FILE NAME: Insurance Industry (INS)

DATE: 1940

DOC#: INS013

DOCUMENT DESCRIPTION: Published Conference Presentations - Underwriting Aspects of the Industrial Dust Hazard [Dr. Lanza] & Following Discussion

PROCEEDINGS

OF THE

THIRTIETH ANNUAL MEETING

OF THE

MEDICAL SECTION

OF THE

AMERICAN LIFE CONVENTION

1940 OFFICERS
MEDICAL SECTION

Chairman—Dr. A. J. Robinson, Medical Director, Connecticut General Life Insurance Company, Hartford, Connecticut.

Vice-Chairman—Dr. W. F. Blackford, Medical Director, Commonwealth Life Insurance Company, Louisville, Kentucky.

Secretary—Dr. B. F. Byrd, Medical Director, National Life & Accident Insurance Company, Nashville, Tennessee.

Member of Board of Managers—Dr. T. H. Dickson, Medical Director, Minnesota Mutual Life Insurance Company, St. Paul, Minnesota.

Program Chairman—Dr. R. M. Livingston, Medical Director, Mutual Life Assurance Co. of Canada, Waterloo, Ontario.

Underwriting Aspects of the Industrial Dust Hazard

A. J. LANZA, M. D., *Assistant Medical Director*
and
R. J. VANE, *Statistical Bureau*

Metropolitan Life Insurance Company
New York, N. Y.

OUR knowledge of health hazards resulting from exposure to various kinds of dust in industrial occupations is based on observations and clinical studies of workmen exposed to these dusts, and upon noting the results of the action of these same dusts upon experimental animals. The former method goes back for centuries and the earliest conceptions of the harmful properties of dust were based on first hand observation.

Many of these conceptions were erroneous. It is only in the past twenty years that anything like a comprehensive knowledge of dust diseases has been attained — due first to the perfection of the X-ray as a means of diagnosis, and the development of successful laboratory technique for conducting tests on experimental animals.

Now we have arrived at a point where intensive studies, both clinical and experimental, conducted in this country and in other countries, have resulted in a crystalization of our ideas. In the English speaking world, the outstanding contributions to our knowledge have come from the South African Institute for Medical Research and the Miner's Phthisis Prevention Committee in Johannesburg; from various official agencies in England and Australia, in Ontario, Canada, and in this country, especially the United States Public Health Service, the United States Bureau of Mines, and the Saranac Laboratory.

In presenting this discussion of industrial dusts, their effects on workmen exposed to them, and the bearing that this subject has on

00154

life insurance underwriting, Mr. Vane and the writer have attempted to summarize what should be the insurance attitude, based on the present state of our knowledge. Briefly, the present concept regarding industrial dust hazards is as follows. Organic dusts, of which the dust arising in textile operations is an example, do not cause any specific pulmonary disease. There is no clinical or experimental evidence to the contrary. Coal, which is an organic substance, is considered in connection with mineral dusts. Organic dusts at times may cause an allergic reaction and also irritation of the upper air passages. They do not cause protracted disability or death. In this classification may be included cotton, wool, silk, sugar, flour, starch, wood, tobacco, and leather.

It is true that some occupations in which is involved exposure to organic dusts manifest a tuberculosis mortality rate higher than the average. Formerly, it was thought that this undue prevalence of tuberculosis was due to the occupational dusts but the dust studies of recent years have demonstrated that, mostly, organic particles are too large to penetrate to the pulmonary tissue and when they do, they are absorbed without producing any specific inflammatory reaction. Nor is much credence given nowadays to the idea that organic dust particles convey tubercle bacilli to the lungs and thus cause tuberculosis.

METALLIC DUSTS

Here the situation is not unlike that of organic dusts. The prevalence of pulmonary disease among workers in certain occupations exposed to metallic dusts gave rise to the theory that metallic dust was a fruitful source of tuberculosis. Such diseases as "grinder's rot" were recognized clinically long before the era of modern diagnosis and laboratory research. Metallic particles in the form of fumes may cause such reactions as zinc chills or brass founder's ague, which are more in the nature of an allergic reaction. The pulmonary disease which afflicts grinders and buffers is silicosis and not in any way a result of exposure to metallic dust. Metallic dust particles are mostly large and heavy (that is, comparatively speaking), consequently they do not penetrate to the lung, in many instances, and when they do they are either absorbed or remain fixed and inert in the tissues. From an underwriting standpoint then, the hazard of metal dust is the hazard of such silica dust as may be associated with it.

COAL

For many years there has been a belief that coal miners had less tuberculosis than the average for wage earners and, on the other hand,

miner's asthma has been recognized as an occupational disease of coal miners. Here again, recent studies have clarified the situation to a considerable extent.

Bituminous or soft coal miners do not have an occupational pneumoconiosis of a disabling quality. Their lungs become pigmented from coal dust, but they do not develop a true fibrosis. For anthracite miners, the picture is quite different and the reason is that anthracite mining includes exposure not only to coal dust but also to silica. Disabling miner's asthma was shown by the United States Public Health Service study of 1935 to be a type of silicosis, to which the term anthraco-silicosis is properly applied. Anthracite seams are often found in country rock which contains a considerable amount of free silica and, as in other silica dust exposures, the development of pulmonary fibrosis was found to be proportionate to the amount of silica in the coal dust and the length of exposure. The incidence of tuberculosis among anthracite miners is very high. Clinical pulmonary tuberculosis was found in 15 per cent of those with early anthraco-silicosis and in 43 per cent of those with advanced anthraco-silicosis. Service upwards of twenty to twenty-five years was associated with a tuberculosis rate of 14 per cent among regular miners and 37 per cent among those with a number of years as rock workers. Animal experimentation, with both bituminous and anthracite coal dust, free from associated silica, produced no specific reaction.

NON-SILICEOUS MINERALS

There is no evidence that non-siliceous minerals produce any pulmonary disease. Gardner, at the Saranac Laboratory, tested upon animals such substances as diamond, aluminum oxide, rutile, marble, gypsum, galena, chalcopyrite, iron sulphide, zinc sulphate, fluorite, and dolomite. The sulphides and dolomite proved toxic but this effect is outside of our present discussion. Non-siliceous minerals proved inactive in animals and there is no clinical evidence of specific disease among workers exposed to this type of dust.

ARTIFICIAL ABRASIVES

Experiments with artificial abrasives demonstrated the incorrectness of earlier ideas that dust caused injury to the lungs proportionate to the hardness and sharpness of the particles. I can remember a time when it was generally believed that the injurious qualities of a dust depended upon the hardness of the dust and the sharpness of the particles. It was not until we had pursued our studies in dust counts and dust experimentation that we realized that when you were talking of

dust particles of one to two and three microns in their largest diameter, that those particles had no physical characteristics in the ordinary sense in which we apply them. Artificial abrasives are among the hardest substances known. Neither silicon carbide nor aluminum oxide produced any reaction resembling silicosis in experimental animals nor do workmen exposed to these dusts show any clinical symptoms or X-ray appearances suggestive of silicosis.

SILICATES

A considerable number of silicate dusts have been studied and their effects upon experimental animals, both by inhalation and injection, noted. They produce little or no reaction in the body tissues and what little reaction some of them do produce does not tend to progress An exception should be noted here regarding asbestos, which is considered separately.

Nor is occupational exposure to silicate dusts accompanied by any such clinical picture of disability and death as is found in silica dust exposures. The Public Health Service studied some talc mines in 1935. Only 66 men were exposed. Tuberculosis morbidity and mortality rates were very high in the county where the mines were located and the dust exposure was exceedingly severe, running into hundreds of millions of particles. Eight men showed advanced pneumoconiosis but tuberculosis was also present in most of them. It is conceivable that exposure to tremendous quantities of any mineral dust, regardless of silica-content, might so overwhelm the lungs that they could not deal with the invading substance. Irritation, inflammation, and probably infection would follow with disability in proportion Such isolated instances do not invalidate the conclusions stated in the previous paragraph.

