Remarks

ON

OCCUPATIONAL DUST.*

BY

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It is usual, in dealing with occupations giving rise to dust and their effect on health, to quote mortality figures for these occupations and to compare them with those which may be considered free from dust. Writers and speakers on this subject invariably turn to mortality tables as indicative of the effects on health in various industries. There is no question that such figures are of the utmost value, but it is not my intention to enter into details of mortality rates for various industries; many and more able men have from time to time emphasized the significance of them. As long ago as 1875, commenting on earthenware manufacture in the Decennial Supplement to the Registrar-General's Thirty-Fifth Report, Dr. Farr wrote: "Earthenware manufacture is one of the unhealthiest trades in the country, and the age of joining it is low; but the mortality after the age of 35 approaches double the average; it is excessively high; it exceeds the mortality of publicans. What can be done to save the men dying so fast in the potteries and engaged in one of our most useful manufactures?"

The works on *Diseases of Occupation* by Arlidge and Oliver are full of valuable mortality statistics for various industries. Collis, in his Milroy Lectures in 1915, deals fully—perhaps more fully than anyone else—with the effect of dusty occupations and mortality rates, emphasizing the effects in particular of the dusts of free silica (SiO_2). Dr. W. F. Dearden, in his Milroy Lectures in 1927, struck a somewhat new note, and with indefatigable care examined the sickness certificates of various groups of workers in cotton-spinning and weaving mills. He elicited therefrom the most interesting and significant figures. This line of inquiry seems to me to be one of the greatest possible value, but it must necessarily be, to some extent, dependent on the accuracy of the certificates given by medical men. Dr. Bradford Hill, on behalf of the Medical Research Council, has carried out investigations on similar lines—one for the committee dealing with the question of the health of workers in humid cotton sheds,¹ and another in connexion with the printing industry.² No doubt further industries will come under similar review, and I think I am divulging no secret when I say that one is now proceeding on behalf of the Dust in Card Rooms Committee. But if we are to complete the picture as to the effect of dust on health, our inquiry must not stop at either mortality or sickness figures. It is essential, when any particular dust is concerned, to probe further and examine groups of workers exposed to such dust, clinically and with all the necessary accompanying examination—x-ray and sputum—and with this we must have a full and complete occupational history of each individual worker in order to study the effects of exposure to dusts of different character. We must go further and confirm our findings, wherever possible, by laboratory experiments.

Dust has been defined as "earth or other solid matter in a minute and fine state of subdivision, so that the particles are small and light enough to be easily raised and carried in cloud by the wind; any substance comminuted or pulverized; powder." Such a definition, in my opinion, would include fume from metal, as it must necessarily be that such fume consists of particles differing only from obvious dust in point of size.

It is fair to state that all manufacturing processes where dry or dried materials are manipulated will give rise to

dust, to a greater or lesser extent. It does not, however, follow that there will be danger to health from the dust in every case. In 1864 the Factory and Workshop Act required every factory to be ventilated in such a manner as to render harmless, so far as is practicable, gases, dust, or other impurities generated in the process of manufacture, which may be injurious to health. In the Factory Act of 1878 an additional requirement was added, to the effect that in a factory or workshop where grinding, glazing, or polishing on a wheel, or where any process is carried on by which dust is generated and inhaled by the workers, to an injurious extent, an inspector can direct that a fan or other mechanical means be provided within a reasonable time. These provisions are similarly required under the Factory and Workshop Act, 1901. The law has interpreted "to an injurious extent" as including every case in which dust is inhaled to such an extent as will be injurious in the long run." I suppose no more difficult task has been put upon medical men concerned with industry, particularly medical inspectors of factories, than to determine whether any one particular dust is or is not likely to be injurious to health "in the long run."

The problem of occupational dust and its effect on health is one of the utmost importance, and as new methods of diagnosis are available, and made use of, in the examination of workers so exposed, it is my belief that we shall eventually be able to answer with confidence the question asked. The problem of injury to health by this means is not merely whether the dust is in itself toxic, but whether it is present in the air breathed in sufficient amount as to produce toxic symptoms. Sir Thomas Legge was, I believe, the first to lay down a degree of concentration of a toxic dust—that containing lead—which would produce poisoning. Legge considered that

"somewhere about 2 milligrams, or 0.002 gram of lead, is the lowest daily dose which, inhaled as fume or dust, may in the course of years set up chronic plumbism. Probably if the air breathed contains less than 5 milligrams per 10 cubic metres of air, cases of encephalopathy would never, and cases of colic very rarely, occur."

