fatal cases and 5 other cases exhibited cancer of other organs. The range is too small to draw final conclusions. It is suggested that the metallic bases of the asbestiform minerals, especially iron, may account for the higher incidence in asbestosis as compared with silicosis. The irritant effect of asbestos may be a contributory factor.

15. Other complications are described, e.g. Bright's Disease, pancreatitis and lithiasis, but they are considered fortuitous.

16. In management of the disease, the prevention of infections is most important. Six cases of

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asbestosis which developed pneumonia are known to have recovered from the pneumonia. One case was treated with sulphapyridine and one with sulphathiazole. Artificial pneumothorax is probably contra-indicated when the case is complicated by tuberculosis. Dusting workers with aluminium dust is theoretically untenable. Cardiovascular complications are treated according to etiology. The obstretrician should conduct a case of labour with a view rather to the heart than the lungs. Ether should be avoided as an anaesthetic. The importance of the psychological approach is considered. Prevention and rehabilitation are intimately bound up with national policies of distribution of industry.

17. Legislation has been generally effective in controlling asbestosis, but not entirely. It has reduced the incidence of asbestosis and altered its characters. But further legislation is probably unwise and unnecessary. The immediate necessities are the development, by teaching, of ethical codes to the new class of technicians together with improved engineering. Asbestos has such important social values as to provide against its relegation and to necessitate efforts to solve its problems until such time as a harmless substitute has been found.

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A P P E N D I X I

PHOTOGRAPHS OF

PATHOLOGICAL SPECIMENS

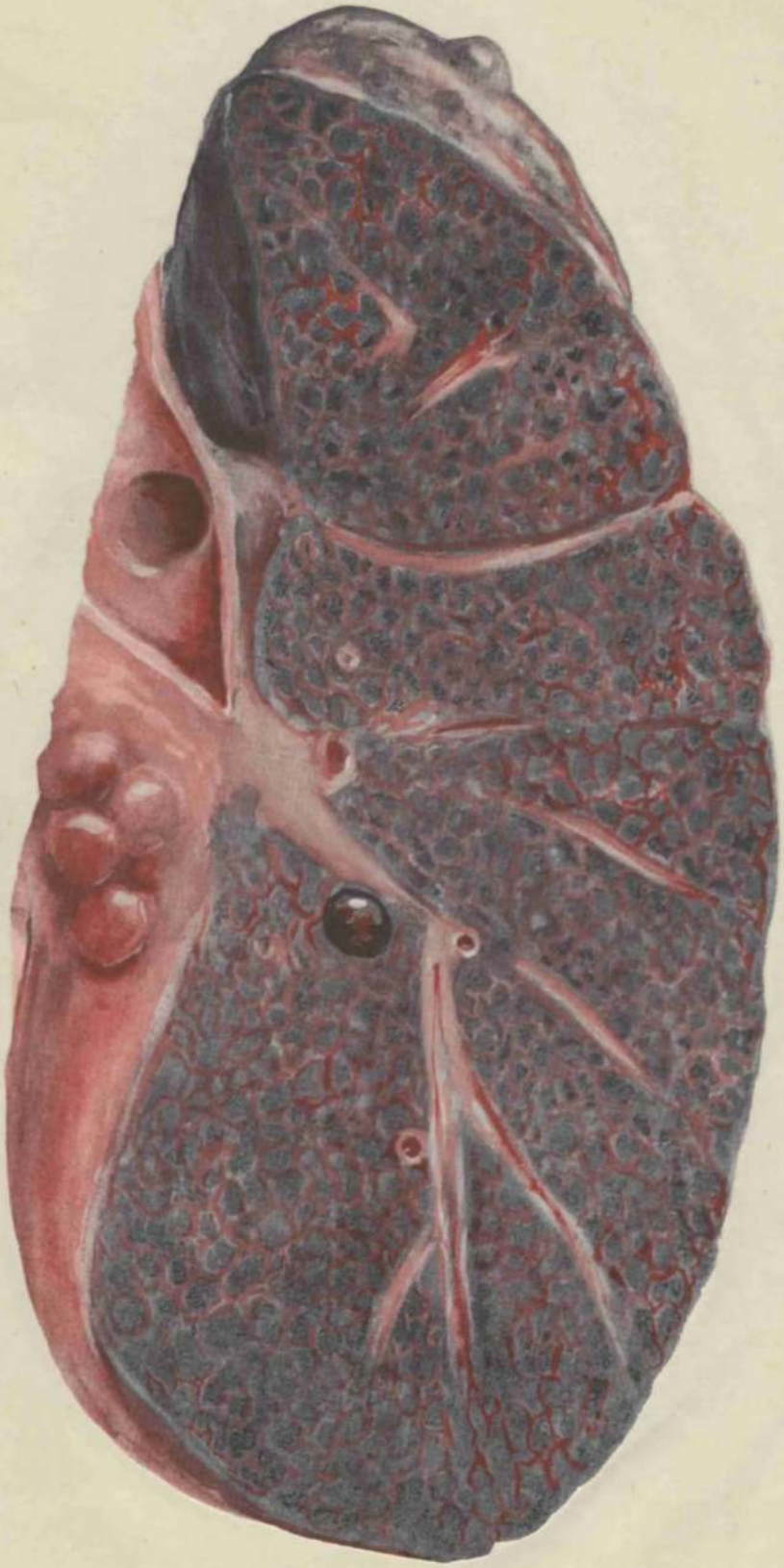
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PLATE 2

THE ASBESTOTIC LUNG

Reproduction of watercolour kindly presented by Dr. Roodhouse Gloyne. The blue polygonal areas, roughly lobular in distribution, are clearly seen on a red background of broncho-pneumonia. These areas of fibrosis are always of the same colour irrespective of the type of asbestos causing the condition. Basal confluence of the areas is well illustrated.

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PLATE 3

THE ASBESTOTIC LUNG

Case R.W. See p.87. Female. Age 41. Spinner. Exposure: 25/7/18 to 15/6/21 and 8/8/21 to 10/7/24. Left employment to marry. One healthy child born 1927. A sister of patient died of asbestosis 1935. Cough for several years, but worse and began to lose weight after an attack of Mumps in 1939. Dyspnoea particularly severe 1940 onwards. No pulmonary T.B. found at P.M. Healed T.B. mesenteric glands found.

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PLATE 4

CASEOUS TUBERCULOSIS AND ASBESTOSIS

Case L.T.N. Male. Age 36. Lagger. Father and two brothers died of T.B.

Exposure: on and off 1924 to 1940, working with 10% to 15% asbestos mixture with magnesia and other substances in the wet and dry states.

First broke down with T.B. in 1935. Said to be always complaining of his stomach and he was very thin. Died 20/4/42. Specimen shows cavity in left upper lobe with roof torn away in removing the lung from the body. There is a black dense composite mass below the cavity and pleural drift of pigment. Bronchogenic spread of caseous T.B. in lower lobes. Background of congestion. Histologically, typical reticular fibrosis. Asbestosis bodies present. Stomach was found to be healthy.

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PLATE 5

CHRONIC FIBROID PHTHISIS AND ASBESTOSIS

Case G.H.S. Male. Age 42. Mattress Department and Boiler Covering. For history and description see Case 5, p.74, of text.

X-Ray Plate No.20, p.172.

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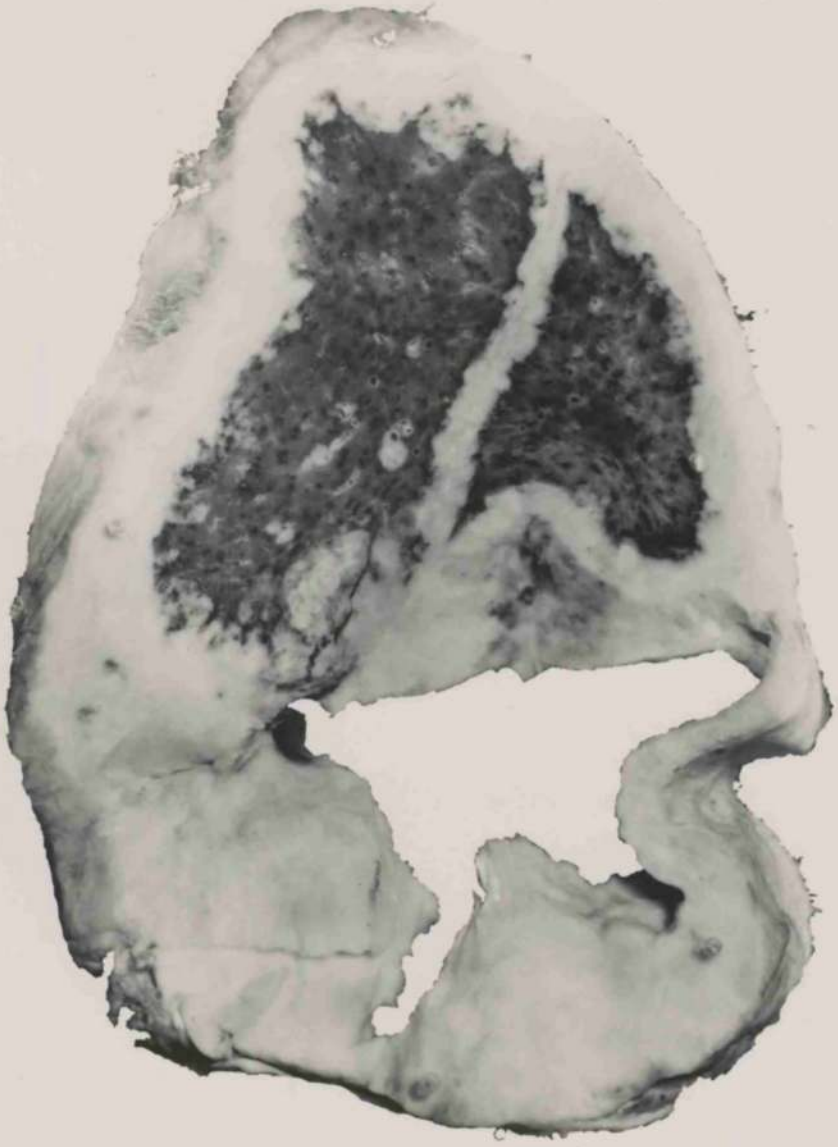


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PLATE 6

ENDOTHELIOMA OF PLEURA AND ASBESTOSIS

Case C.V.W. Male. Age 42. Greaser and Beltman 1921 to 1942. For history and description see Case 14, p.102, of text. A "false empyema", due to liquefaction in the depths of the growth at the base of the lung, can be seen in the specimen. X-Ray Plate 19, p.170.



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PLATE 7

SQUAMOUS CARCINOMA OF BRONCHUS AND ASBESTOSIS

Case L.E.S. Female. Age 35. Spinning and Sectional Departments. For history and description see Case 13, p.101, of text.


GROWTH
Abscess
Cavity

PLATE 8

(A) Photomicrograph showing fibres stuffing the bifurcation of a bronchiole.

PLATE 9

(B) Photomicrograph. Section of asbestotic lung to illustrate fibrosis and its effects in crushing the alveoli.

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PLATE 10

(A) Photomicrograph. Asbestos fibres in sputum seen by dark ground illumination.

PLATE 11

(B) Photomicrograph. Asbestosis giant cell.

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GIANT
CELL

PLATE 12

(A) Photomicrograph. An example of whorling from a case of asbestosis.

PLATE 13

(B) Same, using a green screen.

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PLATE 14

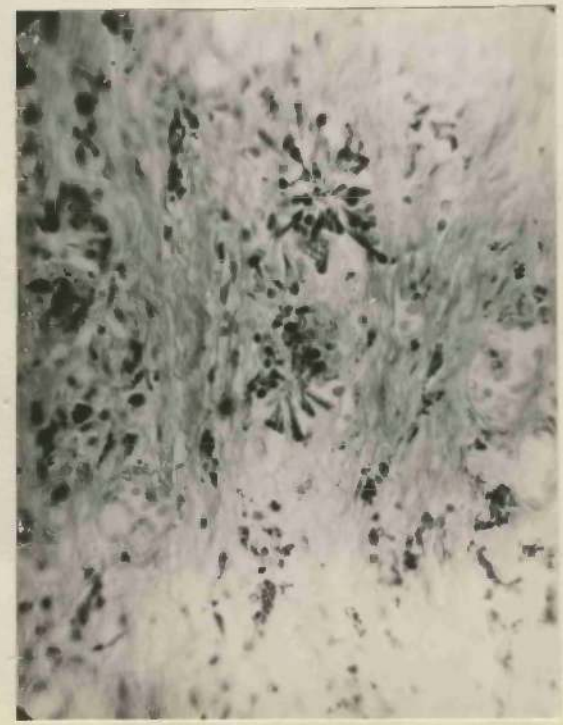
(A) Photomicrograph tinted faintly yellow. Asbestosis bodies in sputum.

PLATE 15

(B) Photomicrograph. Clumping of asbestosis bodies, signifying breakdown of lung tissue.

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PLATE 16

THE FORMATION OF THE ASBESTOSIS BODY

Negative kindly loaned by Dr. Roodhouse Gloyne.

Figures 1 to 3. Deposition of some material on the fibre, producing a sausage shape without differentiation (Figure 4).

