EFFECTS OF DUST IN INDUSTRY ON DISEASES OF THE LUNGS AND ITS PREVENTION

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INTRODUCTION

Dust diseases are continually brought to the attention of the public. There is good reason to believe that the information in the hands of the layman is very meager, and even those rated as experts have more or less a theoretical conception of the diseases, their causation, remedies, and prevention rather than actual practical experience with the various diseases caused by dust.

Not until recently has attention been given to prevention, so the problem fell to the engineer to solve. This deplorable state is now beginning to be changed for in the past few years numerous conditions have arisen to cause those engaged in industry to bring the subject before the public with a view to ascertaining the facts upon which to formulate remedial measures, primarily preventative.

I. Definition and classification of dust

In Webster's unabridged dictionary dust is defined as fine, dry particles of earth or other matter so comminuted that they may be raised and wafted by the wind; that which is crumbled to minute portions, fine powder.

Richardson[1] 1876, included in the term "dusts" all those fine and solid particles thrown off from various substances in the process of manufacture or treatment of articles in common use in daily life.

Drinker[2] in 1930 defined dust as solid particles ranging in

1. Harrington and Davenport. U. S. Bureau of mines. Circular #6835.
2. Ibid.

size from less than one micron to about one hundred and fifty microns.

Dusts have been classified in various ways. A brief mention of the various classifications shall be made. Richardson[3] classified them as follows:

1. Cutting dust
2. Irritant dust
3. Inorganic dust
4. Soluble saline dust
5. Obstructive and irritating dust

Baskerville in 1912 classified dust as:

1. Insoluble inorganic dust
2. Soluble inorganic dust
3. Organic dust

Hoffman in 1918 classified dust as follows:

1. Inorganic dusts
2. Organic and miscellaneous dust

Thompson classified dust as:

1. Insoluble inorganic dusts
2. Soluble inorganic dust
3. Organic dust

Schurman[4] differentiated dust, according to origin, into animal, plant, mineral, and dust from artifacts.

All these classifications are without much practical importance, since in daily practice in industry the harmful effects is

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3. Harrington and Davenport. U.S. Bureau of Mines circular #6835.
4. Ibid.

exercised most frequently by dusts of mixed origin. Still more important is the physical and chemical nature of dust and their effects on the system.

II. Origin of exposure to dust

Prehistoric man who originated the trade of making stone implements probably began the first industrial hazard, the extent and severity of which the medical profession as well as industry has begun to realize only recently. At the site of the Swiss lake dwellers where flint implements were found, evidences of their manufacture are found evidenced by the profuseness of chips scattered about. It is entirely conceivable that many individuals engaged in this occupation suffered from respiratory diseases. According to Collis,[5] the flint knappers of Brandon, the lineal occupational representatives of this oldest of industries who still use tools similar to those used by their ancestors suffer a terrible mortality from phthisis induced by flint dust generated in their work.

Dust of various kinds are carried in the atmosphere in all parts of the world and the inhalation of these dusts over periods of years inevitably produce changes in the lungs, as for example the pigmented lung of a city dweller. Another example of the general exposure to dust is in a report by Soper in 1906 on air and dust in the subway of Interborough Rapid Transit Company of New York City. Chemical analysis of dust showed it contained 61.30% of organic matter and animal origin, 15.58% silica, and

5. Harrington & Davenport. U.S. Bureau of mines circular no. 8635.

1.18% oil. There was an average 61.6 mg. of dust per 1000 cubic feet of air. The source of which was from street dust, underground origin from gradual wear and tear of wood, cement, and other materials used in construction of subway. The men had not been employed underground long enough at that time to show effect on health of inhalation of dust. Although no serious diseases were found, many suffered from inflammatory affections of the nose, throat and trachea and also from "dry pleurisy" unaccompanied by pain.

Hoffman,[6] 1918 prepared an occupational grouping of hazardous dust industries putting 65 into the organic and miscellaneous groups and 52 occupations under inorganic dusts. He gave the number of persons employed in dusty trades, according to the United States Census for 1910 as 3,928,978, of these 1,667,181, were listed as employed in occupations exposed to metallic and mineral dust and dust in the mineral industries. Mineral dust is the most common in stove industry, among potters, in cement manufacture, and in mining.