This standard has been generally accepted.

Recent experimental work done in America in connexion with the toxicity of petrol containing ethyl lead, by exposing men to exhaust gas air mixtures from engines burning ethyl petrol, showed that the maximum amount of lead retained in the body was 27 per cent., and the average was slightly less than 15 per cent., of the lead dust in the inhaled air. Not only, therefore, is the quantity of the dust present in the air an important factor, but also the amount retained after inhalation, and on this question the effect of the electrical charge in the particles of dust may have an important bearing. But our problem does not end here. The size of the particles would also appear to be of much significance in determining the effect of breathing certain dusts. While particles of quartz of 2 μ and under affect the lungs, our knowledge is as yet very incomplete as to the effect of larger particles of other substances in producing pneumoconioses and other respiratory conditions.

In this paper I can do little but indicate some of the more common forms of injury to health produced by occupational dust. I propose to divide such effects into two arbitrary groups, realizing very fully that some may fall into both groups, and that others included, for want of better knowledge, in one group only, ought properly to be included in both.

Group A: Dusts which exercise a general toxic effect;

Group B: Dusts which exercise a localized effect—(1) internal, (2) external.

GROUP A.

This mainly consists of the dust of certain metals, and chiefly their salts, which produce a definite syndrome. In this group probably the most important syndrome, and one which is still met with most frequently, is due to lead and its compounds. Lead poisoning still figures prominently in the notifications made to the Chief Inspector of Factories under Section 73 of the Factory and Workshop Act, 1901.

* Read in opening a discussion in the Section of Occupational Diseases at the Annual Meeting of the British Medical Association, Manchester, 1922.

I give in the table below the number of cases from various industries over a number of years.

Industry.	1928	1927	1926	1925	1924	1920	1910	1900
1. Smelting of metals ...	36 ²	21 ¹	26 ¹	22	38 ¹	45 ³	34 ⁵	34 ¹
2. Plumbing and soldering ...	11 ¹	11 ¹	7 ³	7	6	3 ¹	25 ¹	9
3. Shipbreaking ...	32	32	8	31	131 ¹	3	—	—
4. Printing ...	3	10	8	8	6 ¹	9	33 ⁴	18 ²
5. Tinning of metals ...	1 ¹	3	2	4	3	2	17	5
6. Other contact with molten lead	9 ¹	17	16 ⁴	17 ¹	23 ¹	13 ²	17 ¹	30 ¹
7. White and red lead works ...	9 ¹	21 ¹	13 ¹	19 ¹	20 ¹	28	44 ¹	377 ⁶
8. Pottery ...	23 ¹⁰	14 ⁶	41 ¹⁴	47 ⁵	47 ¹⁸	25 ¹³	78 ¹¹	210 ⁸
9. Vitreous enamelling ...	23	12	8	9 ²	9	2	17	11
10. Electric accumulator works	33 ¹	58	52	73 ¹	101	47 ²	31	33
11. Paint and colour works ...	12 ¹	6	10	8	15	9	17 ¹	56 ¹
12. Indianrubber works ...	1	2	2	2	3	7	3	1
13. Coach and car painting ...	18 ³	14 ³	14 ²	23 ¹	30 ⁴	13	70 ⁶	70 ⁵
14. Shipbuilding ...	7 ²	10 ¹	8	13	8 ¹	9	21 ²	32 ²
15. Paint used in other industries	11	7	16 ³	14 ¹	24 ²	10 ¹	51 ³	50 ⁵
16. Other industries ...	10	11 ¹	11	29 ¹	22 ²	18 ¹	47	122 ⁷
17. Painting of buildings ...	87 ²⁰	98 ²¹	95 ¹⁸	88 ⁹	75 ⁹	46 ²¹	—	—
Total ...	326 ⁴³	347 ³⁵	332 ⁴⁶	414 ²²	561 ⁴¹	289 ⁴⁴	505 ³⁸	1058 ⁵³

The principal figures relate to cases; the raised figures to deaths.

I will not deal with the question of lead poisoning in detail, but simply consider the important part which lead poisoning plays in industry. There is, however, one point in connexion with lead excretion to which I should like to draw attention. Kehoe and other American observers, in investigating the effect of ethyl lead petrol, found that lead was excreted in measurable quantities in the urine of normal healthy persons who had not been exposed to any known risk of lead absorption. These findings were confirmed on a small scale by Professor Chaston Chapman, a member of the Ethyl Petrol Committee; they are of some importance, for the presence of small quantities of lead in the urine has often been regarded as confirming a diagnosis of lead poisoning in a lead worker.