Figures 5 to 7. Appearance of fissures giving appearance of incomplete segmentation at irregular intervals.

Figure 8. Fissures now extending almost to the mid-line of the body.

Figures 9 & 10. Heads or knobs appear at the ends.

Figure 11. A completely, but irregularly segmented body.

Figures 12 to 18. Fissures reach the straight central fibre and appear as a small number of long segments, or

Figures 19 to 34, a large number of short segments.

Figures 32 to 34. Short, regular segments like a necklace.

Figures 35 to 37. Some segments break away, leaving bare lengths of fibre between the remaining segments.

Figures 38 to 41. Bodies viewed end-on or obliquely.

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Asbestosis bodies. (Freehand drawing, 1/12 obj.)

To illustrate article, "The Formation of the Asbestosis Body in the Lung," by S. Roodhouse Gloyne, M.D., D.P.H.

A P P E N D I X II

REPRODUCTIONS OF

X-RAY FILMS

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PLATE 17

Case J.H.H. Male. Age 34. Opening room and cardroom 8/11/30 to 30/8/42.

Examination: Morning cough for half an hour, little sputum, no staining. No clubbing. Accentuated 2nd pulmonic sound and marginal crepitations. B.P. $\frac{140}{100}$.

X-Ray: Fine fibrosis right lower and mid zones and left upper zone. Emphysema left lower zone. See p.69 of text.

Diagnosis: Asbestosis.

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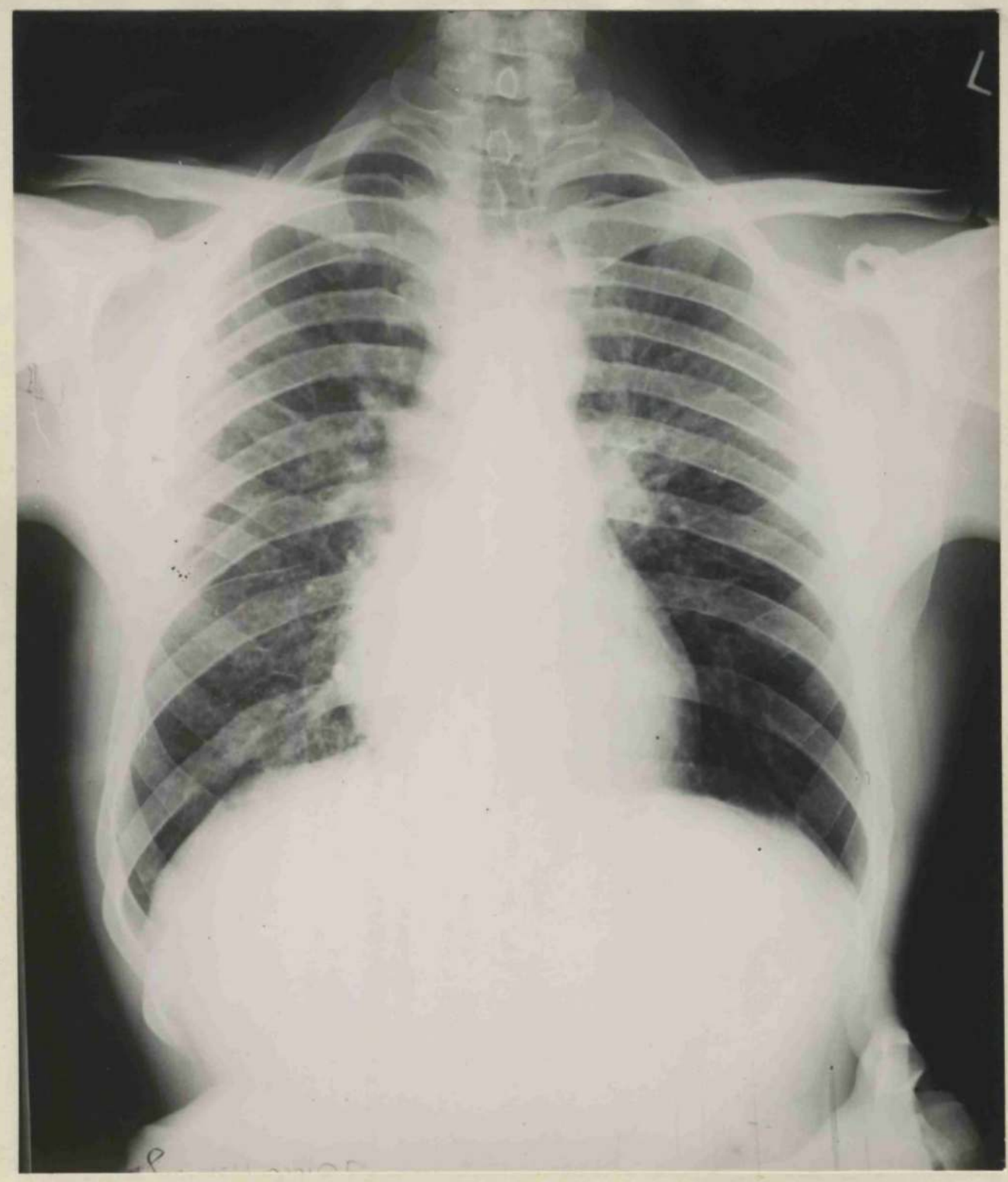

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PLATE 18

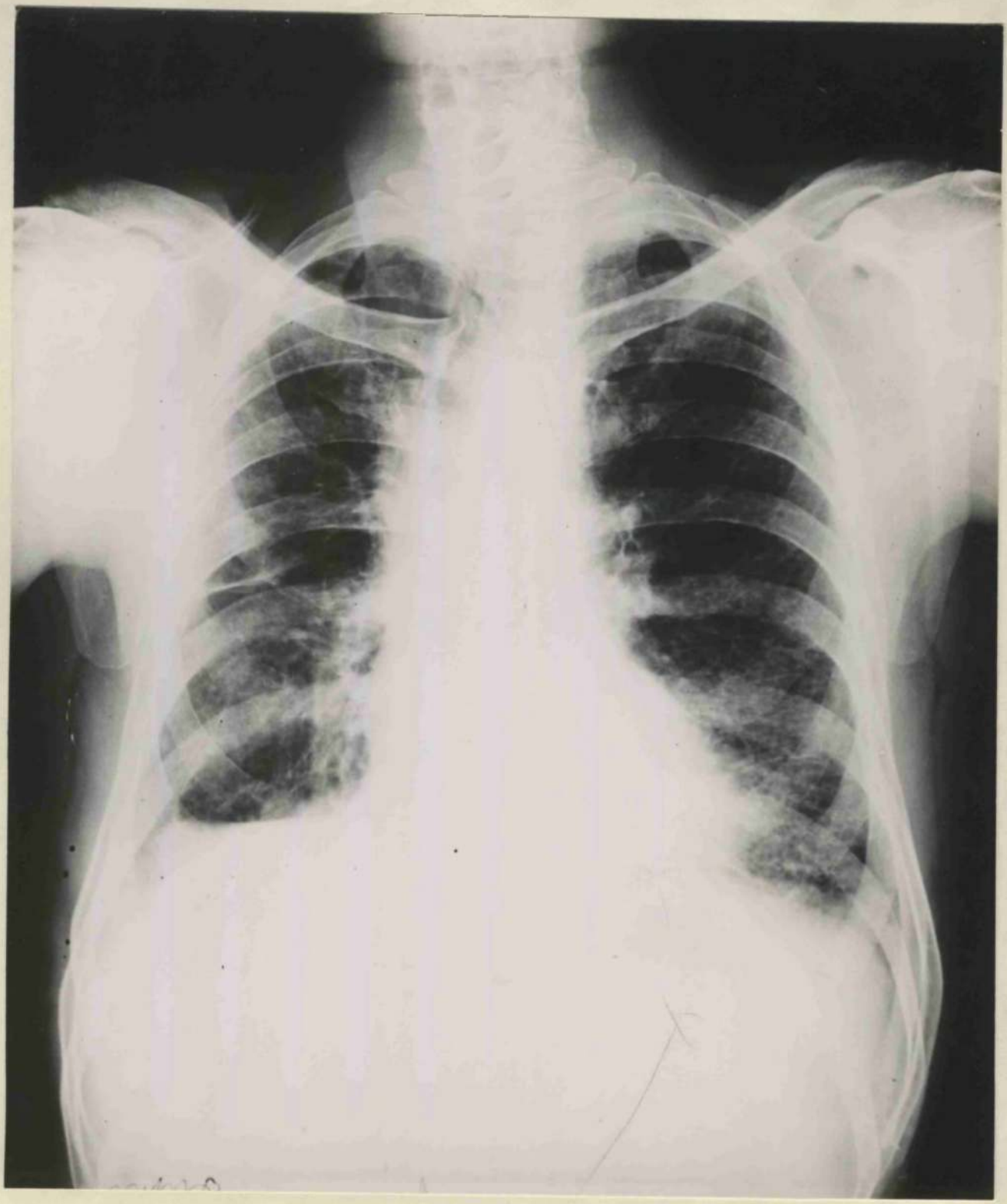
Case A.L.O. Male. Age 54. Weaving Department 8/8/32 to 21/2/44. Died 13/11/44.

Examination: Dyspnoea, cough, little sputum. Marked clubbing of fingers. Anterior chest hyper-resonant. Numerous basal crepitations. 2nd pulmonic +. B.P. $\frac{160}{110}$.

X-Ray: A fair amount of reticulation in the left lung, more marked at the base. Outlines of left cupola and left cardiac border blurred. Changes on right side not so obvious. There is a right diaphragmatic adhesion and coarse strains run towards the diaphragm. The trachea is pulled to the right.

Diagnosis: Asbestosis, confirmed at P.M.

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PLATE 19

Case C.V.W. Male. Age 42. Greaser and Beltman 2/8/21 to June 1942. Died 23/8/43.

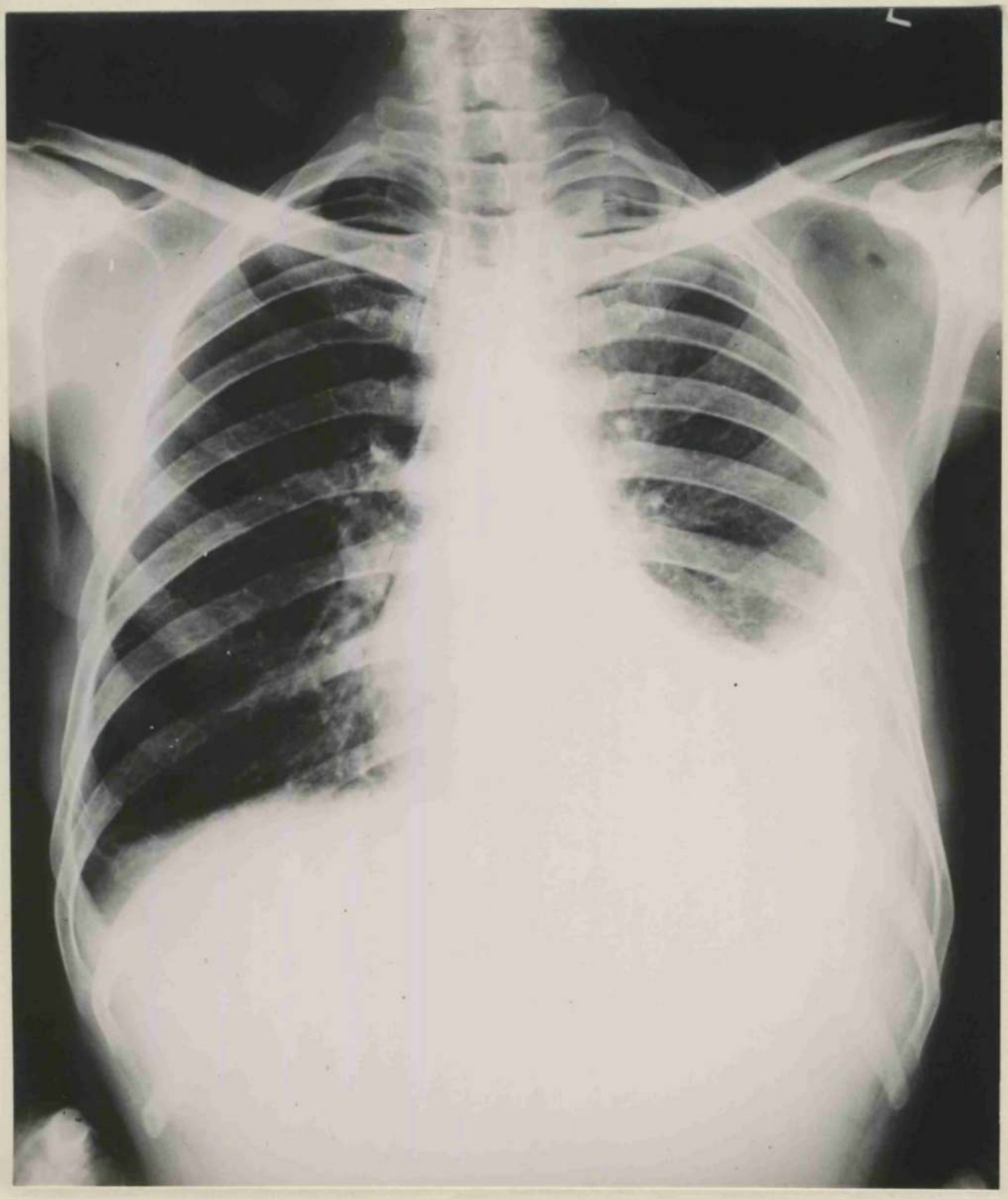
Case described in text under Case 14, on page 102.