Siclen,[7] gives the following estimates based on 1929 decennial federal census of the number of men exposed to dust hazard in various types of mining and industries marking in metals and minerals:

	Total Exposed
Metal mining	62,268

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6. Hoffman, F. L. Mortality from respiratory disease in dusty trades. Bull. U.S. Bureau of Labor Statistics, June, 1918.
7. Harrington and Davenport. U.S. Bureau of mines circular #6840.

	Total exposed
No metallic mining	23,665
Bituminous-coal mining underground	450,513
Bituminous mining-open pit	8,219
Anthracite mining underground	143,063
Anthracite mining-independent washeries	269
Swelting non-ferrous plants	13,666
Swelting ferrous plants	24,960
Cement plants	33,368
Abrasive industry	3,873
Asbestos products	8,092
Clay products	93,336
Cutlery	14,991
Glass manufacture	67,527
Granite, slate, marble & other stone products	28,715
Hones, whetstones, and similar products	174
Iron and steel	39,697
Mineral fertilizers	20,926
Minerals & earths, ground non-ferrous metal alloys	79,183
Pottery, including porcelainware	33,409
Sand, lime, brick	566

In Great Britain in 1930 about 35,000 workers including coal miners were employed in refractories, tin mining, pottery industry, metal grinding, sandstone working, sand blasting, and other dusty trades.

In 1931, 887,140 men were employed in coal mines of Great Britain.

In 1930, 287,546 men were employed in mines of France, 155,-397 in mines of Belgium and 400,654 in mines of Prussia. About 300,000 men were employed in the mines of South Africa. Probably 150,000 persons are employed in quarrying, cement, pottery, and stoneware industries of Germany. The above figures are not submitted as being accurate as to number exposed, but only as a relative measure. It is apparent that silicosis is a widespread industrial hazard so every effort should be directed toward eliminating this preventable scourge of workman.

III. Effects of breathing dust

An accurate estimate as to the number of men suffering from exposure to various dust is not available at this time. A consideration of organic dust shall not be made, because it is considered as relatively harmless. Silicosis the chief representative of the dust diseases will be primarily considered.

Silicosis is a fibrosis occurring in the lungs caused by free silica. It is one of the best known dust diseases of the lung.

At one time it was thought that silicosis was produced because of irritative factors of the silica particle. It is believed now the reason for the pathological process is based on colloidal chemistry.

The silica particle is inspired into the lungs where some is ejected back into the atmosphere and remaining portion picked up by the phagocytic cells. The silica particles on entering the phagocytic cell slowly dissolves and kills the cell. The dissolved silica and undissolved silica are set free then further dissolved by the alkaline body fluid. This dissolved silica is in a colloidal state and is toxic to the body cells which are killed. These dead cells surround the silica in solution. The dead cells and cell stroma form a fibrotic wall surrounding the central mass of dissolved silica. With the loss of water from the silica in solution, the activity subsides and a fibrous nodule is left. Thus silicosis is thought to be the result of a local physio-chemical action of hydrated silica on pulmonary tissue. Silicosis is progressive theoretically as long as undissolved silica remains in the tissue.

The sequence of pathological changes are thought to occur in the following manner which will subsequently be described. The size of the silica particle is limited from 0.5 micron to 5 micron and in some cases up to 10 micron in diameter. The dust is inspired and most of it is removed by the ciliated epithelium of the respiratory tract, and the secretions of the nose, pharynx, trachea, and bronchi retain a large part of it for subsequent expectoration and swollowing. Some of the dust cells eventually reach the alveoli and there institute the pathological process through irritation and catarrhal process, attended by proliferation of certain cells. Some of the proliferated cells become special phagocytis for the smallest dust particles ranging in size from 0.5-5 microns, while those over 10 microns are innocuous. These phagocytic cells or macrophages are expectorated and some enter the pulmonary lymphatic system. These enter at the proximal ends of the lymphatic channels beginning along the alveolar ducts. These may stop here and set up a local fibrosis or go on and stop at the lymphoid deposits at the bifurcation of the bronchioles, bronchi, and vessels with same result.

Some get by this obstacle and eventually reach the various pulmonary lymph nodes. The silica in the phagocyte is dissolved and due to the toxicity of dissolved silica the cell is killed. The dissolved silica and some that remains undissolved is set free in the pulmonary lymph channels and cause a local fibrosis to occur. The lymph flow is blocked by the dust ladened cells, cellular proliferation and fibrosis. Obstruction produced at these various lodging places tends to cause stasis and favors subsequent additional obstruction to lymph flow.