Arsenic.—The classical symptoms—gastro-intestinal irritation, accompanied by abdominal pain—do not often occur in industry due to the inhalation of dust containing arsenical compounds. In the last ten years there have only been three cases so described in the reports received as the result of notification. This is somewhat surprising in view of the large quantities of arsenic oxide used in the manufacture of compounds containing arsenic, especially sheep dip. Some years ago, in the manufacture of Scheele's green (copper arsenite), symptoms of peripheral neuritis were observed. In general, the effect of arsenical dust is manifested by localized affections of the skin, and to these I refer later.

Manganese.—In 1922 Dr. Charles of Bristol drew the attention of the department to two cases of manganese poisoning affecting men who were employed in grinding up manganese ore. The symptoms of manganese poisoning may be briefly summarized as follows: languor, sleepiness; mask-like facies; change in the voice, which becomes monotonous and low; retropulsion and, in some, propulsion may occur early, gait later becoming spastic; tremors of the head and limbs, fine in character, with pain and cramp in the muscles, chiefly of the legs; a slight increase in the tendon reflexes. The mental condition is altered, sometimes with hysterical outbursts of crying and laughing. These cases, in the advanced stages, do not recover, though the disease does not appear to progress if the patients are removed from the toxic atmosphere. The disease is not fatal. Cases have been reported in America by Davis and Huey³ in the manufacture of manganese steel. I give the symptoms somewhat in detail as, so far as I know, no cases have occurred in this country from a similar occupation or

from other occupations where manganese dioxide is used, such as in the manufacture of dry battery cells.

Zinc.—Zinc powder in the cold does not appear to give rise to symptoms characteristic of zinc poisoning—namely, febrile reactions, shivering, headache, followed by cold sweats—but, evolved in the form of fume from molten zinc in brass founding, over the pots in the manufacture of zinc oxide, these symptoms are frequently observed. Some years ago cases of the same character were observed by myself among men employed in breaking up galvanized metal by means of an oxy-acetylene flame. From the experimental work done by Drinker, Thomson, and Finn,⁴ similar reactions are produced by breathing fumes of other metals. These authors state that it is probable that the fumes from the burning of iron, nickel, and aluminium in the electric arc would also cause the same general type of febrile reaction, which they term "metal fume fever." It was also found by them that the inhalation of mild doses of magnesium oxide, generated by the burning of magnesium ribbon, produced a leucocytosis and a febrile condition resembling that caused by heavy metals.

Cadmium. which is present in zinc ores, when oxidized has been known to give rise to symptoms due to inhalation. Stevens of Swansea⁵ drew attention to this form of poisoning, and found cadmium in the liver of a workman whose case had been diagnosed as lead poisoning. A fatal case was investigated by Legge,⁶ in which cadmium was being heated by gas without exhaust ventilation. Two of the workers became seriously ill and an engineer who was supervising the operation died. The symptoms were dryness of the throat, headache, rapid pulse, nausea, and shivering, with brown discoloration of the urine. In the fatal case there was an inflammatory condition of the respiratory and intestinal tracts, fatty degeneration of the heart and liver, haemorrhagia, inflammation of the spleen, and inflammation of the kidneys.

Nitro and Amido Derivatives of Benzene.—The salts of aniline and of the nitro derivatives of benzene and its homologues, from which, in the course of manufacture and handling, dust arises, produce the classical symptoms of anilism—that is to say, cyanosis and dyspnoea. Some of the dust is probably inhaled, but there is also grave risk of this form of poisoning from absorption through the skin. During the past five years aniline poisoning from dust has been mainly reported in connexion with paranitraniline powder, but a number of cases were also found to be due to exposure to dust from dinitrobenzene. Changes in the blood produced by aniline were investigated by Dr. Williamson, in connexion with the work of aniline black dyeing. Dr. Williamson considered that although the number of workers examined was insufficient to justify final conclusions, briefly the order in which the various elements of the blood are affected by aniline was as follows: First, a diminution in the percentage of haemoglobin, which may be altered in its chemical constitution. Following a fall in the percentage of haemoglobin there is a fall in the red corpuscles per cubic millimetre, which, though never very marked, is still quite definite. Lastly, there may be a diminution in the number of white corpuscles, with a relative increase in the number of lymphocytes.⁷ During the war much trinitrotoluene in powder form was used in the manufacture of munitions, and, partly by inhalation of the dust, or perhaps more particularly by absorption of the dust through the skin, cases of severe jaundice (toxic jaundice) occurred. Such cases, I am glad to say, are not occurring at the present time, but dinitrobenzol, in a similar way, gives rise to serious symptoms, the chief being cyanosis, gastritis, amblyopia, and occasionally peripheral neuritis. I have also observed in severe cases some degree of icterus. Fatal cases have occurred. As regards chronic cases, anaemia of the aplastic type is the chief feature.