P.M. specimen illustrated by Plate 6, page 151 in Appendix I.

A skiagram on 2/6/42 indicated fine fibrosis in the right lower zone and the right cupola indistinct in outline. The left costophrenic angle was obliterated. Skiagram reproduced here shows no change on the right side but an effusion at the left base.

Diagnosis: Asbestosis and endothelioma of the pleura. Confirmed at P.M.

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PLATE 20

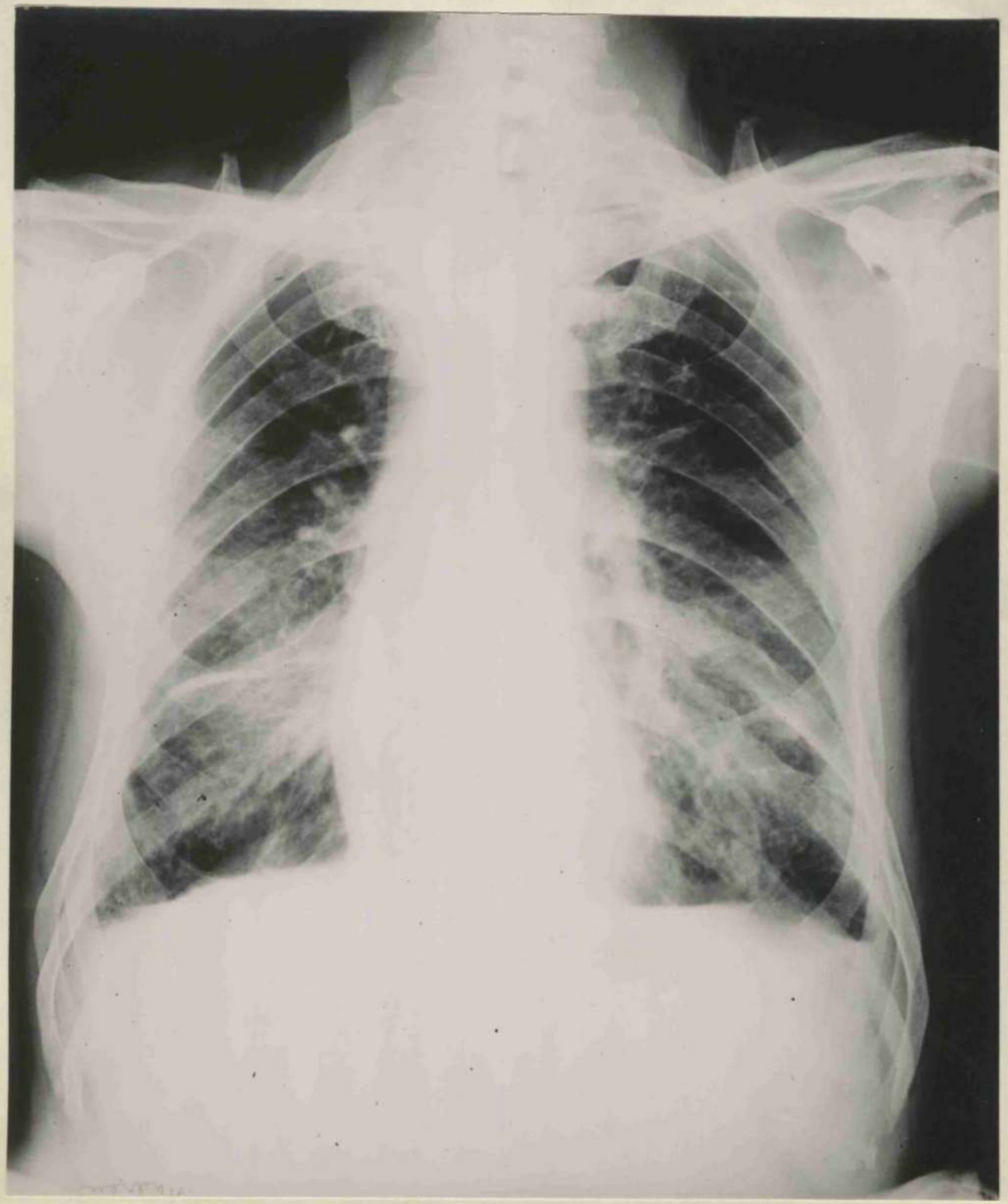
Case G.S. Male. Age 42. Mattress maker and boiler coverer 1/4/19 to August 1943.
Case and X-Ray appearances described in text under Case 5, on page 74. P.M. specimen illustrated by Plate 5 on page 149 in Appendix I.
Diagnosis: Asbestosis and Chronic Fibroid Phthisis. Confirmed at P.M.


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PLATE 21

Case W.A. Male. Age 57. Manufacture of asbestos belting 1925 to 1935. Cardroom 1935 to 1937. Examination: Herpes zoster January 1942. Chief complaint now of dyspnoea, some cough and a slight quantity of sputum. Clubbing +. Basal crepitations. Harsh R.M. in upper zones. 2nd pulmonic not accentuated. B.P. $\frac{142}{90}$.
X-Ray: Fibrosis both bases. Both diaphragms are adherent. Costophrenic angles blunted. Wide mediastinum.

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PLATE 22

Case J.N. Male. Age 41. Sectional Department 1926 to 1938.

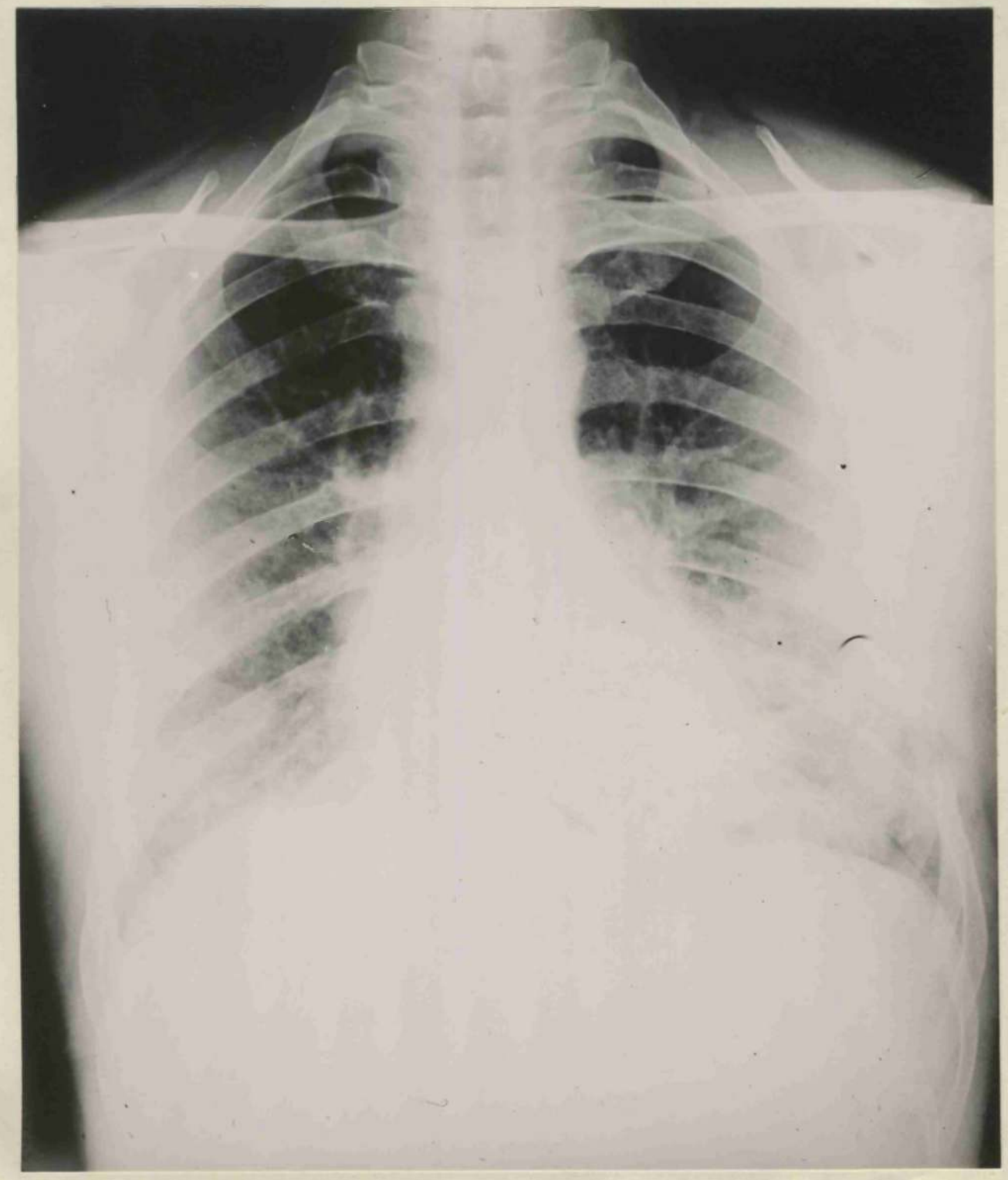
Examination: Tightness of chest, dyspnoea and dry cough. Clubbing ++. Basal crepitations. 2nd pulmonic +. B.P. $\frac{140}{98}$.

X-Ray: Marked fibrosis both lower zones. Shaggy left cardiac border.

See page 30 of text.

Diagnosis: Asbestosis.

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PLATE 23

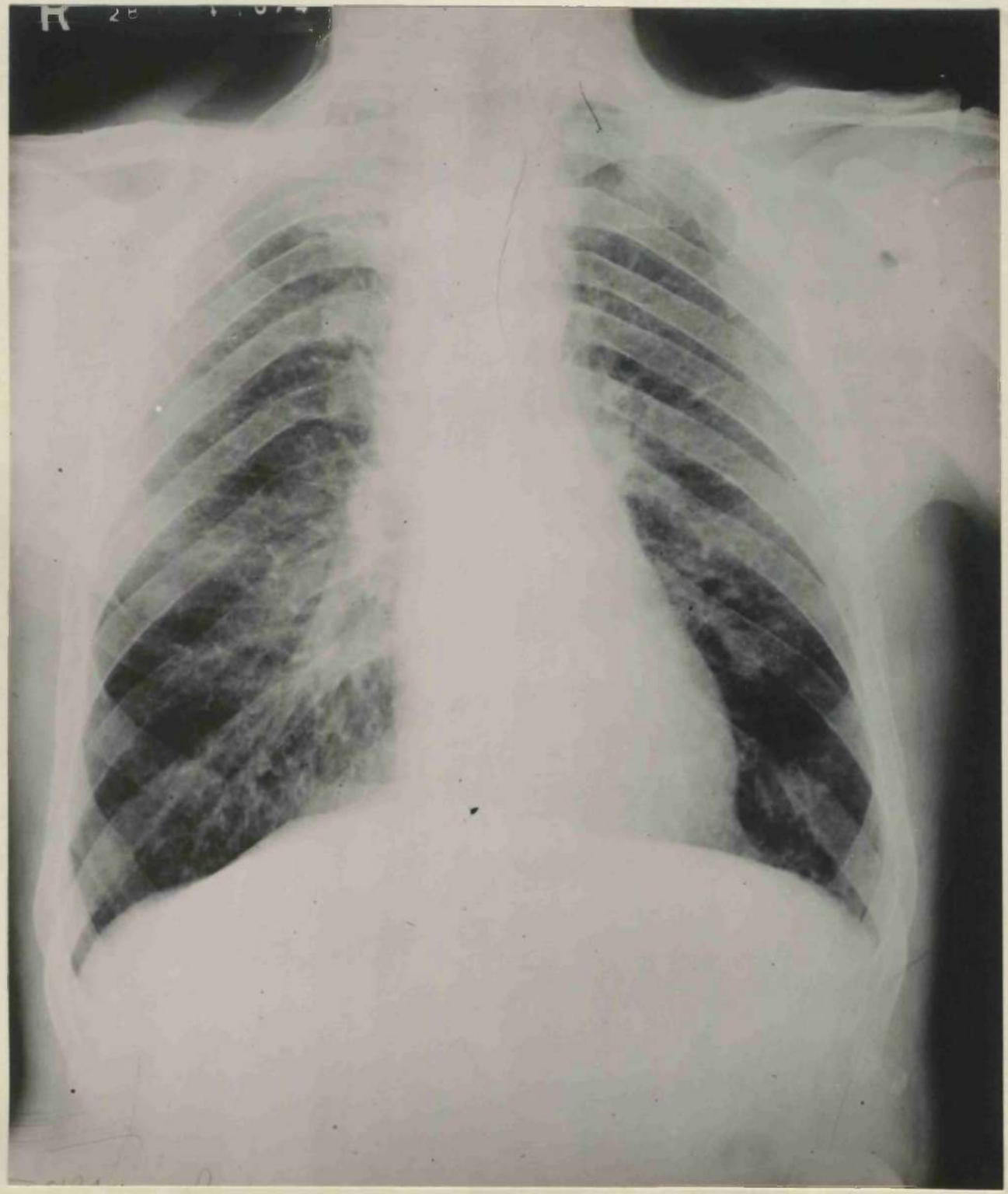
Case J.G. Male. Age 55. Cardroom 1930 to 1936. Opening room 1936 to 1938.