ASBESTOS

Asbestos is the only dust of combined silica which is known to cause a definite pulmonary fibrosis which may result in disability and death Let me make that point clear. When we talk of silicosis and the types of dust that produce silicosis, we mean dusts that contain silica in a free or uncombined state, SiO_2 When the silica is in the form of silicon carbide or magnesium silicate or iron silicate that is what we mean by combined silica. The combined silicate dusts, as I have just mentioned, do not produce any pulmonary disease, with one exception, and that one exception is asbestos, which is a hydrated magnesium silicate mixture, with a little iron in it Most of the asbestos fabricated in the United States comes from the mines in

Quebec. Asbestosis, however, is not found among asbestos miners, most of whom work in open quarries or pits, nor among men engaged in milling operations whereby the asbestos is prepared for shipment and bagged, but among workers in fabricating plants. Fabrication, you will understand, being in the nature of a textile process. Only recently has an explanation been offered for this anomaly. It would appear that not only is asbestos an exception in that it difiers from other silicate dusts in its ability to cause structural changes i.: the lungs but its action appears to be mechanical and not chemical like the action of silica. If asbestos is crushed to fine particles, it will not cause asbestosis in experimental animals nor will asbesto produce any reaction elsewhere than in the lungs. If its action were in the nature of silica, the effect would be more pronounced with smaller particle sizes. In other words, Gardner established that the intensity of a silica reaction upon animals depended upon the smallness of the silica particles, because, to sum it up briefly, the finer the silica particles, the greater is the surface exposure which acts upon the pulmonary tissue.

Asbestos particles, below a certain size, do not produce any effect in experimental animals Furthermore, you can produce a silicotic reaction, the typical silicotic nodule, in any organ by injecting prepared silica. You can get silicosis of the liver or of the spleen or elsewhere. But you cannot produce any reaction in animals with asbestos anywhere except in the lungs, and then only if your particles are sufficiently large. Consequently, Gardner concludes that the relatively long spicules of asbestos produce their effect because the lungs are never at rest and the constant movement provokes the fibrous reaction.

Like silicosis, asbestosis develops slowly, presenting the characteristic symptom of shortness of breath. The number of cases of asbestosis that have come to autopsy is small but there are several cases on record in which death was due entirely to the asbestos and not to any associated infection or other condition There does not seem to be any pronounced tendency to tuberculosis in asbestosis, but most of the fatal cases have been complicated by other disease such as cancer and diabetes so that the extent to which the asbestosis was responsible for death is not clear.

I made a rather exhaustive study a few years ago of all the insurance certificates that I could find in which asbestos was given as the cause of death and then followed up the previous history of these people and got their medical certificates when they were on disability. The diagnosis given on the disability certificates was usually so at variance with the diagnosis given on the death certificates, often by

the same physician, that it was impossible to come to any definite conclusion and say, "this man died of asbestosis," when his previous disability certificates showed that he had myocarditis or high blood pressure or cancer or diabetes or pulmonary tuberculosis.

The pathology and X-ray appearance of asbestosis are entirely different from silicosis and in the absence of a definite history of exposure over a considerable period of time to asbestos dust, the diagnosis is difficult. A great deal of success has been attained in controlling dusts in the plants where asbestos is fabricated so that it is not likely that asbestosis will ever become an important industrial disease, as far as the number of cases is concerned.

The scarcity of clinical data on asbestosis makes the prognosis doubtful. We have had a small group of cases under observation for about ten years — there were originally, I think, some sixty odd men in this group and we still have fifty of them where we can get at them — and some of these show a definite but slow progression as far as can be judged from the X-ray films. However, what the outcome may be remains to be seen. These men are all working and show no signs of any disability.

In the meantime the plants have been cleaned up so that these men are not exposed now to inordinate quantities of asbestos dust, so it is difficult to say what would have been the progress of their disease had their working conditions remained the same.

SILICOSIS

It may be said that there is a very general accord among the leading investigators and research authorities in the opinions and conclusions presented at the international conferences on silicosis. (Johannesburg, August, 1930. Geneva, August and September, 1938.) Such differences of opinion as may be expressed from time to time do not seriously affect the general agreement on the basic factors affecting the cause, incidence, effects, and prognosis of silicosis.

The effect of silica upon the lungs depends on the nature and extent of exposure. That is to say, upon the quantity of dust in the air breathed by workmen, the amount of free or uncombined silica in the dust, the size of the dust particles, the presence of other substances in the dust which may modify the action of silica, and the length of time or duration of exposure. The individual himself must also be considered. Previously existing disease of the lungs, especially a tubercle infection, will influence his reaction to inhaled silica. There may be other phases of susceptibility about which we know little or

nothing but in the main, it may be stated that individual susceptibility to the action of silica dust is an acquired rather than a congenital condition.

In industry we find different types of silica hazards. The severity of a silica hazard is estimated on the amount of dust in the air and this is expressed in millions of particles per cubic foot. The particles counted are those under ten microns in size, though the present opinion is that the particles between one-half a micron and three microns are mostly responsible for causing the disease.

Exposure to excessive quantities of pure silica dust, that is, where the dust count is in the hundred of millions of particles per cubic foot, is now rare. Isolated instances may be found as in the case of a sandblaster improperly protected. Such exposure produces a severe type of silicosis in upwards of five years, even sooner in extreme cases. With such extensive silica damage to the lungs, tubercle infection is almost inevitable, though cases will be seen occasionally that proceed to a fatal termination without infection.

Exposure to amounts of silica dust where the particle counts are in excess of ten million does not occur frequently at the present time, though it is only in the last few years that silica industries have endeavored to cope with their dust problem and effectively reduce the amount. Such exposures produce definite disease in from eight or ten to twenty or more years, in proportion to the actual quantity of dust. The incidence of tuberculosis is high and of those who die of their pulmonary condition, 75 per cent are tuberculous. This does not mean that 75 per cent of silicotics have tuberculosis, but of those whose damage to their lungs is sufficient to cause their death, that is, where death is due primarily to silicosis, 75 per cent of them will have tuberculosis also.

It is also possible to find degrees of silica exposure which, while producing definite silicosis evident on X-rays, will not produce disability or death in an average working lifetime. Mortality statistics are not broken down by industry to an extent sufficient to make available definite figures by degree of exposure. We must rely then on the clinical results obtained in industrial investigations which are conclusive enough for those industries studied. Our own impression is that even where the silica hazard is of a mild type, the incidence of tuberculosis will be higher than the average. It has been stated that where silica dust contains caustic alkalies also, the action of the silica is intensified very considerably. The evidence here is meager. Other

substances apparently have some inhibiting effect upon the action of silica, affecting the pathological as well as the clinical picture.

PROGNOSIS

The prognosis is fair for a silicotic whose lungs are not extensively damaged and who is free of infection. Such persons have an increased susceptibility to infection and if brought into intimate contact with a case of tuberculosis are very apt to become tuberculous also. The outlook for the tuberculo-silicotic is poor. Many cases are essentially chronic and may live for years. Others show rapid progression and die in from one to two years after their infection becomes active. The importance of tuberculous infection among persons exposed to silica dust brings up a very important underwriting consideration, that is, where the worker in a silica occupation is in contact in his home with an open case of tuberculosis. Insurance in such cases should be declined.

The question is frequently asked whether men who had worked in silica dust at one time but later changed to a job in which there was no exposure to silica might be considered standard risks for life insurance if they passed the usual insurance physical examination. While there are no insurance statistics to prove the point one way or the other, there is every reason to believe that a group of such men will not give a standard mortality. The usual type of physical examination will not detect silicosis. The only way to be sure is to secure X-rays of the lungs of such applicants and have them interpreted by some one who has specialized in reading films of this kind.