Organic Dusts.—Dusts acting as carriers of toxic substances or organisms, and producing symptoms of a definite general character, must not be forgotten. Fur dust after carotting—that is, painting with nitrate of mercury—has produced mercurial poisoning. Wool-sorters' disease owes its name to the generalized anthrax occurring among wool workers from the inhalation of dust carrying spores of the anthrax bacillus. I am glad to say there

is a remarkable decline in this generalized infection, which is invariably fatal, as shown in the following figures:

Period.	Cases (all fatal).
1909-1913	13
1914-1918	15
1919-1923	10
1924-1928	2

In this group of occupational dust diseases I think we may include those caused by moulds. To take an instance of special interest in Lancashire, there is the condition commonly known as "weavers' cough," where, although the main symptoms are referred to the lung, yet there are general disturbances associated with the lung conditions—namely, headache, thirst, and a feeling of extreme lassitude. Dr. Collis⁸ formed the opinion that this form of illness was due to the inhalation of the spores of a mildew. Subsequent outbreaks have shown more or less similar symptoms, and the presence of mildew in warps has been observed. So far, however, it has been impossible to identify any particular form of fungus. In this connexion it is well to observe that to establish a definite relation between a lung disease of this description and the causal agent it is necessary to identify a similar fungus in the sputum or lung juice. Closely allied to such a condition is, I believe, the cough which I investigated a few years ago, affecting men working in malt-houses and exposed to a considerable quantity of dust. Here again, although able to show a fungus of pathogenic variety in the dust, we have up to the present been unable to identify the same fungus in the sputum.⁹ No doubt there are other dust-borne diseases due to the presence of a fungus in the dust—for example, in tea dust.¹⁰

GROUP B (1).

The most important and widespread forms of localized internal disease produced by dusts are the various forms of pneumoconiosis. It seems to me that the time is not far distant when it would be as well to consider the best nomenclature of the fibrous diseases of the lung produced by dust. The term "silicosis," as denoting fibrosis caused by the inhalation of free silica, is so well established that it would be difficult to alter it, even if it were desirable to do so; but I have recently seen "tobaccosis" as describing an effect on the lung—a term which, to say the least, seems cumbersome.

Of the various forms of pneumoconiosis, silicosis is at the present time by far the most important. To Collis belongs the credit of drawing attention to the effects of the inhalation of occupational dust containing free silica (SiO_2) in this country, but even Collis did not visualize, I think, the widespread nature of the industries in which dust containing free silica occurs. The recent scheme for compensation under the Various Industries (Silicosis) Scheme gives some indication of this. Previous schemes dealt with silicosis in the Refractories Industries and the Metal Grinding Industries. Silica dust occurs in such widely different industries as the manufacture of pottery, the making of scouring powders containing silica flour, the sandblasting of castings, the making of poultry grit by grinding and crushing of flints, tin and coal mining, and the making of vitreous enamels, as well as in that large industry the sandstone industry, affecting quarriers and masons. The subject of silicosis has, I am glad to say, been dealt with by my colleague Dr. E. L. Middleton, whose unique knowledge of the subject makes any further observations by me unnecessary. (See *Journal*, September 14th, p. 485.)

In 1924 a case of fibrosis of the lung caused by the dust of asbestos was described by Dr. W. E. Cooke,¹¹ in which he pointed out, in addition to the fibrosis, the presence of certain bodies which have since come to be known as "curious bodies." The first recorded fatal case of fibrosis of the lung in an asbestos worker was that described by Dr. H. Montague Murray in his evidence before the Departmental Committee on Compensation for Industrial Diseases, in 1906. The death occurred in 1900, and Murray stated that in the seven years he had not come across another case. In February of last year Dr. Macgregor, medical officer of health for Glasgow, drew

the attention of the Factory Department to an asbestos worker who was receiving treatment in one of the city's hospitals. In March, 1928, the death of an asbestos worker under the care of Dr. Grieve occurred, and at the post-mortem examination the condition of widespread fibrosis of the lung, without tuberculosis, was revealed. A further case, which also terminated fatally, occurred five months later. Recently Professor Stewart¹² of the University of Leeds has shown that the "curious bodies" described by Cooke could be obtained during life by puncture of the lung of an asbestos worker, in the sputum, and after death in the lung juice. It is not suggested, I understand, that the presence of these "curious bodies" indicates fibrosis of the lung, but merely that the occupation of the individual exposes him to asbestos dust.