Examination: Dyspnoea, morning cough and some sputum. No clubbing. Numerous crackling rales anterior chest and to a less degree at apices. A few dry rhonchi at bases. No detectable accentuation of 2nd pulmonic sound, but an occasional extra systole. B.P. $\frac{144}{96}$. He died in June 1945 and at P.M. moderately advanced asbestosis was found and a squamous carcinoma of the left upper lobe.

X-Ray (28/1/44): All zones of the right lung show reticulation and the upper two thirds of the left lung. The cardiac silhouette and diaphragms are well defined.

Diagnosis: Asbestosis and carcinoma of lung.

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PLATE 24

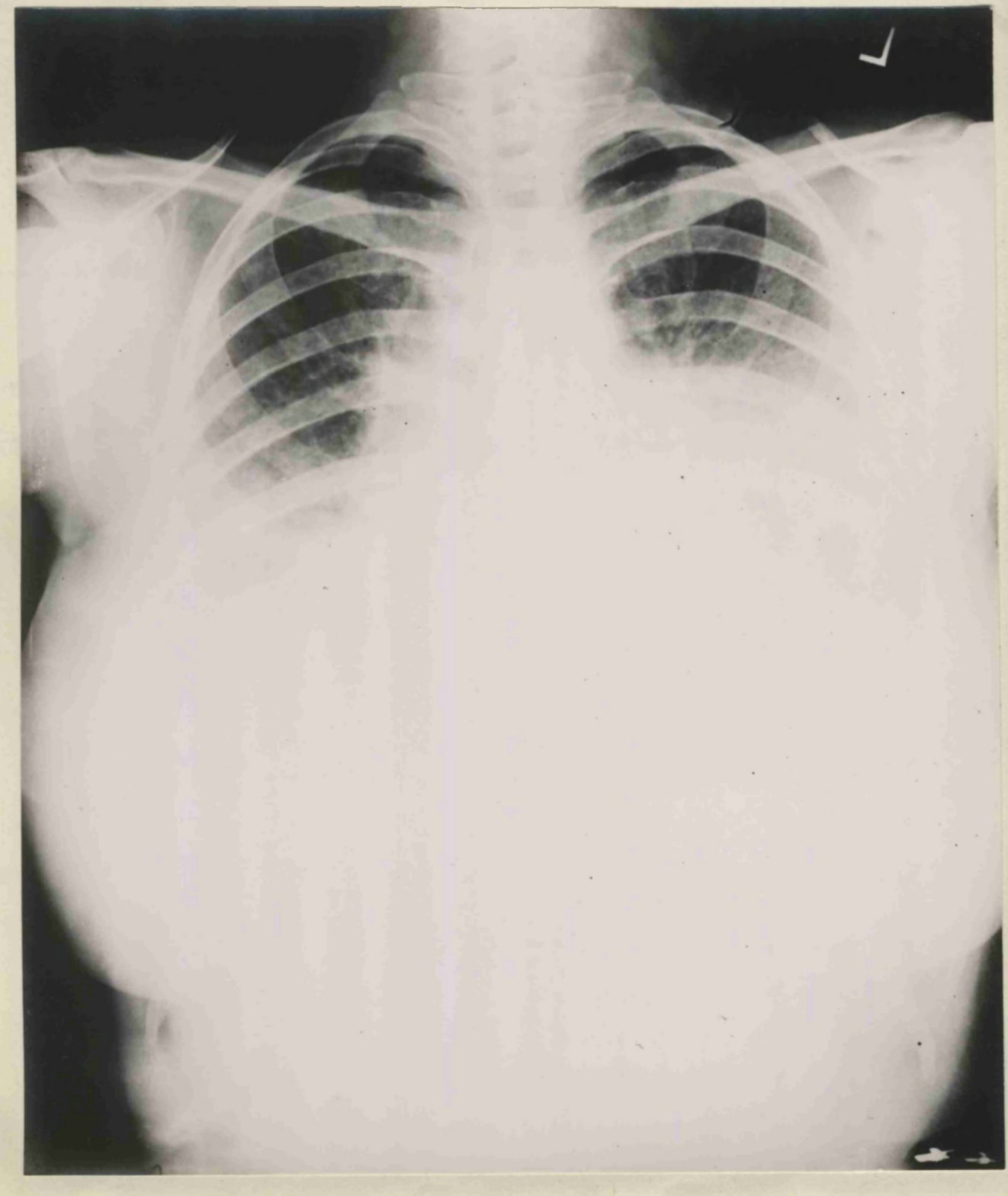
Case R.E.W. Female. Age 39. Disintegrating Department 15/3/27 to 24/12/32. Worked entirely with blue asbestos.

Examination: Dyspnoea and dry cough 2 to 3 years. No clubbing. Friction rub below right clavicle. Harsh R.M. and prolonged expiratory murmur below left clavicle. Crepitations and an odd squeaky rale at right base. 2nd pulmonic +. B.P. $\frac{130}{80}$.

X-Ray (13/7/45): Very stout woman, films poor and some details lost in reproduction. Fibrosis right mid zone and right base. More uniform density at left base, compatible with advanced asbestosis.

Diagnosis: Asbestosis.

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PLATE 25

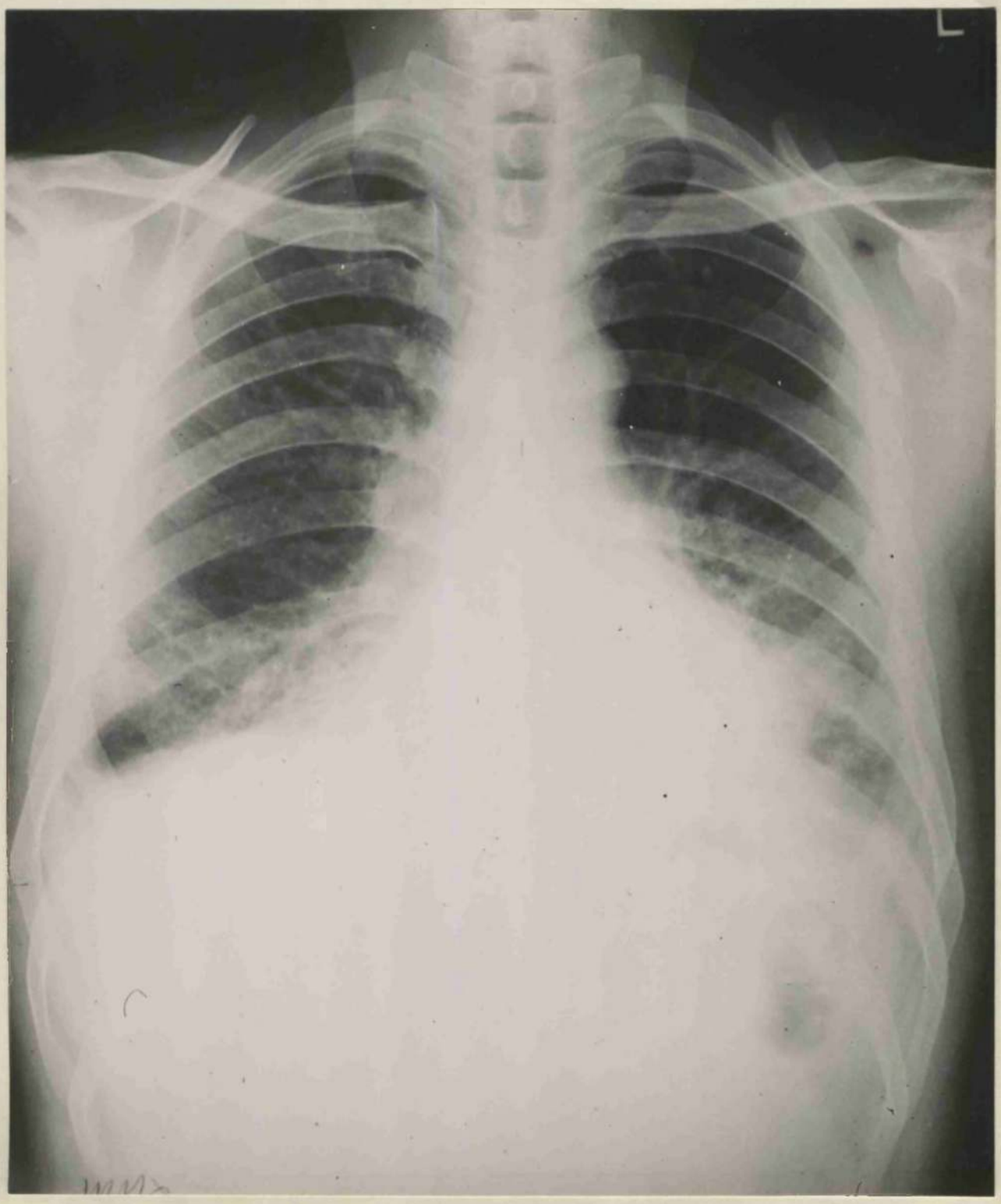
Case J.S. Male. Age 38. Stores 1924 to 1933. Occasional pains left shoulder-tip, morning cough, small quantity of sputum sometimes flecked with blood. Attack of pneumonia (right side) 1937.

Examination: Clubbing ++. Coarse, basal crepitations, more on right side than left. 2nd pulmonic +. B.P. $\frac{144}{110}$.

X-Ray: Both lower zones show definite infiltration with fine, dense miliary shadows. Diaphragm on right side irregular in outline. Both mid zones slightly affected. Shagginess of cardiac borders.

Diagnosis: Asbestosis.

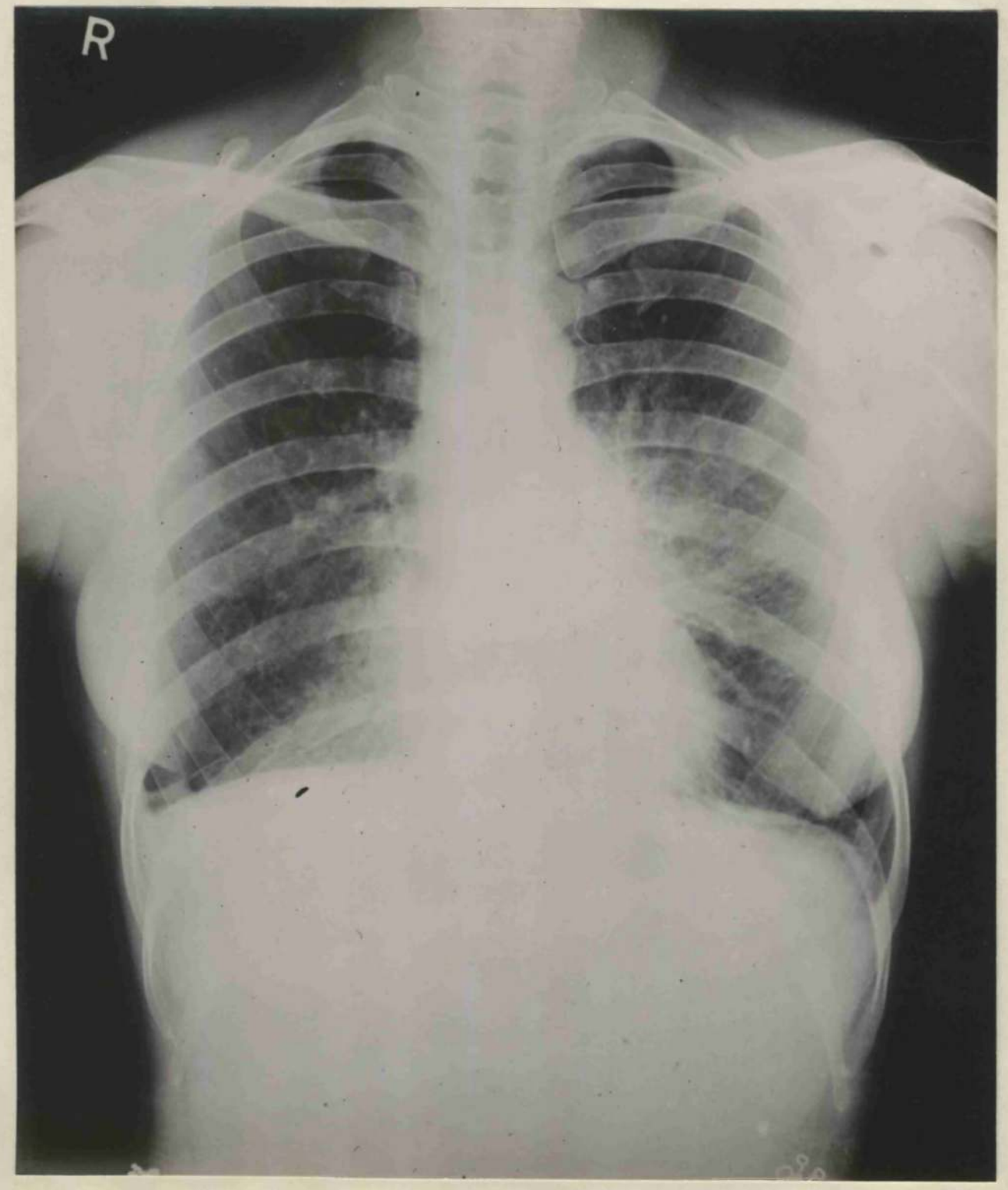
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PLATE 26

Case F.B. Female. Age 41. Weaving and Disintegrating Departments 1/3/21 to 11/2/33. Pains in chest and dry cough. Haemoptysis 1939. Examination: No clubbing. Chest expansion 31" to 32½". Squeaky rales at margins and harsh R.M. below left clavicle. 2nd pulmonic +++ and sometimes reduplicated. B.P. $\frac{154}{100}$.
X-Ray: Stippling right base and left mid zone. Some blurring of cardiac borders. Blunting of right costo-phrenic angle.
See page 83 of text.
Diagnosis: Asbestosis.