Fibrosis begins as practically microscopic nodular fibrosis along the course of the lymphatics from the primary lobule to the pulmonary lymph nodes. This will later appear as typical nodular type or stage of pneumoconiosis. This process continues until the small areas have enlarged sufficiently to cast a definite shadow. This gives rise to appearance of the first stage. The gradual blockage of lymph channels by enlarged nodules and surrounding reaction causes further clogging and accumulation of dust cells and deposition of silica in and around lymphatics due to leakage. Then a more diffuse fibrosis is started in the interstitial lung tissues. The interalveolar tissue and interlobular septa becomes thickened from clogged lymphatics and interstitial diffuse fibrosis occurs which appears in x-ray as a diffuse haze. In a rpaidly progressive case this may be the first noticeable change, instead of nodular fibrosis which may be practically absent or invisible.

As the normal hilumward lymph flow becomes blocked, there is instituted a backflow towards subpleural lymphatics and changes result in their distribution, resulting in more peripheral nodulation, thickened pleura, adhesions, and possible effusion.

In cases of weakly fibrosing dust with very low silica content, the lymph flour is maintained hilumward and the individuals may not progress beyond the first or slight second stage appearance in thirty to fifty years. In more rapidly progressing cases, the inhaling of high percentage silica dust some of the nodular fibrotic areas increase in size and tend to coalesce and in addition diffuse fibrosis becomes more prominent.

The fibrosis produced makes the lung less elastic, increases their bulk, makes aeration of blood less efficient, and decreases the circulation of blood through the lungs. As result increased respiratory effort is necessary. As fibrosis increases and elasticity of lung is lessened, the chest becomes more fixed and diaphragmatic breathing more prominent. Even diaphragmatic breathing may become lessened because of the development of basal pleurisy in some cases.

Silicosis has been conveniently divided into three stages, for convenience of description and possible compensation purposes. This has given use to some confusion so it has been suggested that a new classification be provided, but none has been satisfactory to the majority. First stage (corresponds to anteprimary stage of South Africa). The symptoms and signs of which are: Slight dyspnoea on exertion, some unproductive cough, often recurrent colds, and lessened chest expansion. The x-ray consisted of generalized aborization throughout both lung fields with more or less small discrete mottling. Second stage (corresponds to primary stage of South Africa). The symptoms and signs are: definite dyspnoea on exertion, pains in the chest, dry morning cough, recurrent colds, and chest expansion is decreased. X-ray shows a generalized medium sized mottling throughout both lung fields. Third stage (corresponds to the secondary state of South Africa).

Signs and symptoms are: Dyspnoea on slight exertion, cough with expectoration, capacity for mark diminished, lessened chest expansion, loss of weight, and heart may become dilated. X-ray shows the mottling more intense, the nodules are larger and take on a conglomerate form.

The diagnosis of this state should be based on a careful history particularly a detailed occupational history with investigation of plant, physical examination and x-ray.

Asbestosis is a member of the dust diseases and is characterized clinically by a milder course than silicosis. X-ray appearance is not as clear cut or distinctive as in silicosis and does not readily lend itself to classification. The roentgenogram gives the ground glass appearance and there is no nodulation with consequent tendency of nodules to coalesce and give dense opaque areas in films. Asbestos is hydrated magnesium silicate containing no free silica, but 44% combined silica, 43% magnesium and nearly 13% water with trace of iron and nickle.

Anthracosis and siderosis are mild in nature usually, and no more shall be said about these.

There exists a very high percent of tuberculosis among silicotics.

Tuberculosis rather frequently associated with silicosis will be considered.

At Japlin,[8] Missouri 720 miners were examined, 45.7% and silicosis and tuberculosis and 5.3% had tuberculosis.

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8. Harrington and Davenport. U.S. Bureau of Miners circular #6840.

Investigation of Mining conditions by Harrison and Lunza,[9] at Butte, Montana in 1921, revealed that 42.4% of 1,018 miners showed definite signs of lung damage.

In 1932, the metropolitan life insurance company in cooperation with the Pitcher Clinic at Oklahoma City, examined 27,553 individuals, of these 5,366 were found to have silicosis, 242 silicosis plus tuberculosis, and 320 uncomplicated tuberculosis.

In New York City,[10] 208 men were examined while working in a tunneling project. Of this number, 42% showed early and 15% well developed silicosis. There was found 9% of total number to have active and inactive cases of tuberculosis.