Silicosis is gradually coming under control. The aggravated cases seen twenty years ago are rarely seen now and the clinical picture has changed with the progress of dust control measures. The trend is towards a lessened incidence of new cases of silicosis. This improvement depends upon the maintenance of constant vigilance in detecting unsuspected hazards and in supervising the efficiency of dust control methods after they have been put into effect. It cannot be expected that this improvement will be reflected in insurance mortality until a considerable period of time has elapsed.

The conclusion seems warranted from clinical and laboratory experience, then, that the only dusts which of themselves might have sufficient effect on the mortality of workers exposed to them, so great as to require an extra premium charge for life insurance, are silica and possibly, asbestos.

Life insurance men naturally will turn to mortality statistics for confirmation of a finding so at variance with the common belief, long

held, that exposure to all kinds of dusts is productive of a high incidence of respiratory tuberculosis. To the layman it seems only reasonable to suppose that the difficulty in breathing he experiences when exposed to large quantities of dust must in some way affect his health, and that filling the lungs with foreign particles must have injurious effects on them. The writings of eminent students of occupational mortality statistics have served to strengthen this belief. Nearly all such statistics show the mortality from respiratory tuberculosis among workers exposed to a wide variety of dusts to be well above the average for all workers included in the study. But, when these figures are refined so far as is practicable to make allowance for factors other than dust, which may influence the mortality from tuberculosis of men employed in these occupations, the importance of many dusts as causative factors becomes less evident.

There is a surprisingly large number of factors, other than dust, any one of which may be responsible for the high mortality from tuberculosis among men in dusty trades. Some dusty occupations are carried on by men recruited from the lower income classes, among whom it is now known that apart entirely from exposure to a specific occupational hazard, the death rate from tuberculosis is considerably higher than among persons in the higher income groups. The selection of certain kinds of work by the less robust workers is reflected in the high mortality from tuberculosis of men in those occupations. Racial and nationality groups tend to enter certain occupations, with the result that the peculiar susceptibility or relative immunity to tuberculosis of these stocks, colors the mortality picture of men in such occupations. These are but a few of the many circumstances for which allowance must be made before conclusions can be drawn as to whether a particular dust is or is not the responsible agent in the high mortality found among men in a dusty trade.

Unfortunately there is, for the United States, no considerable body of mortality statistics sufficiently detailed to permit of evaluating separately some of even the most important conditions affecting the mortality of men in specific occupations. Perhaps the best statistical demonstration of the effect of different types of dusts on mortality from tuberculosis is supplied by two British authorities, Dr Edgar L. Collis and G. Udney Yule. These authors selected for study, from the occupations reported upon in the Registrar General of England and Wales' report on Occupational Mortality covering the years 1921-1923, two groups of occupations — one exposed to silica dust, and the other to non-silica inorganic dust. The groups were so chosen that each had, as far as possible, the same amount of dust exposure, physical effort,

exposure to heat or to weather, or any underground environment. Part of the authors' comment on the results as regards respiratory tuberculosis is quoted:

> "For all ages (20 to 65) the Comparative Mortality figure (this figure is defined as the number of deaths that would have occurred in the Standard Population at the rates ruling in the occupation) of the Silica Group is no less than 592.2 against 163.5 for the Standard Population, i. e., the mortality is more than three and a half times the normal. For the Non-Silica Group, the Comparative Mortality Figure, although actually slightly higher than normal, at first sight hardly seems to differ significantly from the normal; nor would one's judgment be much affected if, instead of making comparison with the Standard Population, one had used the Social Groups III and IV, for which the respective Comparative Mortality figures are 159.8 and 164.2. But the summary figures conceal interesting changes with age. A glance at the figures (shown on Table 4 of their study), shows that in the Silica Group the ratio of mortality to the normal, though greater than unity even in the lowest age-group, rapidly increases as age advances. In the Non-Silica Group, the comparative mortality is actually below normal up to age 35; it rises just above normal in the following age-group; but at ages over 45, it is conspicuously above normal."

The facts regarding tuberculosis were not published in this report for each of the six silica occupational classes included in the study. We have, however, determined by reference to the Report of the Registrar General, that each of the silica occupations had over twice the expected number of deaths. The extraordinarily high ratio of 1,150 per cent was recorded for tin and copper miners, while sandstone masons, cutters, and dressers had a ratio of 441 per cent. On the other hand, only the limestone masons, cutters, and dressers of the five occupations considered in the non-silica group exhibited a high ratio, 191 per cent.

Like the English tin and copper miners, American metal miners are exposed to a serious silica hazard and have an inordinate mortality from respiratory tuberculosis as will be seen by reference to the following table. In this table are given the ratios of actual to expected deaths from tuberculosis for a number of occupations exposed to dust and reported upon in the Joint Occupation Study, 1928, and in the Occupational Study 1937, both published by the Actuarial Society of America and the Association of Life Insurance Medical Directors. In the period 1915-1926, the number of deaths from respiratory tuberculosis among

metal miners was over eight times the number expected on the basis of death rates by ages prevailing among Standard Ordinary lives in the same years. In the later study covering the years 1925 to 1935, the ratios were even higher — over 11 times as many deaths were recorded for metal miners as were expected on the basis of death rates among Standard lives in these years.

NUMBER OF DEATHS AND RATIO PER CENT OF ACTUAL TO EXPECTED DEATHS FROM TUBERCULOSIS OF THE RESPIRATORY SYSTEM

Ordinary Department Experience of American Life Insurance Companies.

Compiled from: Joint Occupation Study, 1928, and Occupational Study, 1937, both published by the Acturial Society of America and the Association of Life Insurance Medical Directors.

WHITE LIVES

Occupation	1915-1926 Actual Deaths	1915-1926 Ratio Actual To Expected	1925-1935 Actual Deaths†	1925-1935 Ratio Actual To Expected
Mine Operatives—Underground				
Metal Mines	48	815	52	1113
Copper mines	24	913	29	1381
Gold and Silver mines	9	804	11	940
Iron mines	4	200	12	857
Lead and Zinc mines	11	1833	‡	. . .
Coal Mines				
All mines	125	157	135	288
Pennsylvania (mostly anthracite)	*	.	115	334
Other localities (bituminous)	*	.	20	161
Stone Cutters				
Granite and Sandstone	16	976	38	2639
Marble and Limestone	3	294	10	1053
Other and not specified stones	*		16	1560
Chippers of Metal (except shipbuilding)	8	615	16	1667
Buffers and Polishers of Metals	10	147	*	
Grinders of Metals†	7	710	5	154
Upholsterers	16	225	*	
Cotton Mill Operatives (males)† (Excluding carders and combers)	*	..	14	243
Woolen Mill Operatives (males)† (Excluding carders and combers)	*		10	200**

*Data not available.

†Substandard business only studied, except for grinders of metals, cotton mill and woolen mill operatives for which occupations both Standard and Substandard business are included

‡Three deaths from tuberculosis in approximately 1,200 life years of exposure.

**Ratio reported in text. Expected deaths for this occupation calculated from basic table for males only, for other occupations from basic tables for males and females combined.

While the actual number of deaths for each separate metal mining class was rather small in both investigations, and consequently the ratios are subject to a large margin of error, nevertheless it is interesting to observe that they were quite uniformly high for copper miners, gold and silver miners, and lead and zinc miners.

The situation as regards iron miners is interesting because it was thought for a long time that they had little or no exposure to silica. More recent studies show that there is actually a moderate degree of silica hazard which produces silicosis in upwards of twenty years. However, in both insurance studies there were too few deaths from respiratory tuberculosis to permit definite quantitative conclusions therefrom. In the later study there were 12 deaths from tuberculosis which was over eight and one-half times the expected.

A great many workers in mining occupations, it must be borne in mind, are common laborers among whom we should expect a much higher mortality from tuberculosis than among Standard Ordinary policyholders. From the limited data available for unskilled workers, from these studies it would appear that the tuberculosis death rate for this class of workers runs between two and three times the rate for Standard Ordinary business. Thus, it is clear that even when allowance is made for the important factor of social-economic class, the death rates for the metal miners are extremely high.