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PLATE 27

Case A.M. Female. Age 37. Doubling Department 15/1/26 to 23/4/30 and 10/11/39 to 31/12/41.

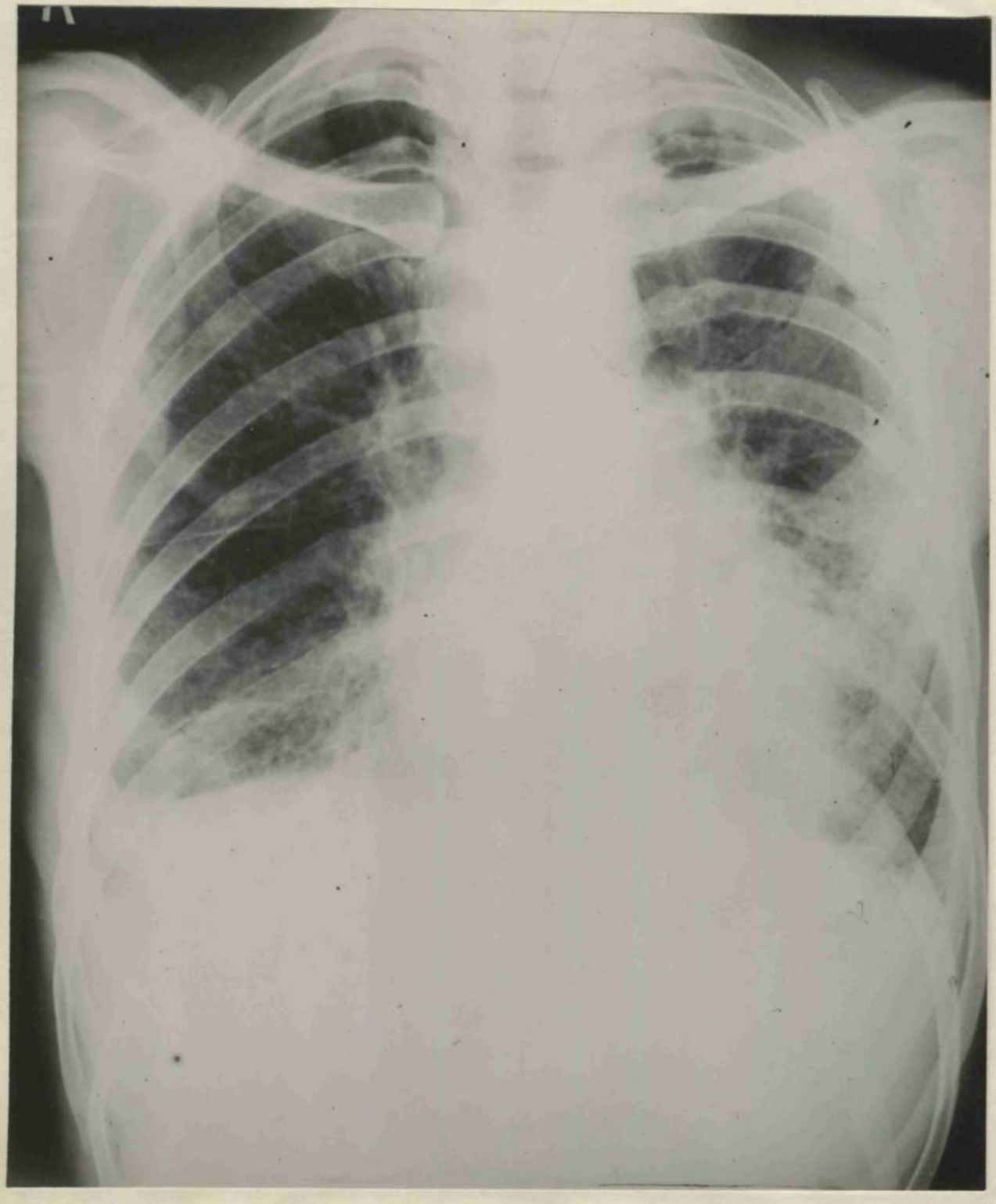
Hard cough and slight, stringy sputum. Dyspnoea not prominent.

Examination: Emaciated. Left chest less mobile than right. No clubbing. A few squeaky rales at bases and left axilla. Dullness and crepitations third left interspace anteriorly, dullness continuous with upper left cardiac border (? pleural plaque). 2nd pulmonic ++. B.P. $\frac{96}{64}$.

X-Ray: Extensive lung changes, especially at bases and marked density left mid zone. Striation of lung markings. Shagginess of heart contour. Extensive pleural adhesions. Highly suggestive of advanced disease.

Diagnosis: Asbestosis.

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PLATE 28

Case W.P. Male. Age 43. Stores 1926 to 1930. Opening Room 1931 to 1939.

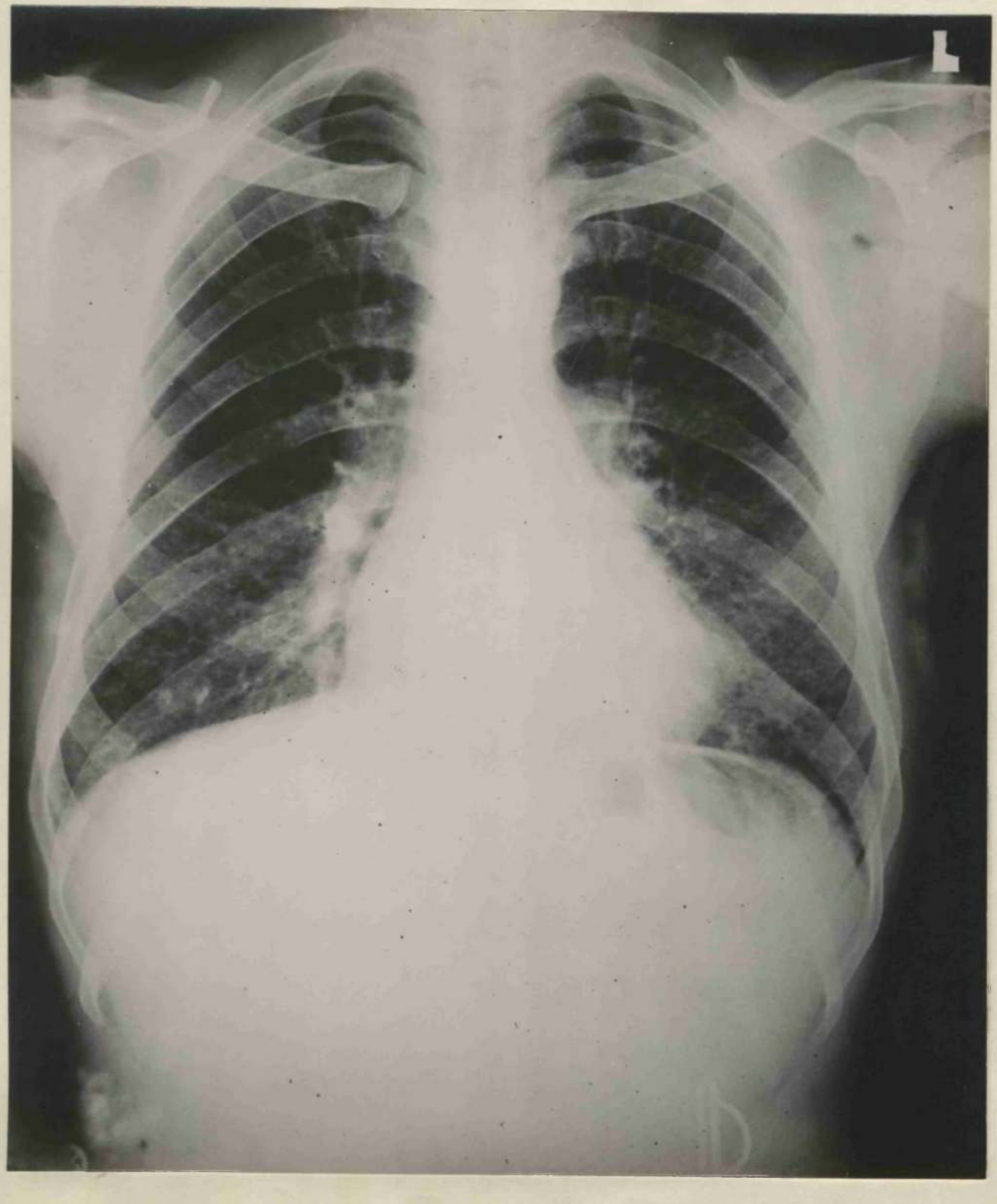
A myxoedematous patient on a maintenance dose of thyroid gland 1½ grains daily. Only complaint definitely referable to chest: dry cough.

Examination: A few faint marginal creps in left parasternal line. Clubbing of fingers and toes +++. 2nd pulmonic sound +. B.P. $\frac{122}{80}$.

X-Ray: Fine, basal fibrosis and blurring of left cardiac border. Appearances suggest a slight degree of the disease.

Diagnosis: Asbestosis and myxoedema.

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A P P E N D I X III

PHOTOGRAPHS OF

RAW MATERIALS & OF

FINISHED PRODUCTS

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PLATE 29

AN ASBESTOS MINE IN THE TRANSVAAL

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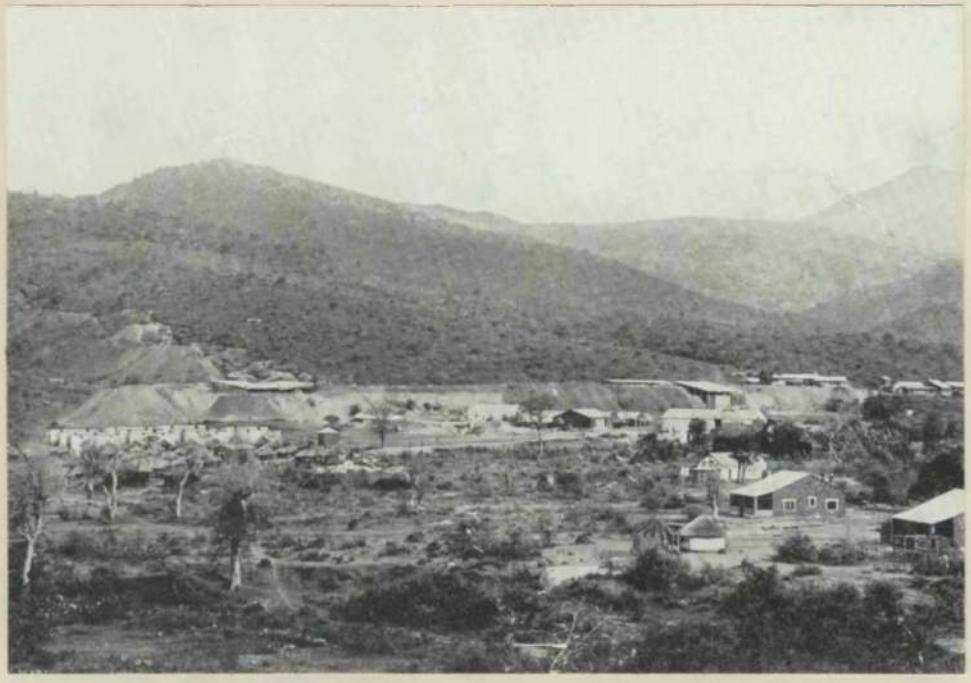


PLATE 30

TWO SPECIMENS OF BLUE CROCIDOLITE ASBESTOS AS THEY WERE RECEIVED FROM THE MINE. THE BLUE COLOUR SHOULD BE SOMEWHAT DEEPER. IRON-STONE IS SEEN CLINGING TO EACH SPECIMEN.

PLATE 31

A SPECIMEN OF CRUDE AMOSITE ASBESTOS.

PLATE 32

SOUTH AFRICAN CHRYSOTILE ASBESTOS.

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PLATE 33

TWO SPECIMENS OF WHITE ASBESTOS, THE FIRST AS IT WAS RECEIVED FROM THE MINE AND THE SECOND SLIGHTLY OPENED TO ILLUSTRATE ITS FIBROUS STRUCTURE.

PLATE 34

TWENTY OUNZES OF CRUDE AMOSITE ASBESTOS COMPARED WITH THE SAME WEIGHT OF SIMILAR MATERIAL AFTER CRUSHING AND OPENING.