Granite workers showed wide variations in tuberculosis frequently. The excess in death rate being more marked among the men employed in the use of pneumatic tools.

In the state of Massachusetts, a decrease in pulmonary tuberculosis death rate of adult males from 288.5 per 100,000 exposed during 1895-99 to 203 during 1915-18. There was an increase in corresponding death rate of granite cutters of New England states from 432 per 100,000 during 1895-99 to 1056.7 during 1915-1918. These statistics, for the New England states are confirmed by similar data for every other stone cutting center of United States., proving with absolute certainty that in every section of the country the tuberculosis mortality of this group of industrial workers is increasing in contrast to a locally diminishing death

9. Harrington, D., and Lanza, A. J. U.S. Bur. of mines. Tech. proper 260, '21.
10. Fehnel, J.W. A study of silica dust in hard rock drilling in N. Y. C. J. indust. hyg., 1929, p. 69-81.

rate from tuberculosis.

It has been shown that pulmonary tuberculosis present death rate among granite workers is five times the normal experience in the population at large and probably six times what it should be on basis of strictly non-injurious occupation carried on largely under hygienic conditions and in open air.

The introduction of pneumatic and electric tools in processing stone has caused a tremendous increase of tuberculosis of the lung. In 1890 when stone was cut and polished with comparatively crude tools, the death rate from tuberculosis among these workers was 150 per 100,000. By 1910, it had increased to 1,080 per 100,000 and by 1925, had reached 1,950 an increase of thirteen times over the period when cruder methods in processing stone was used. The explanation of course, is that modern equipment creates many times the density of dust in air produced by manual labor. The cases of silicosis have also increased.

Other complications of silicosis are: Chronic bronchitis, emphysema, bronchioctasis, lung abscess, and gangrene of lungs.

IV. <u>Prevention</u>

As no cure is known for silicosis after it has developed, prevention is the only effective remedy.

There are two main lines of approach to the problem of dust disease prevention in industry--the engineering, through control of dust production and the medical, through selection of workers by examination; success probably depends upon the combination of the two.

Hatch,[11] considers the following measures essential to a complete well-balanced program of control.

1. Selection of workers by means of physical examinations in order especially to eliminate those with tuberculosis and other disorders of respiratory system. X-ray should be used as aid to physical examination.
2. Reduction of the dust concentration below the standard of permissible dustiness through an adequate means of dust control and good housekeeping.
3. Limiting the frequency and duration of employment in the dusty occupations, thus reducing the rate of deposition of dust in the lungs.
4. Routine measurement of the effectiveness of the dust control program of means of periodic medical examinations and dust survey.

The principle factors now thought to determine whether exposure to dust will produce pulmonary pathology are nature of dust, particle size, quantity of the dust dispersed in the atmosphere, and length of exposure.

Free silica or quarty has been considered the outstanding dust factor in industry with excessive mortality from dust diseases. Silica occurs quite widely in nature. Silica is found in burhostone, flint, agate, aventurine, amethyst, bloodstone, bra-

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11. Hatch, T. Dust Control. Present and future design considerations. Presented at Annual meeting of American Soc. Mech. Eng. New York City. Dec. 3-7, 1934.

zilian pebble, cornelian, cats eye, chalcedony, chert, cairngorm, chrysaprose, certine, flint, tairstone, jasper, jaspilite, lydion stone, milk quarty, tigers eye, sandstone, feldspar, mica, quartzite, ganister, granite, pegmatile, and ordinary sand.

Silica is used in a variety of ways in industry, varying in size from the massive form to particles--minute in size. It has been used in abrasives, sandpaper, sandblasting, metal buffing, burnishing, polishing, sawing and polishing granite, tube mill lining, lithographers, graining, wood polishing and finishing, refractory uses, metallurgical uses, flux in smelting, molds, chemical industries, manufacture of sodium salicilate, corborundum, paint, fertilizers, filler, road asphalt, pottery, building stone, monuments, glass, chemical apparatus, insulation, structural material, and manufacture of lenses.

The size of the dust particles is quite important. It is thought that the smaller the particle the greater the danger. Probably the most dangerous dust is that ranging from 2 microns to 0.5 microns or smaller because those unpalpable particles readily remain in suspension in the air for long periods of time. It is thought that there may be a lower limiting level as well as too higher particle size as to danger. Unless otherwise warned, employers and workmen are prone to regard impalpable non-visible dust as harmless. Dust particles under 5 microns in size readily pass through the mouth and nose, traverse the bronchial passages, and lodge in the alveolar tissues. The size of the dangerous dust (0.5-5 micron) is roughly the size of the tubercle bacillus.