It may be the subject of speculation why this disease has only recently attracted notice and become a problem in this country. A number of factors have probably contributed to obscure its existence among workers in asbestos, of which the most important seem to be the comparatively small numbers of workers engaged in essentially dusty processes, the insidious nature and general slow progress of the disease, and the difficulty of accurate diagnosis, particularly where tuberculosis is superimposed, as occurred in the first case reported by Dr. Cooke. It may be, too, that the application of exhaust ventilation to the dusty processes in this industry has checked the earlier evidence of the disease by materially reducing the concentration of the dust. Dr. Cooke, in his paper on the subject of fibrosis of the lung produced by asbestos, will, I hope, in addition to other interesting particulars of this condition, throw some further light on the presence of the "curious bodies" which he has described.

In a report of the Director-General of Public Health for New South Wales, for the year ended December 31st, 1927, Dr. Charles Badham, medical officer of industrial hygiene, contributes a section which he calls "Notes on a fine type of fibrous pneumoconiosis produced by silicates and other minerals." This type of fibrosis, which Dr. Badham desires to call "silicatosis," is found, he says, only after exposure to certain mineral dusts, and can in most cases reasonably be assumed to be caused by various silicates. It is not found in workers who have only been engaged in activities where they are exposed to a high percentage of quartz—that is, 70 to 90 per cent.

Dr. Mavrogordato, in his *Studies in Experimental Silicosis and other Pneumoconioses* in 1922, found, after subjecting the lung of an animal for eight weeks to dust free from uncombined silica, that most of the dust remained within the cells, and these had not become aggregated, but were scattered more or less uniformly throughout the tissues. Dr. Badham, commenting upon these findings, suggested that the fibrosis found was probably the fine type that he describes.

Collis and Gilchrist,¹³ who have studied the effects of the inhalation of coal dust upon trimmers, state that "the clinical observation by x-ray examination discloses that, after many years of work, the lungs of coal trimmers are not normal, and exhibit signs similar to those widely regarded as characteristic of silicotic fibrosis." American workers have reported the appearance of pneumoconiosis among cement workers, and Dr. Badham in his notes mentions a case of a fine type of fibrosis which he has seen in a cement worker in New South Wales.

These few observations show, I think, that we are but at the beginning of our knowledge as to the effect of the inhalation of mineral dusts upon the lungs of workers exposed to such dusts. It may well be that in time, with a more complete survey of workers exposed to particular dusts, it will be possible to determine, by clinical and radiological examination, the particular form of dust by the character of the fibrosis to which it gives rise. I hope that the inquiry which is now being carried on by my colleague Dr. Merewether of the Factory Department, into the effect of asbestos dust, will materially assist in this direction.

Vegetable Dusts.

Of the effects of vegetable dusts on the lungs little or nothing is known. In 1924 Dr. Middleton with Mr. McNair, H.M. Engineering Inspector of Factories,¹⁴ made some investigations into the dust to which workers were

exposed in the processes of discharging and handling of grain cargoes. Dr. Middleton collected samples by means of Owens's dust-counter, and examined these samples. He found that the dust in the atmosphere consisted of two kinds: (1) sharp spike-like hairs, and (2) fragments of vegetable and mineral matter, spores, etc. Dr. Middleton formed the conclusion that the dust in the case of wheat, barley, rye, and oats contains hairs derived from the grain which on *a priori* grounds appear to be capable of causing irritation of, and inflammatory changes in, the respiratory passages. No other constituent was discovered in the dust met with during this investigation which would be capable of producing the symptoms experienced by the investigators or of causing serious injury to health.

Dr. Middleton investigated a case of loss of sight in a woman employed for twenty-five years at a bench where she had been exposed to inhalation of pure tobacco dust, most of it caused in the intermittent process of sieving. The case—the first recorded in this country—was diagnosed by the ophthalmic surgeon of a general hospital, and other possible causation, as far as possible, excluded.