PLATE 35

TWO POUNDS EACH OF WHITE CHRYSOTILE, BLUE CROCIDOLITE AND AMOSITE. THE FIBRES WERE PRODUCED FROM CRUDES OF SIMILAR VALUES. TO ILLUSTRATE THE RELATIVE CAPACITIES OF THE THREE TYPES IN RETAINING AIR, THUS ADDING TO THEIR VALUE FOR HEAT INSULATION.

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2.



1.





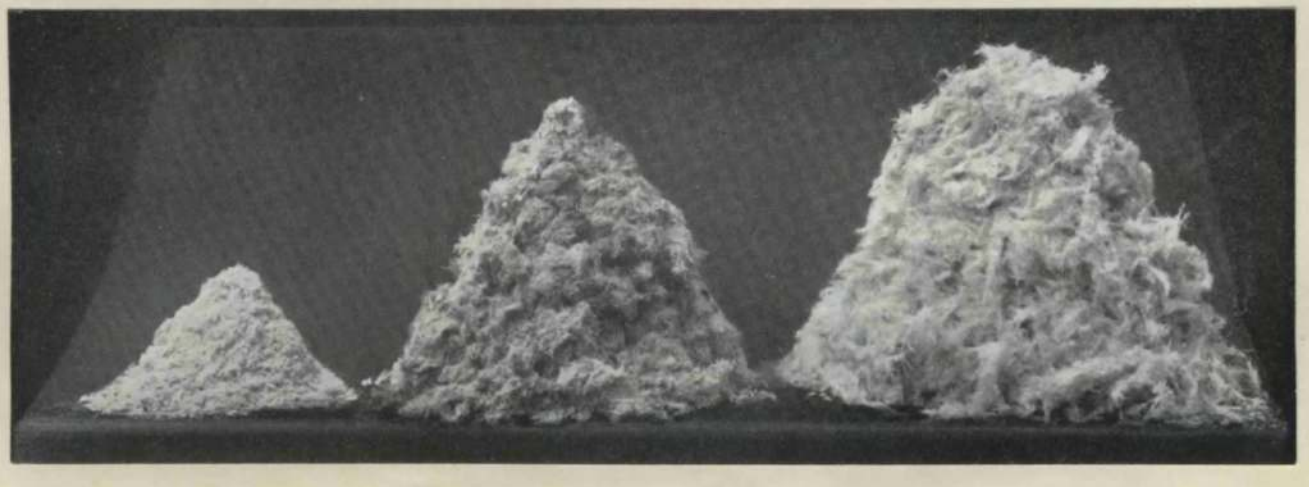


PLATE 36

A CARDING MACHINE. FRONT VIEW

PLATE 37

A SPINNING FRAME

PLATE 38

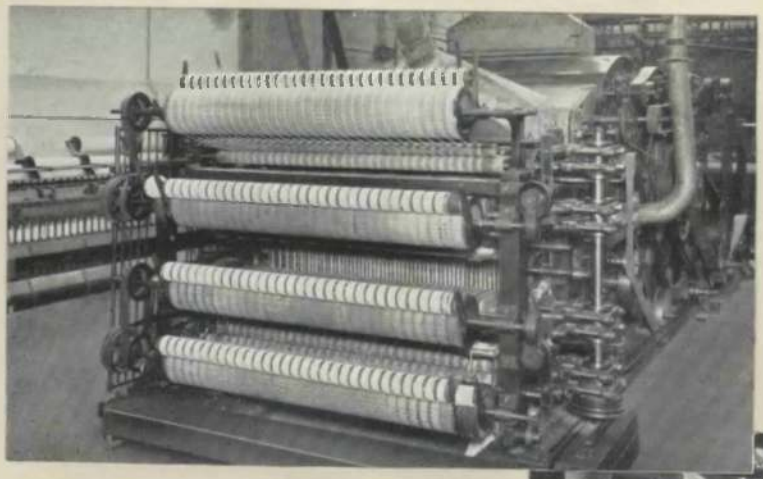
A DOUBLING MACHINE.

FOR PRODUCING A THICKER, STOUTER YARN

PLATE 39

A LOOM

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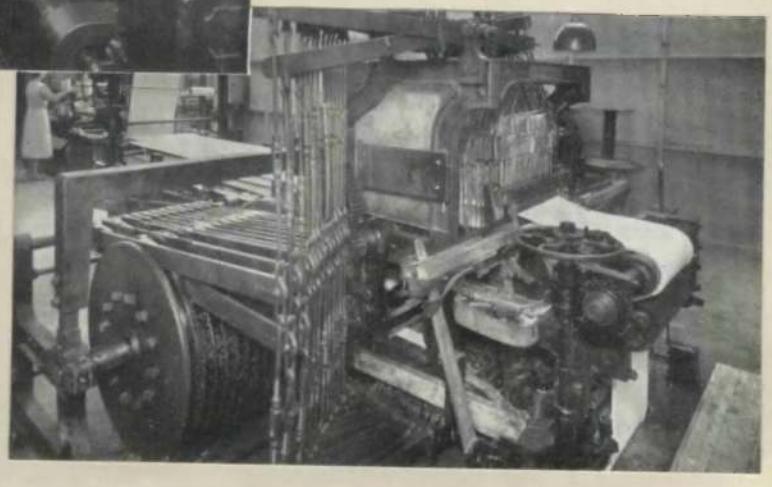


PLATE 40

BLUE ASBESTOS CLOTH

PLATE 41

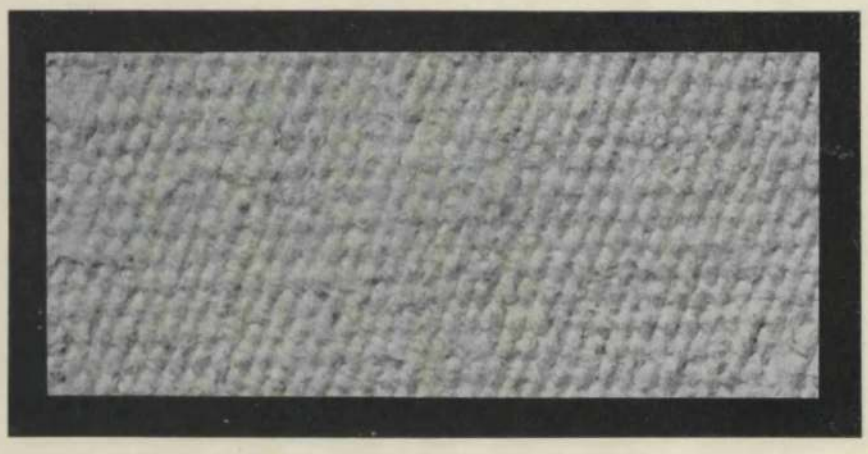
WHITE CHRYSOTILE CLOTH

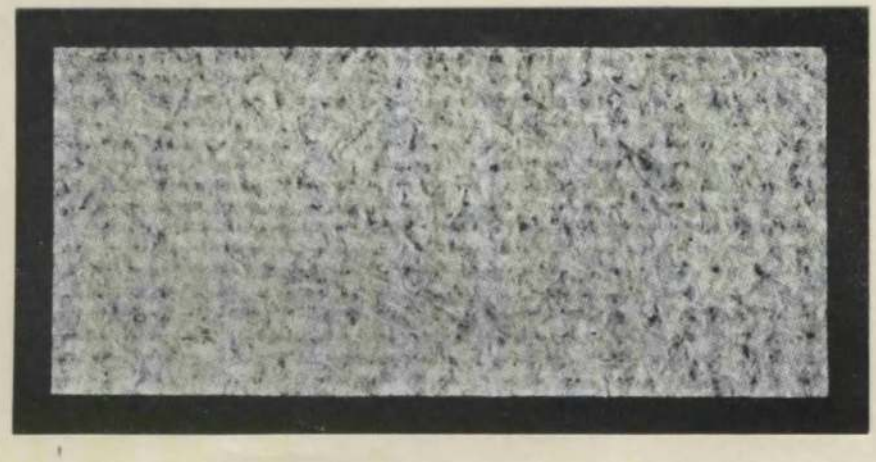
PLATE 42

CLOTH MANUFACTURED FROM AMOSITE

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PLATE 43

WHITE ASBESTOS CLOTH. SPECIAL WEAVE FOR DIAPHRAGM IN FAUSER ELECTROLYTIC CELLS, USED IN SEPARATION OF GASES DURING PRODUCTION OF SYNTHETIC AMMONIA.

PLATE 44

BLUE ASBESTOS CLOTH. SPECIAL DIAGONAL WEAVE. FOR USE IN FILTER PRESSES, e.g. FOR TARTARIC ACID.

PLATE 45

BLUE ASBESTOS CLOTH, SIMILAR TO THAT ILLUSTRATED ABOVE BUT OF LIGHTER WEIGHT FOR USE IN ELECTROLYTIC CELLS PRODUCING ELECTROLYTIC SODA.

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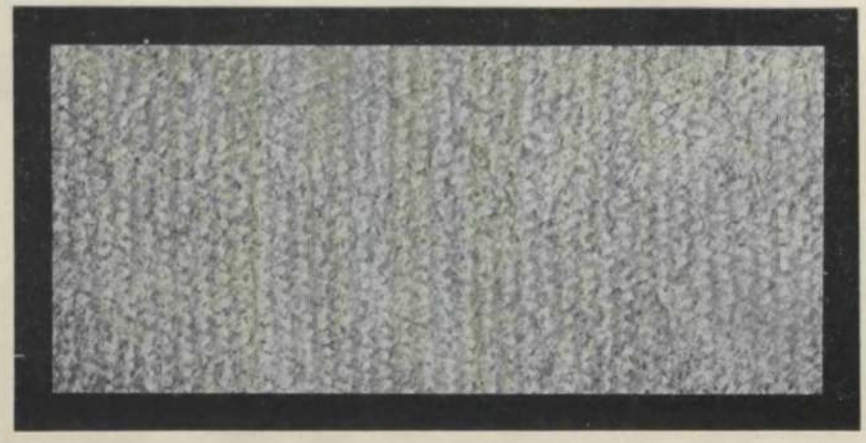






PLATE 46

ASBESTOS HELMET, LONG COAT, GLOVES AND BOOTS

PLATE 47

ASBESTOS APRON, GAUNTLETS AND BOOTS

PLATE 48

ASBESTOS HOOD, CAPE, APRON, LEGGINGS AND GAUNTLETS

PLATE 49

ASBESTOS HOOD, LEGGINGS AND GAUNTLETS

PLATE 50

ASBESTOS HAT WITH NECK PROTECTOR, JACKET, TROUSERS AND MITTENS

PLATE 51

AIRMAN'S OR RACING MOTORIST'S ASBESTOS SUIT

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PLATE 52

ASBESTOS SLIVER ROVING. A THIN RIBBON, CONTAINING SOME COTTON, WHICH, WHEN IMPREGNATED WITH A NEUTRAL SUBSTANCE AFTER WINDING ON AN ELECTRIC CONDUCTOR, INSULATES IT.

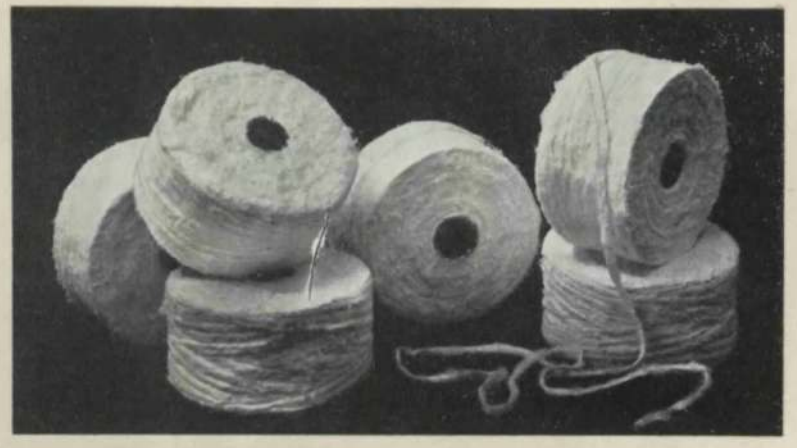
PLATE 53

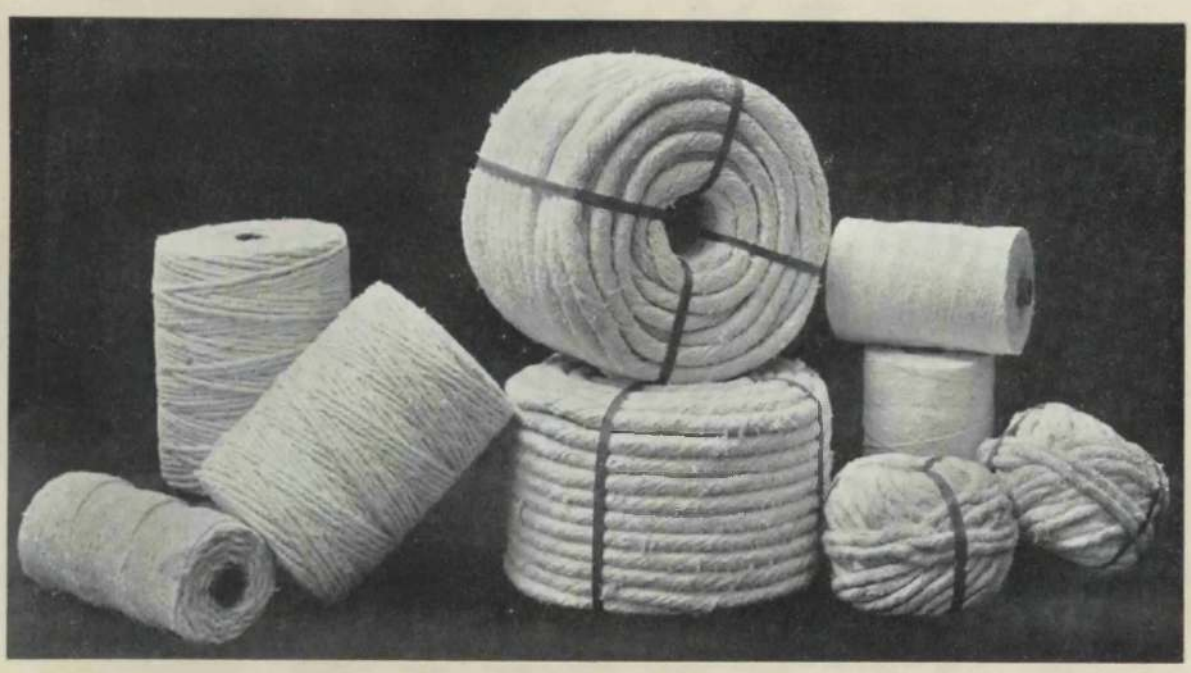
PHOTOGRAPH OF VARIOUS TYPES OF ASBESTOS YARN. SOME ARE REINFORCED WITH BRASS WIRE SPUN INTO THE YARN AND OTHERS MAY BE TREATED WITH LUBRICANT AND GRAPHITE COMPOUNDS.