The quantity of dust required to produce silicosis is unknown, for one person might resist a proportion of 100 particles per cubic centimeter, while another would react unfavorably to 50 particles; there is also the whole question of the rate of inhalation.

In a study by the public health services; the workers were divided into four groups, depending upon the average exposure in terms of amount of dust in the air, with the following results:-

In Group A, which included hand pneumatic tool operators and in which the exposure averaged about 59 million particles per cubic foot of air, it was found that practically 100% developed silicosis within ten years from the time of beginning employment. Also in this group a high rate of tuberculosis was discovered.

In Group B, other than hand pneumatic tool operators, the average dust concentration was nearly 45 million particles per cubic foot of air. This group showed the same reflection of a dust hazard as Group A.

In Group C, consisting of those groups exposed to average of about 20 million particles per cubic foot of air, silicosis developed much slower.

In Group D, the average exposure was less than 10 million particles per cubic foot of air. Although, certain amounts of silicosis was found in this group. There was no indication of serious results even when workers had been employed for years.

From this study it was found practicable to suggest a tentative standard for upper limits of allowable dustiness between 10-20 million particles per cubic foot of air for workers exposed to dust resulting from granite cutting. The same limits would presumably be applicable in the case of other dusts with the same physical characteristics, particularly with a quarty content of about 55%.

Silica particles have been found in the lungs ranging from 0.5 micron to 10 microns, but there is still the controversy as to what constitutes the upper border of safety.

The length of dust exposure to produce silicosis is a variable one.

In pottery industry, most cases developed after twenty years exposure.

In the sandstone--quarry workers, the time required to produce silicosis was on the average of fifteen years.

The granite industry seems to produce silicosis quite early for cases have been seen after two years exposure.

In the cement industry, the earliest cases of pneumoconiosis appeared after three years exposure.

The earliest case of silicosis reported was after eight months exposure to alkaline dust before appearance of respiratory symptoms. The process was probably due to the accelerated formation of silica hydrosal in the presence of an alkaline soap dust with high silica content.

Data collected by the United States Bureau of Mines at Pitcher Clinic in Oklahoma showed the following results:[12]

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12. Harrington, D. and Davenport, S. J. U. S. Bureau of Mines. Circular No. 6857.

	Average years
First stage silicosis	14.9
Second " "	14.3
Third " "	17.1
First stage silicosis plus tuberculosis	19.4
Second " " " "	18.0
Third " " " "	11.5

The following table is taken from the colliery Guardian for 1932.[13]

Industry	No. of deaths	Average age at death	Duration of employment years		
			Largest	Shortest	Average
Pottery Silicosis	72	55.6	57	10	39.8
" with T.B.	75	55.2	67	13	37.9
Sandstone Silicosis	21	57.3	57	20	40.4
" with T.B.	39	52.3	53	16	34.8
Grinding metals Silicosis	4	47.3	45	18	30.8
" with T.B.	26	51.1	48	2.8	30.2
Sand blasting Silicosis	7	40.7	16	4.5	10.3
" with T.B.	16	44.2	20	2.5	8.3
Scarring powder Manufacture Silicosis	3	37.0	11	5.25	8.8
Silicosis with T.B.	2	33.5	10.75	2	6.4
Miscellaneous Silicosis	7	55.3	45	2.8	20.4
" with T.B.	9	50.9	34	11	24

The diagnosis of all these cases was verified by post mortem examination.

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13. Colliery Guardian. Dust prevention in industry. Vol. 149, 1932. pages 90-95.

The development of silicosis depends upon a number of factors which are variable in themselves so definite conclusions can't be drawn.

The recognition of inhaled dust as the main factor in the production of pulmonary disease among miners drew attention to the quantitative aspects of the problem and the importance, as regards health, of determining the amount, weight, and particle size frequency of the dust in air breathed by workers in various industries. The chief methods used for sampling aerial dusts are: condensation, filtration, washing, sedimentation, impinging, electrostatic and resistance.