One cannot leave the effects of occupational dust on the respiratory organs without reference to the increase in cases of cancer of the lung, to which Dr. Duguid drew attention in an article published in the *Lancet* of July 16th, 1927. His observations are intriguing and suggestive, particularly when read in conjunction with the work of Rostoski and Schworl on lung cancer. Nor must we forget that it was from a survey of cases from the Manchester Royal Infirmary, by the late Dr. Wilson and by Dr. Southam, that the now well-recognized cancer of the scrotum among mule-spinners was first brought to light.

The part played by inhaled dust in the etiology of cancer of the bladder cannot be ignored. The following table gives particulars of cases occurring in workers in the manufacture of synthetic dyes in chemical works since 1924, which have come to the notice of the Factory Department.

Initials.	Age at recognition.	Occupation.	Result.
R. J.	41	Collier 13 years, army 4 years, colour drier in chemical works 6 years	Died Sept. 17, 1925.
J. W.	54	Bra emaker 22 years, in soap department of bone factory 11 years, army 1 year, filling casks with size 2 years, burning sulphur stone 2 years, in chemical work 6½ years	Died in 1927.
A. S.	38	Brush stock wood turner 16 years, in chemical works 10 years	Died June 30, 1925.
A. B.	48	Worsted spinner 12 years, wool comber 11 years, in chemical works 18 years	Died July 10, 1924.
W. B.	66	In chemical works 20 years	Died Nov. 11, 1925.
A. S.	51	Aniline black dyer 20 years, in chemical works 20 years	Alive.
W. A. S.	60	Mechanical printer 20 years, in chemical work 20 years, picric stor keeper 7 years	Died Sept. 29, 1925.
T. G. G.	63	Purifier of anthracene 6 years, in chemical work 33 years	Died Dec. 9, 1924.
J. T. F.	73	In chemical works 2 years, watchman years	Died Dec. 16, 1925.
R. H.	56	In chemical works 33 years	Died Dec. 19, 1924.
W. T.	52	In chemical works 38 years	Died Aug. 19, 1927.
J. N.	63	In chemical works 48 years	Died Aug. 13, 1927.
W. H.	61	In chemical works 21 years	Died Apr. 22, 1927.

A few fatal cases of cancer of the bladder have also been noted among pitch workers. Their occurrence may be of importance.

GROUP B (2).

The localized diseases produced by occupational dust are those affecting the skin and mucous membranes. Many may not agree that the skin lesions are localized, but often general, affecting in some cases the whole surface of the body, yet I believe it correct to say that the starting point is a particular area, and for this reason I include them in this group. It is an interesting fact, and I say it with due deference, that the general extension of a localized skin lesion produced by dust has not been fully explained.

There are few, if any, dusts occurring in industry that will not produce, in certain individuals, an inflammatory condition of the skin, and this not only makes difficult the decision as to whether or not a dermatitis is of occupational origin, but also the actual causal agent in many cases a matter of some doubt. The importance of the dermatoses produced by dust, and the loss to industry by such conditions, is apparent by the number of cases for which compensation was paid for dermatitis produced by dust or liquids in the last five years—namely, 390 in 1923; 564 in 1924; 691 in 1925; 837 in 1926; and 1,029 in 1927. The marked increase in the number of cases ascribed to industry is difficult to attribute solely to altered industrial conditions.

It would be impossible to refer to even a small number of the dusts which produce skin reaction, nor would it be profitable. I feel I am treading on holy ground when I so much as mention occupational diseases of the skin in Manchester: the work of Prosser White in connexion with these is so well known, not only in Manchester and Lancashire, but wherever these diseases engage the attention of those interested in industrial medicine. From my own experience the dusts which appear to exert the most rapid and severe conditions are the nitro compounds of benzene and its homologues—for example, dinitrochlorobenzene, trinitrophenol (picric acid), paranitraniline. Of simple compounds, the sodium and potassium salts of chromium and the caustic alkalis produce not only skin lesions, but ulceration of the mucous membranes and septum of the nose, leading to complete perforation. The absence of untoward symptoms following such definite destruction of the internal nares, as is so often seen, is a matter of surprise to me. I have never seen any serious disturbance in the normal functions following this loss, nor, so far as I know, has any case of such a nature been recorded. One might have supposed, at any rate, that the septic infection necessarily occurring during the process of ulceration would, in some instances, extend beyond the cartilage and bones of the internal nares.