Coal miners in different sections of the country present an interesting contrast. Among the bituminous miners, exclusive of the Pennsylvania miners, the mortality from tuberculosis was over one and one-half times that of Standard Ordinary lives, a low ratio for this type of worker. Pennsylvania miners, on the other hand, most of whom were employed in the anthracite field, had a very high ratio of actual to expected deaths — well over 300 per cent. Anthracite miners, as we have stated, are exposed to an appreciable amount of silica dust, which accounts for their relatively unfavorable position.

No dusty trade has received more careful study in the United States than that of granite cutting. Long recognized as a dangerous trade, it has been found time and again to take a heavy toll in deaths from tuberculosis. The amount of free silica in the dust breathed by these workers is very large. In the years 1925 to 1935, the mortality from tuberculosis among granite and sandstone cutters was the highest recorded for any of the groups of insured lives studied. There were more than twenty-five times as many deaths from tuberculosis as expected, whereas in the earlier years there were nearly ten times as many deaths as expected, based on a small exposure. Marble and limestone cutters did not show anything like as high a ratio as the granite

and sandstone cutters. There were ten deaths where one was expected in the years 1925 to 1935.

It can be but regretted that there are so few mortality statistics for occupations exposed to dusts other than silica. Grinders of metals, most of whom for many years have been using composition wheels in place of the old sandstone wheels, and consequently have had little or no exposure to free silica, experienced a relatively low extra mortality from tuberculosis in both studies, based, however, on a small exposure. An interesting point to be noted here is that this occupation also offers exposure to metallic dust. It would appear that neither the dust from the composition wheels nor the metal dust has produced excessively high death rates from tuberculosis. Buffers and polishers, a somewhat similar group, also appear to have a low extra mortality from tuberculosis in the years 1915 to 1926.

Cement, lime, and artificial stone workers were reported upon in both the Joint Occupation Study and the Occupational Study, but the number of deaths from tuberculosis was in each instance less than the number required for separate tabulation. This fact in itself is significant, although it does not mean necessarily that the death rate from tuberculosis was below the average. Nevertheless, from the facts presented in these studies, it is quite evident that the tuberculosis death rate for this occupational class was comparatively low

Three groups of male workers exposed to organic dusts are included in the table — upholsterers, cotton mill operatives, and woolen mill operatives. The ratios for all of these classes run about 200 per cent, based on small exposures.

It must be admitted that these occupational mortality statistics are meagre and do not permit of very definite conclusions regarding the effects of specific dusts. So far as they go, they fully substantiate clinical and laboratory findings regarding the injurious character of silica dust. The death rates from respiratory tuberculosis for men in occupations where there is a serious exposure to silica dust are so high as to leave no room for doubt that silica is the offending agency.

Contrary to clinical and laboratory findings, however, the mortality statistics, presented by Collis and Yule, suggest that long continued exposure to non-siliceous inorganic dust has an injurious effect on the lungs as is indicated by the higher mortality from tuberculosis among exposed workers after age 45. About all that can be said on this point from the limited American life insurance company experience, is that the tuberculosis death rates for grinders, metal polishers, and buffers who are exposed to inorganic dusts, most of which do not

contain a large percentage of silica, are not high, having in mind the economic status of these workers as compared with the average ordinary policyholder. Lime, cement, and artificial stone workers, had a quite favorable mortality. The very high ratio for the small group of marble and limestone cutters, however, may be suggestive of some untoward influence. Dr. L. U. Gardner at Saranac Lake, found that animals dusted with marble dust by inhalation, developed little reaction, but became temporarily more susceptible to infection with tuberculosis. The increased susceptibility was not marked. I understand that nobody else has been able to duplicate that experiment; that is, that dusting animals with marble dust increases their susceptibility to tuberculosis for a short time.

The three occupations in which there is exposure to organic dust had from two to two and one-half times the number of tuberculosis deaths expected. Even allowing for the economic position of these groups as compared with Ordinary policyholders generally, the mortality is high. It is difficult to see in the light of present clinical knowledge, how it is possible to account for the excess mortality on the score of damage to the lungs caused by such dust.

Mortality findings, then, are still not in complete harmony with clinical and laboratory findings. But from a practical underwriting standpoint, it may be said that the only dust which of itself might so affect the total mortality by increasing the incidence of tuberculosis as to require an extra premium charge for life insurance, is free silica. We have no mortality data for asbestos workers but, it may well be that exposure to asbestos dust in large quantities will also be found to produce a high mortality.

Chairman Bender: Discussion of this most excellent paper will be opened by Mr. Morris Pitler, Statistician, Research Section, of the Mutual Life Insurance Company of New York. Mr. Pitler has contributed a respectable number of papers to the Home Office Life Underwriters Association, dealing particularly with the underwriting of hazardous industries and occupations. He has served as Chairman of the Occupational Committee of this organization and at present is Editor of the Suggested Reading List for Underwriters. Mr. Pitler.

Mr. Pitler: Before proceeding to a discussion of Dr. Lanza's paper, I would like to take this opportunity to express my great pleasure at being here with you, among the beautiful surroundings of this beautiful hotel. I wish to personally thank Dr. Dickson, your Program Chairman, for making my visit here possible by his invitation to take part in your proceedings.

Discussion—Underwriting Aspects of the Industrial Dust Hazard

MORRIS PITLER, *Statistician, Research Section*

Mutual Life Insurance Company of New York
New York, N. Y.

THE discussion to which we have just listened — and I might say, Dr. Lanza, listened with great pleasure — leads up to a somewhat startling conclusion.

We in the life insurance business have for many years shared the general concept that exposure to excessive concentrations of dust, any dust, must affect adversely the health of those exposed to such concentrations for any appreciable length of time. We believed, too, that dusts varied in danger according to their character or chemical composition. And in the statistics of our mortality studies we seemed to find ample proof for these beliefs. The so-called "dusty trades" almost always showed higher than average mortality ratios, with excess mortality from tuberculosis of the lungs; and the differences in amount of excess mortality indicated that some dusts were more harmful than others.

True that for a number of years now we have repeatedly heard that there is little or no clinical or experimental evidence of lung damage from exposure to industrial dusts other than silicia and, possibly, asbestos and such information has come from a number of sources. The thought, therefore, is not entirely new to us. Nevertheless, it does come as a surprise to be confronted with the definite statement by two such well-known and reputable investigators as the authors of this paper that for life insurance purposes silica dust alone, and perhaps asbestos, can sufficiently affect the mortality of workers

exposed thereto as to require premiums in excess of standard for life insurance.

With respect to the hazard of silica dust exposure this conclusion involves no change from our previous thought. Industries and occupations with such exposure have exhibited very high mortality ratios, caused largely by extremely high death rates from pulmonary tuberculosis. The authors have assembled such mortality data in their paper and I shall attempt no repetition of them.

As for other dusts, again excepting asbestos, the suggestion is made that our reasoning may have been wrong. "Organic dusts," we are told, "do not cause any specific pulmonary disease" and "there is no clinical or experimental evidence to the contrary." Metallic dust, too, is not harmful to the lungs and such damage as has been observed among metal grinders and buffers is caused by the silica dust from the abrasive wheels rather than from the metal dust itself. And so with coal dust per se, and the dust of non-siliceous minerals and abrasives in general, and also silicate dusts, always with the possible exception of asbestos.