Dust control, as practised today, is to some extent the offspring of dust collection, but since they are opposite in purpose, the requirements of one are not necessarily like those of the other. The manufactures of dust collectors may guarantee collection of 99½% and in the production of commercial powder, this is entirely satisfactory, but it has no meaning in the performance of dust-control equipment. It is the escaping dust, not that collected, which is important, and quarantees of performance must be based upon permissible amounts of dust at the breathing zone of the workmen and in the clean air discharged from equipment.

Standardization of allowable dustiness in air is too young to be made into a law, because once a law they could only be changed with difficulty; but the use of standards by individuals, state authorities is a more flexible method of approach.

An outstanding remedy for the bad situation with respect to dust disease is education of public, employer, worker, doctor, and insurance carrier. An organized attempt should be made to show the workmen and employer the advantages of the safety measures advocated and if a condition of indifference exist remove the offenders in order to prevent a further economic loss. The physician should be educated as to means of diagnosis of the dust diseases, and the engineer to cooperate with the doctor for better understanding of individual problems.

In the mining industry dust control has been in force for some time. It was given its first real start in South Africa mines. The principle sources of dust in these mines was from jackhammer drilling, dry undercutting, and undercutting coal. Methods employed in South Africa are very good so some are just listed as follows:-

1. Wet drilling
2. Water
3. Spraying
4. Filtration
5. Dust sampling
6. Ventilation
7. Mask
8. Limitation of drilling
9. Dust traps
10. Dust filters

Elimination or at least reduction of dust may be accomplished in a variety of ways. Some of the few methods of control are:-

1. Use of water under pressure
2. Compressed air blowing
3. Vaccuum cleaning
4. Use of brushes and brooms
5. Suction systems
6. Good housekeeping
7. Elimination of bad practices
8. Maintenance of equipment in a dustlight condition
9. Isolation of dusty processes
10. Supplying protective devices to workers

The solution of the silicosis problem is important and necessitates the co-operation of the engineer and physician for a solution of the problem.

Probably the most important weapon in medical control of dust diseases is the physical examinations plus x-ray. Laboratory methods should be used to aid in making a diagnosis. The examinations should uncover defects and diseases that render employment hazardous to him who seeks it or to his fellow-workers. It should uncover imperfections that contribute to inefficiency and diseases and substandard health condition. A prospective employee need not be rejected outright and be deprived of the privilege of gainful occupation, as there are positions in virtually every plant of reasonable size that persons of varying substandard conditions of health and body can fill efficiently to the profit of their employers and themselves. Only the manifestly unfit and the victims of communicable diseases should be rejected.

A good program to follow in medical control of all phases of dust hazard is:-

1. Establishment of a medical department adequately equipped.
2. Routine examination of all applicants for employment.
3. Rating and placement of applicants.
4. Periodic physical examination with x-ray plus laboratory aids.

In order to carry out a constructive program of industrial hygiene, the requirements are:-

A. Physician thoroughly trained in public health procedure and having a comprehensive knowledge of the effects upon health of the various materials and processes used in industry.

B. An engineer who is also trained in industrial hygiene and who is familiar with industrial process.

C. Completely equipped laboratory for carrying on studies in industry.

The problem of caring for and treating silicotics necessitates consideration. The silicotics with active pulmonary tuberculosis should be isolated from their fellow workers, because of the increased susceptibility of existing silicosis to tuberculosis and momentary provisions made for them.

The worker who is old and has spent thirty years in acquiring a mild case of silicosis should be permitted to continue at his job if he is able or place him at some easier task.

If a young man who after three or four years, shows silicosis, he should be removed from the dusty atmosphere and given a task where the environment is relatively dustless.

In the case of the advanced silicotic, compensation should be given and amount determined by a committee of medical, legal, governmental worker and employer.

CONCLUSION

Dust is present universally and is a problem that must be solved. It cannot be satisfactorily handled by one group, but by co-ordinated efforts of several agencies.

In reviewing the literature one is struck by the great number of volumes written in the subject of dust diseases. The question that comes up next is why should all this work be published? Is it a pure scientific curiosity that prompts the many workers to work in this field or is it some commercial reason? The latter is probably true for after industry began to pay dearly for compensations, a great cry went up as how to prevent this disease so there is probably the answer to the volumes written on the subject.

It is known that the sources of dust are many, and methods of control are several. With this knowledge of both, the industry has a method by which dust diseases may be controlled to a minimum. It requires intensive work and co-operation of all agencies concerned. This may be carried to the point where in the future silicosis will be unknown for it is preventable and can be wiped out.

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