PLATE 54

PHOTOGRAPHS OF ASBESTOS SHEETING AND TAPE PROOFED WITH RUBBER. THEY ARE USED IN JOINTING STEAM-PIPES.

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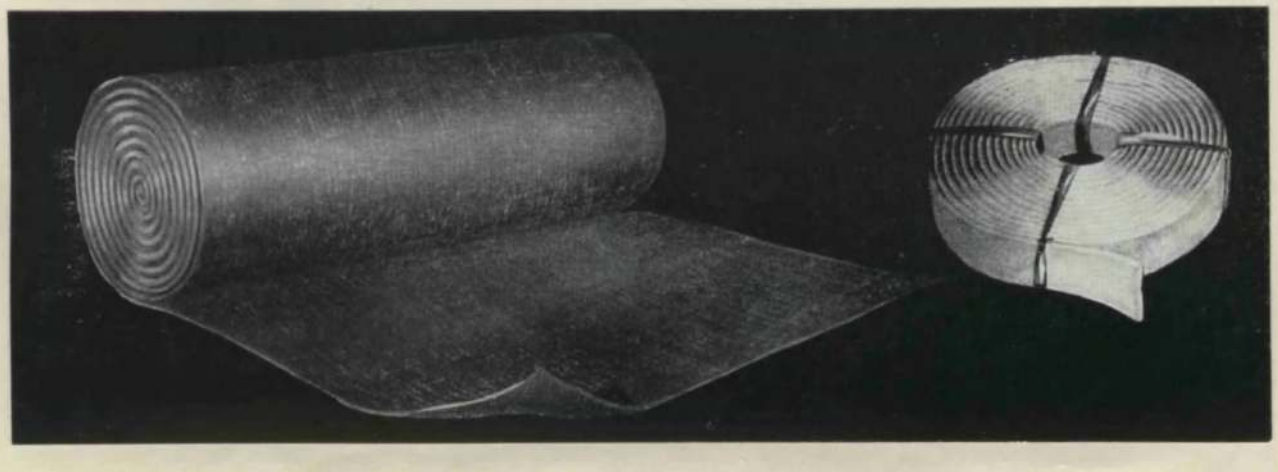


PLATE 55

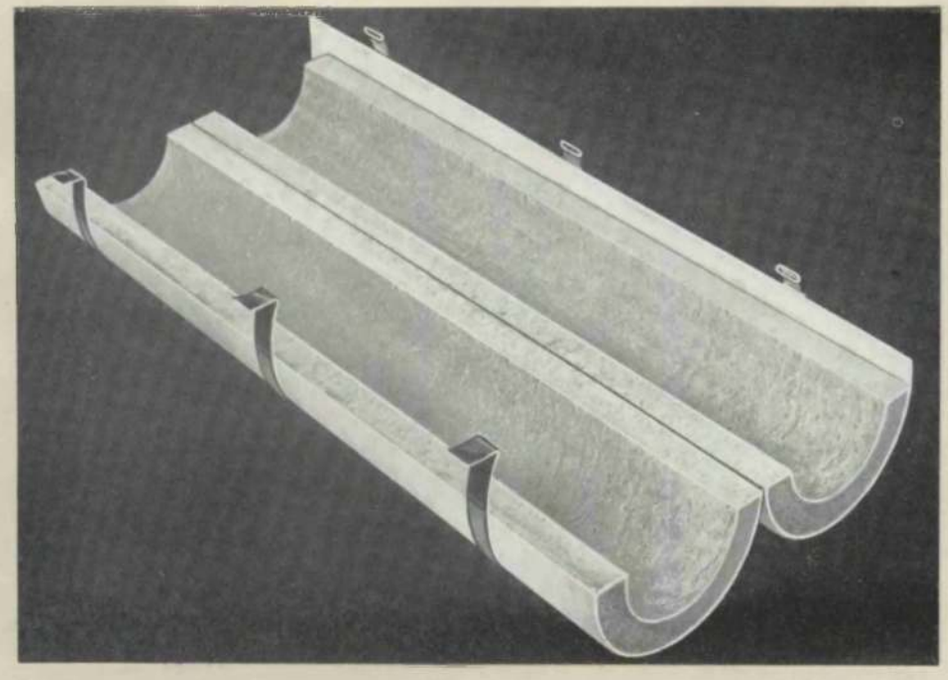
ASBESTOS PIPE SECTION OPENED AND READY TO BE PLACED OVER A STEAM-PIPE FOR CONSERVATION OF HEAT.

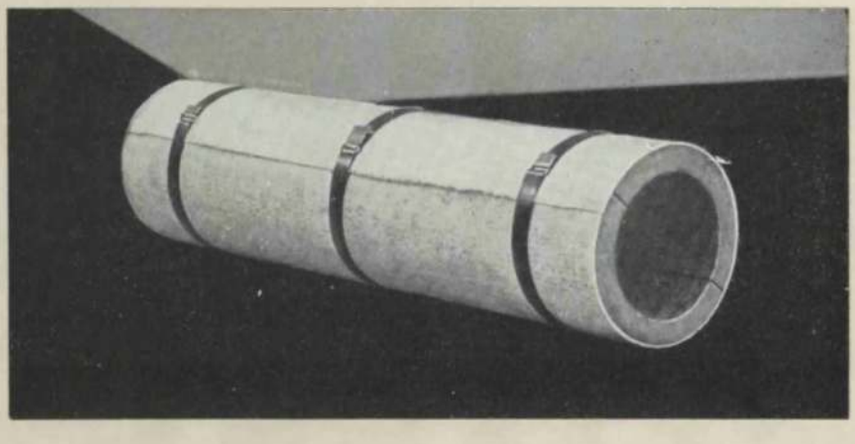
PLATE 56

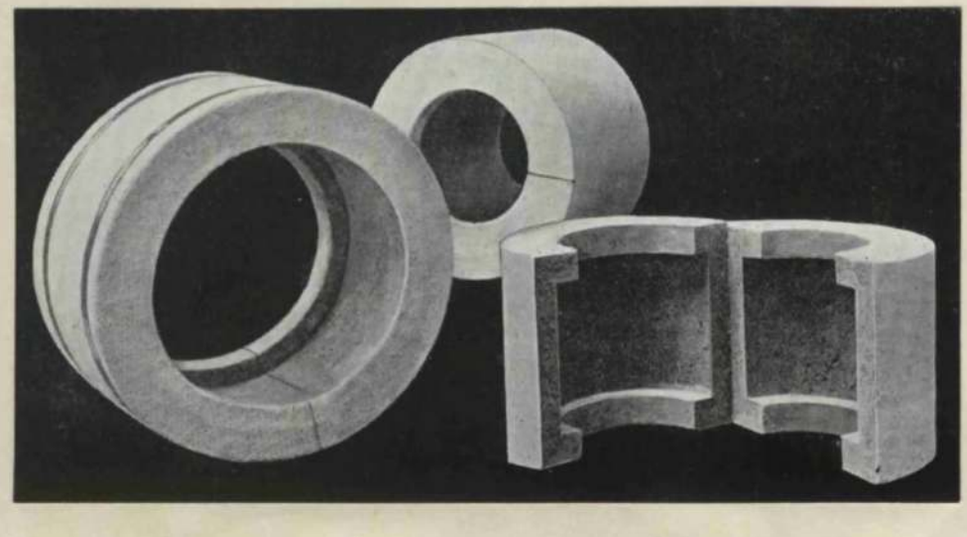
A SIMILAR PIPE CLOSED BY SECURING BANDS.

PLATE 57

MOULDED SECTIONAL FLANGE COVERS.







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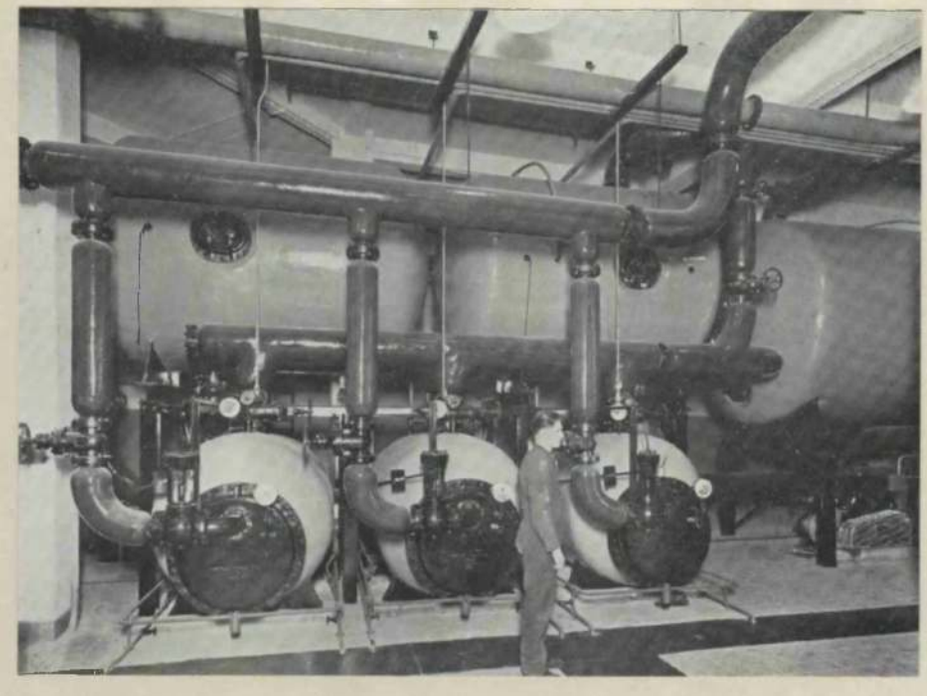
PLATE 58

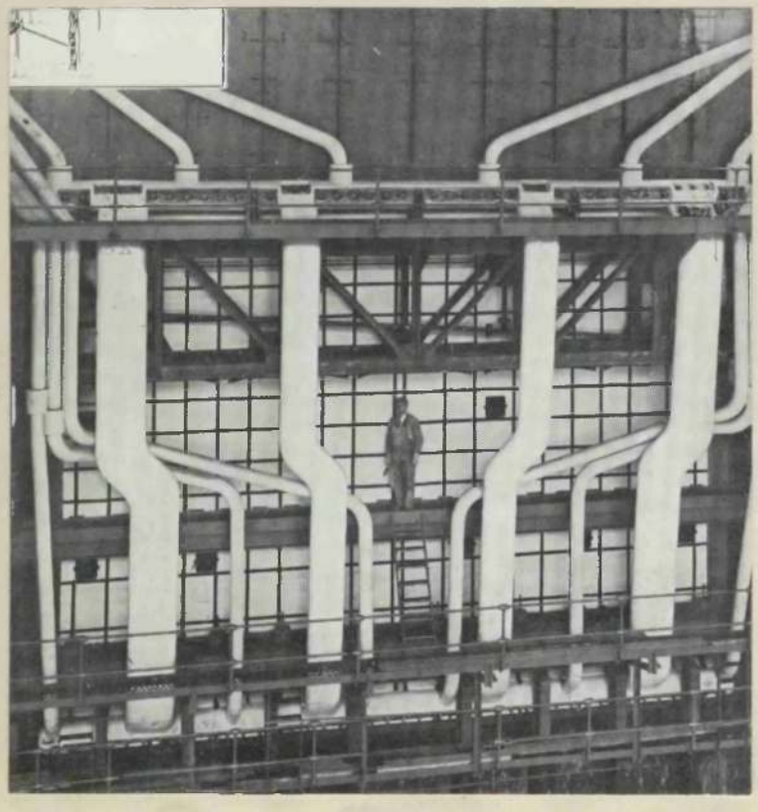
BOILER HOUSE FOR A LARGE BLOCK OF FLATS. BOILERS, CALORIFIERS, STORAGE TANKS AND PIPE-WORK ARE LAGGED WITH ASBESTOS COMPOSITION WHICH IS FINISHED OFF WITH A HARD, SELF-SETTING CEMENT.