But what of the greatly excessive mortality ratios from tuberculosis of the lungs in many of the industries and occupations with exposure to such dusts, ratios ranging as high as 200 per cent to 300 per cent of the expected or normal mortality? In the table compiled by the authors from the Joint Occupation Study of 1928 and the Occupational Study of 1937, there are listed bituminous coal miners with a ratio of 161 per cent of the expected, upholsterers with 225 per cent, cotton mill operatives with 243 per cent and woolen mill operatives with 200 per cent. The authors have apparently anticipated this question by pointing out that many factors other than dust may be responsible for a high tuberculosis mortality, factors such as grade of worker, or selection of certain classes of work by less robust individuals, or race and the tendency of some races or nationalities to drift into certain industries. The further question might be asked, "Can such high mortality rates from tuberculosis, two and three times the average, be entirely accounted for by these considerations, thus eliminating dust as a causative factor? The authors point out that available data for unskilled workers do reveal a tuberculosis death rate of this magnitude, even without the presence of dust exposure. From my own perusal of the joint insurance company studies, previously mentioned, I have selected as another example the experience on male waiters in the Occupational Study of 1937, among whom deaths from tuberculosis of the lungs were three times the normal. Here, perhaps, is an instance of a group wherein factors of initial

selection, race and low economic status, with all that the last-named implies in standards of living, play their parts in the causation of a very high mortality from pulmonary tuberculosis.

I must admit that Dr. Lanza and Mr. Vane have presented a very logical argument in explaining away non-siliceous dusts as the cause of the high incidence of tuberculosis in certain dusty trades and thus bringing the insurance mortality data into line with clinical and experimental findings.

If we accept their conclusion that only silica and asbestos offer real hazards and that other industrial dusts may be ignored for purposes of life insurance rating, what are some of the further practical implications for us? This question is, perhaps, beyond the scope of the original paper, but it may be well to touch upon it briefly.

We cannot, of course, get away from the fact that the excess mortality in the dust-exposed occupations under discussion is still there, whether caused by dust or by other unfavorable factors, and such excess mortality must be covered by our premiums.

Most of you are undoubtedly familiar with a certain Standard Classification of Occupations and Industries published by the Home Office Life Underwriters Association several years ago, drawn up and recommended by its Occupation Committee for life insurance use. This classification, which has been widely adopted by life insurance companies, with more or less modification, lists a number of dusty industries, some with silica dust-exposure and others with exposure to organic dust or to non-siliceous mineral dusts. And within these industries there frequently appears a separation of dust exposed occupations, with the implication that such occupations should be more severely treated than other occupations of similar grade and skill within the same industry, but not so exposed to dust. If the presence of organic dust and non-siliceous mineral dust is to be disregarded as a causative agent in higher mortality, then such distinction between workers is no longer justified in many industries, *unless* reasons other than dust exposure exist. It is quite likely that because of the disagreeable personal reaction to work in a very dusty atmosphere, or the unskilled nature of some such work and its low remuneration, such employment is acceptable only to a lower grade of individual and that, therefore, such a group of workers will yield a higher mortality; not because of dust exposure but for other reasons. This situation may justify the separation of the dust-exposed occupations and a higher insurance rating therefor. But dust is no longer the basic reason for separation, except when the dust is silica or asbestos.

I was particularly interested by the statement that exposure to greatly excessive concentrations of silica dust is now rare and that even moderate exposure does not occur frequently at the present time, because during the last few years silica industries have recognized the hazard and are endeavoring to minimize or eliminate it by controlling the amount of silica dust in the working atmosphere. And in time we can hope that our mortality experience will reflect the efficacy of these measures for controlling the hazard.

Our business is fundamentally conservative, and rightly so. But as soon as experience shows that underwriting practices are too severe or premiums too high, I think it can justly be said that changes are readily made. However, when we are faced, not with an accomplished fact but with a prediction as to future improvement because of certain technical changes relating to one of our problems, it is only natural for us to say, "Let us wait a while and see how these predictions work out." In many instances such delay may involve just a short lapse of time. The aviation hazard and its treatment by life insurance companies is a very good recent example. Last year a favorable accident experience followed closely upon new developments and regulations for the air transport industry and already revised practices of many companies reflect this betterment, with many other companies considering such revision at the present moment.

But in the treatment of the silica dust problem we can look forward to no such speedy results. Ten and twenty years and upwards may be necessary for mortality statistics to give evidence of the expected improvement. It seems only fair that we make some concession to the factor of time in such a situation and try to anticipate the future improvement. Such a course points to a possible reduction at the present time in extra premium charges for silica dust industries unless our present charges have been and still are inadequate. For example, it is my opinion that under present scales of rates the metal mining industries have, in general, been treated too favorably and liberalization here may not be warranted.

A further consideration is based on my understanding that the prognosis for individuals with any considerable amount of previous silica dust exposure is not favorable, even though future exposure is much reduced or entirely avoided. This means that improved working conditions in the future may not be of much benefit to a large proportion of workers already in the silica dust industries and that only newcomers and those who have entered within the last few years will get the real benefit of the improved working atmosphere. Some practical method of assessing extra premiums might be devised for

recognizing this difference, such as, perhaps, higher premiums for the older ages, age being a very definite measure and reflecting to some extent the duration of employment in the industry. The duration of exposure itself would undoubtedly be the best measure but it presents certain practical difficulties in the problem of understatement to secure a more favorable rate and in the determination of previous exposure in other dusty trades.

Since the hazard of breathing a specific siliceous dust is dependent on the extent or duration of exposure, it follows that the worker exposed for less than his full working time should be a better risk and accordingly should be assessed with a lower extra premium than is assessed for full-time exposure. In one group of industries in particular, the stone working industries, we have found many instances of such part-time exposure. There are many small units, dressing mills as well as retail monument yards, where the proprietors and salesmen do some carving or cutting or lettering as the need arises. If the facts as to extent of exposure can be determined, it seems to me that a pro rata charge would be sufficiently conservative, since the hazard probably decreases in greater proportion than the dosage.

Before closing my remarks I should like to emphasize a point hurriedly passed over by the authors because it is outside the scope of the scheduled discussion. Although there may be little evidence that non-siliceous dusts produce any pulmonary disease, some of these dusts do have toxic effects and must not be ignored for purposes of life insurance underwriting.

Let me take this opportunity to congratulate Dr. Lanza and his co-worker Mr. Vane on a very distinctive contribution to the literature of life insurance underwriting.

Chairman Bender: Discussion was to have been continued by Daniel Harrington, Chief of the Health and Safety Branch of the Bureau of Mines. However, Dr. Dickson received a wire from the gentleman stating it was impossible for him to leave Washington. His remarks will, however, appear in the printed Proceedings of this meeting.

Discussion—Underwriting Aspects of the Industrial Dust Hazard

DANIEL HARRINGTON, *Chief*

Health and Safety Branch, Bureau of Mines
Washington, D. C.

IN discussing this paper which is both concise and informative, the viewpoint will be that of one engaged in health and safety work confined almost wholly to one industry, mining, and it is noted that the paper by Dr. Lanza and Mr. Vane stresses mining as being of especial importance in connection with the industrial dust hazard. The Lanza-Vane paper is concerned largely with the health aspects of the problem and this discussion will take a somewhat more inclusive viewpoint with safety, rather than health predominating, though on the other hand the discussion will in large part be limited to mining rather than industrial work in general.

In connection with the problems likely to confront the employer in future compensation obligations with regard to dust, it should be remembered that breathing dust is by no means the only hazard offered by dust in industrial work that may require compensation payment; even here, the harm to the health caused by breathing dust is not necessarily confined to the lungs, as dust of some kinds is known to have a detrimental effect on the nose, throat, and bronchial passages as well as (indirectly) the heart, stomach, and possibly other internal organs. Externally, dust may cause injury to the eyes, ears, and skin; and some dusts on contact with the perspiration are absorbed with definite harm to the health of the victim. Moreover, heavy concentrations of dust in the air materially reduce visibility; in some instances, as in the storms in the Dust Bowl in the Central West, dust in the air reduced visibility to virtually zero in midday with the sun shining brightly. Obviously, in industrial occupations dependent on artificial

light or with but limited amounts of natural light available, any decrease in visibility caused by air-dust concentrations (and the decrease in visibility may under some circumstances amount to 75 per cent or more) is likely to decrease efficiency materially and also increase accident occurrence, with a resultant increase in compensation commitments. Some dusts (coal, zinc, aluminum, and scores of others, mineral and non-mineral) are explosive, and many are subject to spontaneous combustion with consequent hazards to health, safety, and property. Moreover, many dusts have detrimental effects of various kinds on property as distinguished from persons; machinery, growing vegetation, animals, dwelling houses, and other types of property are damaged by dusts, and compensation of some kind is often exacted from industrial concerns through damage suits or otherwise.