Sometimes it is not the dust itself which produces the skin lesions. I well remember an outbreak of rash affecting men unloading a cargo of barley from a ship, which I investigated. Not only were the workers affected, but the crew of the vessel, including the master, also suffered. Intense itching was the chief cause of complaint, and there was no difficulty in demonstrating the cause to be due to the presence of a mite, the *Pediculoides ventricosus*. Although the cause was easy to find, the cure and prevention were different matters.

However distressing and economically wasteful simple lesions of the skin produced by practically all dusts may be, they are as nothing compared with cancer of the skin produced by dust. Epithelioma produced by pitch and arsenic dust must be of special interest in Lancashire, owing to its similarity to the cancer affecting mule-spinners. Even though the main incidence occurs among the patent fuel workers in South Wales, the pitch workers in gas works, the loaders and unloaders of pitch, and the workers exposed to pitch dust in tar distilleries, wherever they may be, share the risk. The number of cases reported since the disease became notifiable in 1920 is as follows, and for interest the number of cases among mule-spinners is also given:

Industry.	1920	1921	1922	1923	1924	1925	1926	1927	1928
Pitch workers ...	32	24 ²	23 ¹	25 ²	23 ¹	35 ⁴	49	34 ¹	32
Mule-spinners ...	—	—	—	15 ¹	79 ¹⁷	75 ³⁵	88 ⁶	101 ³¹	101 ²⁶

The principal figures relate to cases; the raised figures to deaths.

The higher incidence of mortality from cancer of the skin among coal trimmers exposed to coal dust has been pointed out by Collis and Giehrst. What relationship there is between the disease and this occupation is as yet speculative, but it is significant.

Though the number of cases of skin cancer among workers in arsenic is few, yet the connexion between their exposure to the dust and the disease is, I think, well established, and confirmed by the animal experiments of

Leitch and Kennaway.¹⁵ The occupation chiefly affected is that of the manufacture of arsenical sheep dip.

Of the oldest form of cancer of the skin—namely, that of chimney-sweeps, the number of cases recorded is small, though still occurring. In 1927 two received compensation. The spread of soot on the land has possibly been responsible for cases among agricultural workers. One case which I believe may have been thus caused came to my notice last year.

This brief review will, I hope, give some idea of the extent of the problem of occupational dusts; but the problem does not end when it has been established with certainty that any particular form of dust is a cause of a pathological condition. While once the masters of cure, we are now, or ought to be, the slaves of prevention.

Prevention does not, in the case of disease produced by occupational dusts, rest with the medical profession, although we may be able to assist. The sure and only certain way of preventing dust affecting the worker is to prevent its formation, or, if this is impossible, to secure its removal before reaching the workers. It therefore seems to me that just as there is need for research on the medical side in regard to the effects of dust, the methods by which dust can be eliminated, either by a modification of the process or alternatively by effective removal, call no less for research. Chemists and engineers take so very great a share in the prevention of disease that I look with hopeful anticipation to their efforts. Our own task may be the easier, but though their difficulties be great, they are, I believe, not too great for our colleagues in the other sciences to overcome. A dust-free industry may be ideal, but it should be our goal.

REFERENCES.

- ¹ Artificial Humidification in the Cotton Weaving Industry: its Effect upon the Sickness Rates of Weaving Operatives, by A. Bradford Hill, Ph.D. (Report No. 48; Industrial Fatigue Research Board, 1927.)
- ² An Investigation into the Sickness Experience of Printers, by A. Bradford Hill, Ph.D. (Report No. 54; Industrial Fatigue Research Board, 1929.)
- ³ *Journal of Industrial Hygiene*, December, 1921.
- ⁴ *Ibid.*, May, 1927.
- ⁵ *Ibid.*, August, 1920.
- ⁶ Annual Report of the Chief Inspector of Factories, 1923, p. 74.
- ⁷ Annual Report of the Chief Inspector of Factories, 1921, p. 75.
- ⁸ Report on Unusual Illness of Weavers of Cotton Cloth, by E. L. Collis, M.B., H.M. Medical Inspector of Factories, 1913.
- ⁹ Annual Report of the Chief Inspector of Factories, 1926, p. 95.
- ¹⁰ *Manual of Tropical Medicine*, by Castellani and Chalmers, third edition, 1919, p. 1880.
- ¹¹ *British Medical Journal*, December 3rd, 1927, p. 1024.
- ¹² *Journal of Pathology and Bacteriology*, vol. xxxii, 1929, p. 172.
- ¹³ Effects of Dust upon Coal Trimmers, by E. L. Collis and J. C. Gilchrist, *Journal of Industrial Hygiene*, April, 1928.
- ¹⁴ Annual Report of the Chief Inspector of Factories, 1924, p. 103.
- ¹⁵ *British Medical Journal*, December 9th, 1922, p. 1107.