PLATE 59

PHOTOGRAPH SHOWING THE COMPLETED LAGGING OF A REAR BAILEY FURNACE WALL INSULATED WITH PURE ASBESTOS FIBRE, CARRIED OUT ON THE PANEL SYSTEM. THE BOILER IS CAPABLE OF A CONTINUOUS RATING OF 210,000 lbs. OF STEAM PER HOUR.

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PLATE 60

ASBESTOS BRAKE LINING IN COURSE OF FITTING TO THE BRAKE POSTS OF A WINDING ENGINE FOR DEEP MINE WINDING. PEAK H.P. 7,800. LOAD HOISTED PER WIND 16,000 lbs.

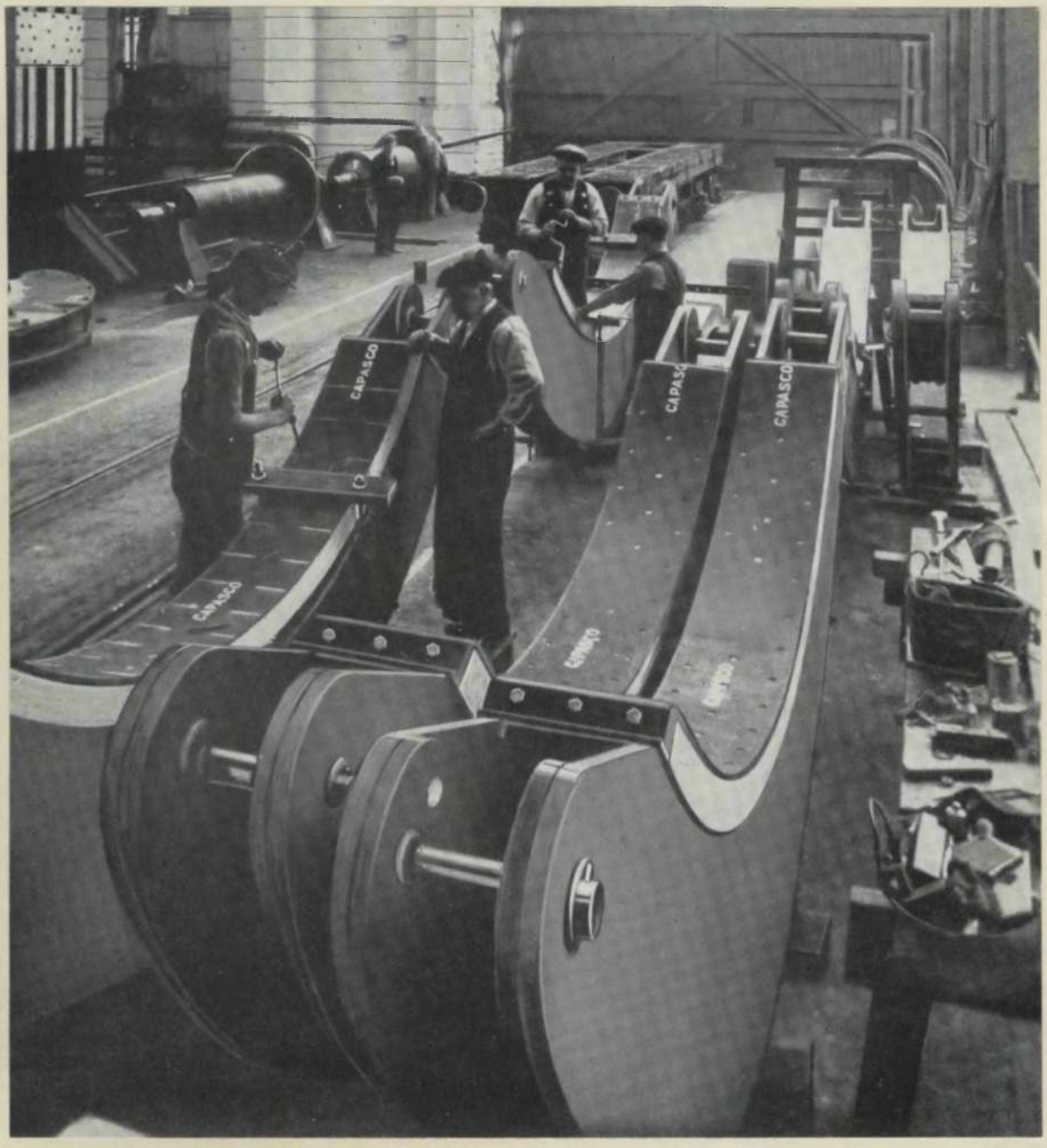
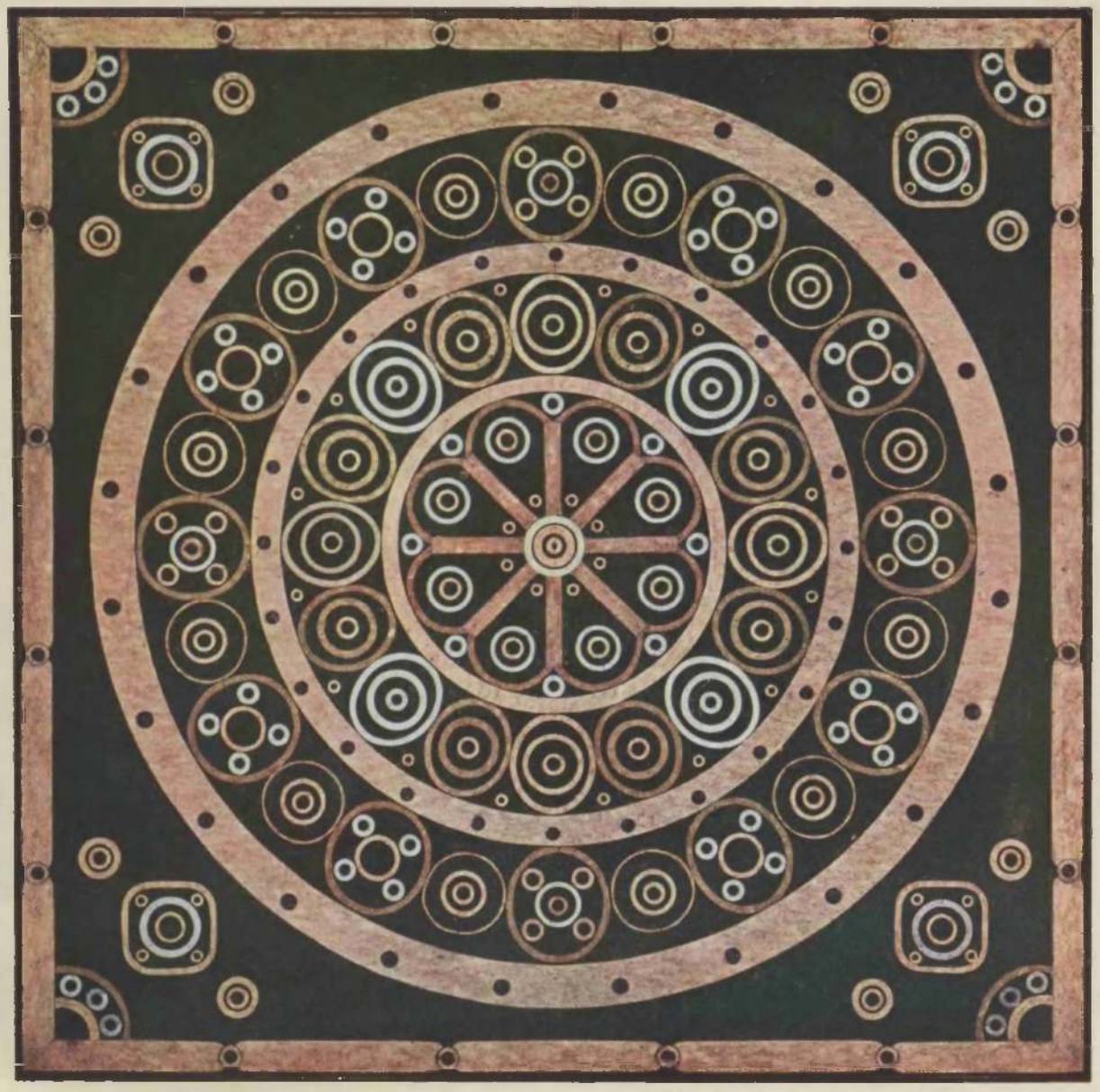

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PLATE 61

A SELECTION OF CUT COMPRESSED ASBESTOS FIBRE JOINTS AND GASKETS. COMPOSED OF ASBESTOS FIBRE AND VULCANISED RUBBER.

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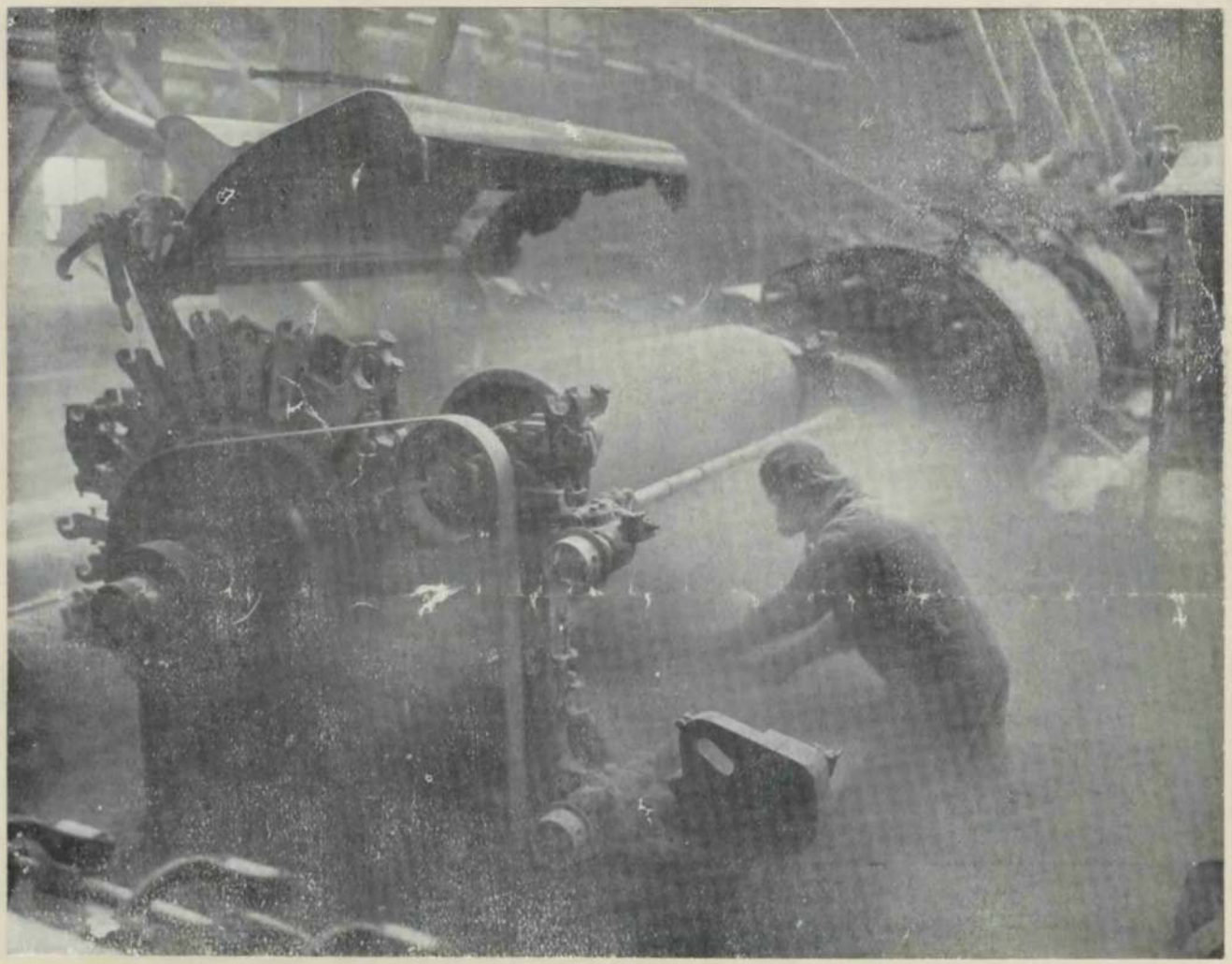


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PLATE 62.

"STRIPPING" AN ASBESTOS CARDING MACHINE BEFORE A MODERN TYPE OF LOCAL EXHAUST VENTILATION WAS APPLIED. REPRODUCTION KINDLY PRESENTED BY DR. HENRY, UNTIL RECENTLY H. M. MEDICAL INSPECTOR OF FACTORIES.

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