With knowledge of these facts concerning the numerous ways in which dust is likely to be unsafe, unhealthful, or otherwise harmful, it would seem logical for an industrial executive, who is trying to protect his employees from harm and his organization from undue avoidable expenditures because of various dust hazards, to disregard technicalities and concentrate on reduction of dust in the air at his plant instead of splitting hairs as to the size, kind, quantity, or any of the scores of other uncertainties as to dust. No answer is yet available to some of these questions and in many instances probably never will be.

Prevention of dustiness in the air where human beings must work and elimination of dust or retention in confined places and under complete control emphatically should be an integral part of the operation of any industry in future if operators are to avoid heavy expenditures for compensation and other damages where dust is left uncontrolled and sufferers can assign responsibility for its origin. The progressive industrial executive who has become convinced of his obligation in trying to protect his employees as well as his company, wants to know immediately what constitutes a harmful amount of dust and how to eliminate or reduce it; here, again, the answers are not easy or as specific as might be desired.

From the viewpoint of harm to the respiratory system, it is generally stated (though actual knowledge is by no means certain) that the particles of dust, such as those of free silica, that affect health adversely when they enter the lungs are those less than 10 microns in size; various persons have disagreed as to what the maximum may be (10 microns or 8 or 6) and some say that the harm is really done by particles under 2 microns in size. In opposition, it is stated that fibers of asbestos dust as long as 200 microns have been found in the lungs of men and undoubtedly exerted at least some detrimental effect.

Since a micron is only about one twenty-five thousandth of an inch, or much smaller than the naked eye can see, it is usually assumed that the larger dust particles that can be seen floating in air and settle out relatively fast do not harm the health. This is certainly a mistake, as the larger particles that float in the air (some of them 100 or more microns in size), if present in considerable quantities, clog the air passages leading to the lungs; some of these passages have agencies that, under ordinary conditions, intercept dust particles before they can reach the lungs, and this clogging allows the smaller and probably most dangerous dust to enter the lungs unimpeded. A common-sense definition of an atmosphere so dusty that dust-prevention action should be taken is that any atmosphere in which dust can be seen by the naked eye is too dusty not only for health but also, at least in many instances, for safety and efficiency. If the visible dust is eliminated, much of the invisible and probably most dangerous dust will have been removed also, and health and other hazards very likely much minimized or even removed. After the visible dust has been removed it may be necessary under some conditions or with some types of dust to investigate more thoroughly the occurrence of invisible dust by means of the impinger, konimeter, or other instruments; but unless litigation is effective or threatened, such intricate studies are relatively unimportant if the visible dust has been eliminated from the air of working places.

Manifestly, if dust is not produced it cannot get into the air or otherwise become harmful, and some relief from dust troubles in industry can be achieved by using construction, practices, processes, or equipment that tend to obviate dust.

In designing and equipping the places in which employees must work, the progressive industrialist would be wise to try to prevent the formation of dust (or to control it if it must be made), and should choose machinery, methods, processes, construction, etc., with dust-prevention or elimination features instead of those that produce or disseminate dust, even though such features may increase the cost of the machine or process. The emphasis now being placed on dust in its various phases and the high costs contingent upon it make it almost imperative that hereafter more thought be given beforehand to dust prevention than has been given in the past. For example, excessively dusty processes or practices should be discarded for others that produce less dust, or they should be isolated somehow, as by housing them tightly in a separate part of the plant or using them when a minimum number of persons is exposed to them or when there is a maximum of time to remove dust as well as other harmful properties

or constituents. Such installations or practices should be kept in full, efficient operation constantly, however, if they are to perform the service desired. Moreover, a reasonable amount of common sense should be applied in handling a dust-control practice or system; certainly it is the height of folly to use an elaborate exhaust system to take the dust out of part of an establishment and then exhaust the dust-laden air at such a point as to allow it to be taken again into the same or some other part of the establishment (or of an adjacent one) through doors, windows, fans, or by other means.

If dust must be formed, as is true of many industries, several effective methods are available for preventing its dissemination into the air or into places where it may cause trouble. These include wet methods, such as wet instead of dry drilling, wet instead of dry crushing, wet instead of dry handling, and wetting or washing down floors, walls, and rock faces. In general, water is among the most effective available aids to prevent air dustiness, even though in some instances wet drilling does give off some dust and wet grinding is by no means dustless. Notwithstanding a growing tendency in some quarters to decry the effectiveness of water or wet methods, at present their use is probably our best defense against dust dissemination in mining and tunneling.

The use of dust-exhaust devices such as hoods, traps for drilling, etc., has a definite place in dust control, particularly in surface plants and operations. Under some conditions exhaust systems, when properly installed and kept in repair and operation, not only perform the dust-removing function but also more than pay their way in actual value of the recovered dust. An installation in a surface plant processing one of the metals cost approximately $7,000 to install, and the dust recovered daily averaged about $25 in value; moreover, the air of the plant and the surrounding walls and other surfaces were kept virtually free of dust.

On the other hand, although dust traps may be used with dry drilling in certain types of surface or near-surface rock-excavation work, they have not been found applicable to general rock-drilling practice underground. There are several reasons for this, some of which may be removed eventually. Some types of rock cannot be drilled dry with any degree of effectiveness, and as yet the available types of rock-drilling traps or trapping systems are not workable with wet drilling. If dry drilling can be done, some of the traps now available will ordinarily do a good job of removing the dust from drilling, but this will leave the rock and other surfaces (top, timbers, ledges, sides, and floor in many cases) dry. Subsequent blasting, mucking,

timbering, hauling, etc., will then help to throw into the air the dust certain to be created in large quantities by blasting and in smaller quantities by the other processes; the dust thus thrown into the air will settle on dry surfaces, to be thrown into suspension by any and all activities. Dust traps also are cumbersome, especially when used in the confined places typical of mining and tunneling. As previously indicated, however, dust traps with dry drilling may have a definite place in surface or near-surface operations, where dry drilling can be done, and under some circumstances may have advantages over wet drilling, especially in very cold weather. Some experiments are now under way with use of dust traps in wet drilling, and these may ultimately have a place in some dust-prevention campaigns.

Ventilation is probably the most effective dust-control practice available for many industries, although it ranks second to wet methods in others. Where conditions permit effective ventilation, air-borne dust can be removed or its concentration kept diluted to give almost complete immunity from harm of a respiratory or contact nature occasioned by dust and to minimize dust hazards of almost all kinds, including those connected with spontaneous combustion, though it may enhance hazards of dust explosibility in some instances. The ventilation in some cases should be induced by exhaust methods, but in others forced or blowing systems are more effective, especially in connection with underground work in places where temperature of the air is high, owing to rock, dripping water, or some such other condition as rapid oxidation of surrounding rock, timber, etc.

To obtain really effective ventilation in such work as tunneling and metal mining is usually very difficult, because ordinarily only one opening is driven, instead of having workings in pairs, as in coal mining, where fresh air flows in one opening and return or contaminated air in the other. Conditions caused by the dust problem may force parallel openings to be used in connection with many tunneling and metal-mining operations of the future, and, notwithstanding the violent objection this suggestion will raise, it is logical to believe that in many instances there would be definite savings in dollars and cents as well as in safety by the parallel-opening system, with its numerous advantages as to ventilation, health, and drainage, compared with the many disadvantages and hazards of the present one-opening system. In fact, about the only real advantage of the latter is the fact that its use is customary.