SOME POINTS IN CONNEXION WITH CATARACT EXTRACTION.

BY

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THE advice given to cataract patients in Europe varies so greatly with different surgeons, and is so often opposed to my own practice, which has been founded on many thousands of operations for cataract, that in compliance with requests from various sources that I should restate my views, I desire to do so shortly.

The Stage at Which the Operation Should be Undertaken.

The usual advice given is that a patient must wait until the cataract is mature before undergoing operation, even if the sight in the opposite eye is so defective as to make him or her nearly helpless, or at least partly dependent on others. The first point I would insist on is that the exact date for operation should be decided after a careful consideration of all the factors in each individual case. One cannot lay down a general hard-and-fast rule for the guidance of students. The patient's age, the state of his health, his prospects of life, the condition of the second eye, and a number of other factors must be carefully

weighed before giving any definite advice. A few points may be considered with profit.

A patient is frequently told that if the second eye sees well, it is a mistake to have the first one touched. Such advice seems to me eminently unsound in a number of cases.

1. To be blind on one side exposes a man to injury. He runs into people, lamp-posts, etc.; moving objects run into him because he has not sufficient warning of their approach to avoid them. Serious accidents happen in this way, especially from bicycles and trade tricycles ridden by careless young people. It is admitted that one cannot fit up an aphakic eye with a lens and enable it to work accurately together with an unoperated and useful eye on the other side, but this does not mean that an aphakic eye cannot be made a very useful organ. On the contrary, it can, even when uncorrected by glasses, be made to add a large element of safety and security to the patient's life as well as to his comfort and happiness—no unimportant matter.

2. Cataract operations are practically always performed on elderly people, and elderly people go downhill, so far as health and resistance are concerned, at a rate quite disproportionate to that met with in the young or even in the middle-aged. Apart altogether from disease, every year lessens a patient's resistance to operation. He is a better subject at 70 than at 71. This is even more the case between 80 and 81, and so on. A case that will do well one year may easily end in failure a year or two later. The incidence of an attack of influenza, of bronchitis or bronchial pneumonia, of a stroke, or of many another illness, may just make the difference between a patient being a good subject for an operation or a very bad one. The opportunity of successful operation may thus be lost, never to return, with the result that the man is condemned to permanent blindness. All the interest is thus taken out of his life, and he too often sinks to the level of a human cabbage, a burden to himself and to all about him. This does not mean to say that any patient is too old for a cataract operation, provided there are sufficient indications for performing it, but it is a strong argument against putting off operation. Much more depends on the patient's actual state of health than on the number of years he has lived. One man is old and worn out at 55, another is playing games and hunting at 60, or is still robust and leading an active life at 70 or even at 80. I have three times operated at 90 or over. All three patients did well, including an old lady of 96.

3. No patient should be allowed to go blind and to drop his normal activities. With modern methods of operating, any lens can be safely removed, however immature the cataract that darkens its vision. More care is required and the convalescence may be lengthened owing to the need to allow left-behind cortex to be absorbed, but, always provided the operation is skilfully and aseptically carried out, the added risks are, in my experience, so slight as to be almost negligible, and certainly not worth weighing against the serious disadvantages of allowing an elderly person to drop the activities of a lifetime with the risk that he will never take them up again. It is much easier for the old to drop their habits than to take them up again, and few things are more tragic for the relatives than to look on helplessly at the decay of everything which once endeared the patient to them, or brightened the life of one they loved. When a patient reaches the stage in which he can no longer get about comfortably, or in which he cannot read, write, and carry on his usual occupations with moderate comfort, or cannot do either of these things, he should be operated on without delay.

4. The hypermaturity of a cataract may bring in its train definite risks to the eye. These are less appreciated in Europe than they are in India, for in the great majority of cases over here a cataract is removed as soon as it is mature, or very soon after, whereas in India, owing to the ignorance of the people, to the difficulties of obtaining skilled relief, and to other causes, a very large number of patients are allowed to retain their mature cataracts for years, and often indeed up to death. The opportunities for observing the dangers consequent on hypermaturity are therefore much greater in India than in