Dry drilling is without doubt the dustiest occupation in mining, tunneling, and quarrying, and it continues to be used far too much in mine, quarry, and tunnel work in the United States; certainly, if dry

drilling is allowed at all, it should be done only with auger twist drills or with simultaneous use of effective dust traps. Wet drilling, as practiced in the United States with water forced or drawn through the drill steel by compressed air, is now "under fire" because it produces relatively large quantities of dust even though the material floating in the air usually is wet. Much of this criticism is imaginary but some of it is legitimate, and the trouble is due largely to the fact that the holes are collared or started dry and, in some instances at least, drilled dry for several inches. The dust caused by this dry starting impregnates the usually stagnant air of the ordinary underground tunnel or metal-mine working place and leaves it so for much if not most of the working shift after the dry starting has been completed. Sometimes the driller unduly limits the amount of water sent through the drill steel, and while he nominally is drilling wet, the actual practice is closely analogous to dry drilling because the compressed air that flows through the drill steel forces excessive quantities of water-entrained dust bubbles or particles out of the drill hole and into the air of the working place. Sometimes, too, the drill is out of repair and allows far too much compressed air to flow through the drill steel. When reasonable procedure is followed and wet drills of present types are used, the amount of dust from drilling can be controlled; in fact, this is actually being done by numerous alert, conscientious mining and tunneling companies. Some companies in the United States and Canada are now experimenting with rock drills that do not allow compressed air to go through the drill steel but require the water to be forced through the steel by its own pressure; these drills are said to cause less dust than the usual types in which water is sent through the steel by compressed air, but the practicability of the newer type of drill is debatable and its use is still rather uncommon in the United States or Canada.

Without doubt, present-day wet drills can be used so as to keep dustiness of air well within safe limits if employed with care and judgment in collaring wet, using plenty of clean, dirt-free water, keeping the drills in good repair with adequate regulation of the proportion of water and compressed air flowing through the drill steel, preventing the exhaust from the drill from impinging on nearby dusty surfaces of muck piles, floors, walls, or timbers and throwing dust into the air, and planning, insofar as feasible, to reduce drilling of upper holes to a minimum. Upper holes, whether drilled dry or wet, are relatively heavy producers of dust in the air of working places.

Next to dry drilling, blasting rock in mining, quarrying, and tunneling causes the most finely divided dust to be thrown into the

air of working places. Also, blasting with the heavy charges of explosive required in many mining and tunneling operations impregnates the surrounding air as well as the muck piles with considerable quantities of poisonous gases, such as carbon monoxide, possibly oxides of nitrogen, and, in some kinds of rock, hydrogen sulfide and other dangerous sulfurous fumes. Virtually all of these occur in such quantities and percentages under some circumstances as to asphyxiate persons who may breathe them or to cause serious, often permanent, illness. Many of those who have studied the incidence of dust disease believe that breathing even small quantities or percentages of extraneous or harmful gases such as carbon monoxide, oxides of nitrogen, hydrogen sulfide, etc., in the atmosphere inflames or otherwise adversely affects the respiratory organs, especially the lungs, making them much more susceptible to harm from breathing dust particles.

Without doubt, blasting practice of the past must be modified and reformed if employers are to avoid heavy penalties in compensation and other charges due to the inclusion of occupational diseases (one being dust disease) as cause for compensation payments in the laws of the various states. If at all feasible, blasting should be done at the end of the working shift or on an off shift, and the dust and gas-laden air from blasting should be removed or diluted thoroughly before men return to work after blasting has been done. Ventilation is, of course, an essential agency in cleansing the air after blasting; in addition, the place (walls, floor, roof or top, timbers, etc.) should be wetted thoroughly before blasting, a water blast should be kept in operation during and after blasting, and the region, including the muck pile, thoroughly wetted when the workers return to the face region after blasting. The muck pile should also be kept well-wetted at all times while it is being loaded out, as the water not only tends to settle the dust but also either to absorb or otherwise aid in the dilution or elimination of harmful or poisonous gases from blasting, which usually cling to the blasted material. In general, the larger the quantity of explosive used, the more finely divided is the blasted material, the more heavily is the air impregnated with dust and smoke, and the greater is the quantity of the poisonous gases left at the place; hence, methods should be used that will tend to reduce the quantity of explosives used and the gases and dust produced. In this it will be found that the use of stemming or of some types of blasting plugs now available will confine the explosive more definitely and therefore increase its efficiency and reduce the quantity of explosive used. It will also reduce the amount of poisonous gas evolved and possibly the violence of the blast as well as its tendency to produce dust and disseminate it into the surrounding air. It would be well for users of

explosives to investigate types known to give off minimum quantities of harmful gases on detonation and to use them; moreover, elimination of the use of fuse and substitution of electric blasting would help to reduce the poisonous gases from the blasting process. Explosives that have been held in storage too long or stored under unfavorable conditions as to moisture, temperature, etc., are likely to give off large quantities of harmful gases if they detonate at all; hence, the utmost care should be taken in storing explosives and in seeing that they are not held in storage too long before being used.

Dust respirators of various types are now available for use in excessively dusty atmospheres. They are cumbersome and inhibit freedom of movement, making them undesirable for constant wear throughout a working shift, though they can be worn readily for several minutes at a time; nevertheless, they do give adequate protection to wearers against inhalation of dust into the respiratory system but essentially no protection against poisonous or asphyxiating gases. Dust respirators should be regarded as useful for emergency purposes only and then for relatively short periods, and adequate dust protection for workers should be of a more fundamental nature; that is, the quantity of dust should be reduced at its source.

What might be designated good housekeeping has a definite place in dust-prevention activities. Dusty materials should be stored in dust-tight containers or bins. Dusty processes, including the handling of dusty material, should be conducted, as far as feasible, in dust-tight compartments or structures, tightness of which should be maintained. Ledges or surfaces on which dust may lodge and accumulate, to be easily thrown into the air by some shock or movement, should be changed, as far as feasible, to prevent opportunity for such accumulation; if this cannot be done, such surfaces should be cleaned periodically by vacuum process, by washing down, or by sweeping (the latter is a relatively poor method and should be used if possible only when a minimum number of other workers is present). Floors should be kept as dust-free as practicable, and the use of water or washing methods is recommended where they can be used. Air for the use of workers certainly should not come from dusty sources.

One of the most important features in connection with prevention of harm to the health from dust is the physical examination; unfortunately, this is clouded by so much controversy that its effectiveness is largely nullified, though it should be one of the principal cogs in the machinery operating to minimize harm to the health from dust in industry. Without doubt, every person employed in an industry

where considerable dust may be present in the air should have a rigid physical examination by a competent doctor before employment; if the examination reveals respiratory trouble, especially if tuberculosis is involved, the person should not be employed. Moreover, at intervals not exceeding 12 months (possibly 6 months) every person in an industry wherein much dust is present in the air should have a rigid physical examination; if disease is shown to be developing, the victim should be transferred to non-dusty employment, if such is available. Handling physical examinations in a manner fair to the worker as well as to the employer is a problem that should be worked out, and it must be admitted that up to date this has not been done at all adequately.

Prevention of dust disease is chiefly an employer responsibility and he should not hesitate to shoulder it with a view not only to safeguarding the health of persons under his supervision but also to relieving his firm from the heavy financial burden likely to be assessed by compensation or legal authorities should any considerable number of employees become afflicted with dust disease. When one realizes how little is known about the cause or cure or even the alleviation of this disease, it seems sensible to do anything practicable to reduce the amount of dust in the air of working places. This does not mean that the employee should not help himself, as it is manifestly as much to the employee's interest to avoid dust or any other occupational disease as to avoid having accidents. However, information on occupational disease (including dust disease) is not readily available in a form understandable to the worker: the employer should therefore conduct an educational campaign to inform his foremen about matters pertaining to occupational diseases and have them transmit the information to the workers. The latter will then be able to cooperate more wholeheartedly in trying to eradicate these dread diseases from industry or at least control them more effectively than has been done in the past.

In this somewhat extended discussion there has been much deviation from the paper by Messrs. Lanza and Vane and this has been done with the idea of giving a viewpoint of a somewhat different nature than that embodied in the very excellent presentation made by Dr. Lanza and Mr. Vane.

Chairman Bender: We have Mr. R. J. Vane of the Statistical Bureau of the Metropolitan Life with us. I wonder if he will talk on this subject?

Mr. Vane: Mr. Chairman, ladies and gentlemen: I hadn't expected to come up and say anything. I thought with the long paper which we had prepared and which Dr. Lanza read, and also the rather long, full discussion, which Mr. Pitler gave, this story about dust and underwriting might be concluded.

I am in quite hearty agreement with many of the suggestions Mr. Pitler has made with regard to applying these data to our underwriting practices. He pushed the implication to the limit but I think, on the whole, the conclusions are sound.

In my own mind, it is very clear that for underwriting purposes we have just those two dusts to consider. Present classification lists which divide up trades in which there is exposure to organic dust on the basis of exp to such dust, I think are not up to the minute. We will all ha sit down and revise our occupational rating lists, throwing out ma of the classifications based on exposure to dust, unless we are dealing with silica and asbestos.

You may have noticed that Dr. Lanza and I limited the discussion to tuberculosis. We could not get sufficient data for other diseases of the lungs upon which to base any conclusions. It may be that pneumonia and some of the other acute respiratory diseases may show a greater frequency in some of these occupations. From what data we have, I doubt that they would be of sufficient magnitude to affect the total mortality and, consequently, the underwriting practices.

I don't want to say anything more, but I would be very glad to answer any questions with regard to the underwriting phase. Any questions on the clinical or laboratory side, you will please address to Dr. Lanza.

Dr. Dickson: Mr. Vane, will you make a comment about foundry workers? I don't recall any reference to that in the paper.

Mr. Vane: The mortality of foundry workers, you mean?

Dr. Dickson: Well a general comment. The whole foundry is dusty, of course.

Mr. Vane: Yes, the whole foundry is dusty, but the only place where we have found extremely high death rates from tuberculosis has been among the chippers. Probably if we had a breakdown for all the casting cleaners, that is, the tumbling barrel operators, the sand blasters, the scratchers and all the variety of operations that are performed to scrape off the sand which adheres to the casting, we would find very high death rates from tuberculosis, because the sand is, as you know, silica.

However, as regards the other workmen, the casters, the furnace men and the men in the other large operations that are carried on in the foundries, the peculiar thing is that they have a tremendous death rate from pneumonia. Whether that is due to the exposure to heat and sudden variations in temperature or whether it is due to the smoke, I do not know. I don't think any studies have really proved the point one way or the other, but it is a striking fact that pneumonia is the leading cause of death among foundry workers, — a situation we have found for no other occupation. But as regards dust, I would come back to the point and say that in the casting cleaning group, you would expect a high mortality from tuberculosis.

Dr. Evans: May I ask the doctor's experience with the dust bowl people?

Mr. Vane: Dr. Lanza, the question has been asked as to what is the mortality in the dust bowl, among people living in the dust bowl area I don't think we have any statistics on that.

Dr. Lanza: You mean with respect to the effect on the lungs?

Dr. Evans: Silicosis, pneumonia and other things.

Dr. Lanza: I don't think that such a thing is possible from a clinical point of view. Outside of industry, you don't get concentrations of dust containing silica sufficient to cause silicosis.

For instance, on certain types of roads in Southwest Missouri and in Oklahoma, they use chats, which are the tailings from the mines, to build the roads, and those chats are practically 100 per cent silica. When you get a high wind blowing, you see dense clouds of dust in the air and one might naturally think then that the effect upon the non-industrial population of these quantities of dusts stirred up by the wind would be comparable to the industrial effects upon the men who are working underground, but, as a matter of fact, most of the dust that you can see in dust storms, and when a wind sweeps across chat roads, is far too large to get into the lungs. You can't see the dust that causes silicosis and whereas very frequently the evidence of dust in the air may be an indication of a hazard, just as often as not you can have a hazard with no indication of dust in the air. So you can't tie those two things together.

Then, again, the large quantities of dust in the air due to dust storms or wind blowing across chat roads and that sort of thing, or chat piles, lasts but a short time. You don't get what you get in industrial occupations, where a man is exposed to large quantities of dust for eight hours a day, day in and day out, week in and week out, month in and month out and year after year. There is nothing comparable to that.

I don't know anything about the question of dust in the dust bowl and that sort of thing and what it may cause in the way of bronchitis or other respiratory infections, or influenza or pneumonia. I don't doubt that there are many circumstances under which those dusts are irritating to the upper air passages but I don't know of any specific or accurate data on that subject.

Chairman Bender: Are there any other questions on this paper?

Dr. Clark Young: Just one question about the distinction between silicosis itself and tuberculosis arising from silicosis. Do you assume that the silicosis deaths are practically always tubercular in nature or do some of them have it without tuberculosis?

Dr. Lanza: I think perhaps that can best be illustrated in this way. Men go to work in an occupation and what do you find? Well, the majority of men who go to work in any occupation have relatively normal, healthy lungs. Some of them have had childhood infections, with healed scars up here at the apex of the lungs, the sort of thing that you find in occasional X-rays. Then, a few, in proportion, will have had tuberculous infections, maybe an arrested tuberculosis or slightly active. As you know, when you make studies of thousands of men, it is quite surprising to find men who have had active tuberculosis who are healed and those with very considerable tuberculosis who have never been sick and never knew they had it.

These men go to work in an industry in which they are exposed to silica dust. The fellow who has an old healed childhood scar up here will suffer in no way, I believe, from the fellow who goes to work and whose lungs are ordinarily healthy. The man who has had a certain amount of tubercle damage to his lungs is going to get a silica effect sooner than the other two. Now, if the silica exposure is severe enough, ultimately the fellow who had healthy lungs is going to get sufficient silica damage to those lungs to cause him a clinical condition, which may then result in an activation of a tubercle infection.

A large amount of time, energy and oratory has been expended in trying to decide whether tubercle infection in silicosis is exogeneous or endogeneous. I am sure I don't know. I don't think it is of very considerable practical importance. But what does happen when he has sustained a certain amount of silica damage and he has activated a tubercle infection, those two react upon each other, the tubercle stimulating the formation of fibrous tissue and intensifying the action of the silica, intensifying the clinical picture, intensifying the dyspnea and making the prognosis much worse.

Such cases run to two general clinical types, — the essentially chronic case that goes on for years and finally succumbs to his pulmonary disease, which is a combination of silica and tubercle, and those who, when they activate their tubercular infection go right to pieces and die in a period of months.

You can't generalize too far, but, as a rule, the more severe the silica damage to the lungs, the more pronounced is the tubercle feature of the condition as well.

Chairman Bender: Are there any other questions? We thank you very much, gentlemen, for your definite contribution to our program.

The final paper of the morning is one on Injuries to the Brain and Spinal Cord — Effects and Treatment, by Dr. W. McK. Craig, Associate Professor of Neuro-Surgery of the University of Minnesota Medical School and Neuro-Surgeon of the Mayo Clinic, Rochester, and certified by the American Board of Psychiatry and Neurology and by the American Board of Surgery. He is a member of numerous societies, the author of at least 128 medical papers, including numerous chapters in textbooks and systems of medicine. Dr. Craig.

Dr. Craig: Mr. Chairman and members of the American Life Convention and guests: To be included in this delightful group is indeed a rare privilege, but your program chairman gave me a rather difficult task, and when I received Dr. Dickson's letter inviting me to appear here, he assumed no responsibility for what I might say, because he told me that I could select my own subject. This perturbed me for some time until a report from the National Safety Council came to my desk, and it occurred to me that in view of the fact that accidents both from motor cars and the accidents in the home seem to be increasing to such an extent that, if it has not already affected your statistics, it should in the near future.