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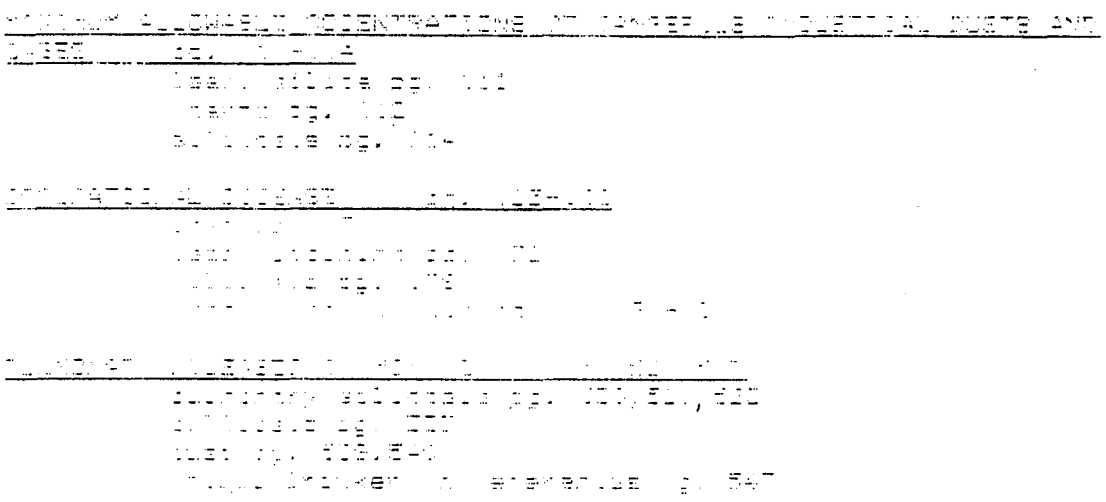

NATIONAL
SAFETY
COUNCIL

TRANSACTIONS

1940 Pt. 1

TRANSACTIONS

29th Council NATIONAL SAFETY ~~CONGRESS~~

Transactions

GENERAL AND INDUSTRIAL
SESSIONS

CHICAGO
OCTOBER 7-11, 1940 - pt 1

NATIONAL SAFETY COUNCIL, INC.
20 North Wacker Drive, Chicago

Contents

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of prompt and continuous delivery of essential weapons of defense we must not only train new workers and retrain older workers, but we must restrain accidents, which fritter away our supply of skilled hands producing these weapons.

Maximum Allowable Concentrations of Dangerous Industrial Dusts and Gases

By ALICE HAMILTON, M.D.

Medical Consultant to the U. S. Division of Labor Standards, Washington, D. C.

I have been asked to make a brief summary of our present-day knowledge with regard to dangerous degrees of air pollution from gases, vapors, dusts and fumes, and the data which have been secured so far as to the maximum allowable concentration which can be assumed to be within safe limits, below the danger point. A few of the dangerous dusts and vapors have been studied intensively enough to afford us fairly full data on which to establish such standards, but only a few, and even with regard to them our knowledge is not complete, and it would be a great mistake to embody in legal regulations the standards which we are at present tentatively recommending, for it is certain that, as our knowledge increases through investigations in this field, we shall have to change our figures. I believe that the State departments of labor or of health which have adopted standards of allowable concentrations regard them as advisory only, and that is right.

The first attempt at the establishment of a maximum figure of air contamination by an industrial poison was made in England in 1910 by Duckering and Legge, who used the procedure since generally adopted; that is, they determined the amount of lead in the air of a workroom and matched it up with the record of lead poisoning in that room. In this way they were able to state that, if the amount of lead were kept under 0.5 mg. per cubic meter of air, there would be no serious plumbism, no palsy or encephalopathy and only rarely colic. This amount, you see, was not to be taken as safe, only as free from serious danger. In 1933 our own Public Health Service concluded that the amount of lead dust or fumes in the air should be kept 0.15 mg. per cubic meter for safety. This was on the basis of an investigation of a storage battery plant, and a sickness study of 2,303 men employed there. For "prolonged exposure," the figure must, it seems, be still lower, for some cases were found which had developed even at 1.2 mg.

I believe the next important contribution in this field was made by the Benzol Committee of the National Safety Council, which, as you all remember, established 100 parts per million as the maximum allowable concentration for benzol. That figure still stands, though we have a good deal of evidence that it is too high. You are probably familiar with two remarkable reports on industrial benzol poisoning which appeared in 1939; one from the Massachusetts State Department of Labor on benzol in the manufacture of shoes; the other from the New York State Department, on benzol in rotogravure printing. In both studies cases were discovered that had developed after exposure to concentrations below 100 ppm. As a result, Dr. Francis Hunter of Boston declared that, "It is doubtful whether any concentration of benzene greater than zero is safe over a long period of time."

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American Standards Association Committee

Substance	Parts per Million	Length of exposure,
Carbon monoxide	100	Not over 8 hours daily
	400	" " 1 " "
Benzol	100	" " 8 " "
Carbon disulphide	20	" " " " "
Hydrogen sulphide	20	" " " " "

Suggested Standards for Illinois and Massachusetts

Substance	Parts per Million		Milligrams per Cubic Meter	
	Illinois	Mass.	Illinois	Mass.
Ammonia	50	100		
Aniline	5	5		
Benzol	75 (and derivatives)	75		
Carbon tetrachloride	100	100		
Chlorine	0.35	1.0		
Chloronaphthalenes			5	1 to 5
Chlorodiphenyls			5	1.0
Chromic acid			1.0	0.1
Gasoline	1000	1000		
Glycols	25			
Hydrochloric acid		10		
Hydrocyanic acid	10	10		
Hydrofluoric acid	3	3		
Lead			1.5	1.5
Mercury			1.5	1.5
Methyl alcohol	100	200		
Nitrogen oxides	10	10		
Nitrobenzol		5		
Ozone		1		
Phosgene		1		
Phosphine		2		
Sulphur dioxide	10	10		
Tetrachlorethane	100	200		
Tetrachlorethylene	100	200		
Trichlorethylene	100	200		
Toluol	75	200		
Turpentine	500	200		
Xylol	100	200		
Zinc oxide	150			15

ject, composed of industrial physicians, ventilation engineers, industrial insurance carriers and toxicologists, who are trying to set up standards for use in industry.

So far we have dealt conclusively only with two gases and two volatile solvents, carbon monoxide and hydrogen sulphide, benzol and carbon disulphide. The limits adopted for the present are shown on the accompanying chart. Benzol stands at 100 ppm., chiefly because of the insistence of our insurance members that this is an ideal that can be realized practically while a more rigid standard would be rejected, and we should be left with no standard at all. Myself, I am always willing to accept half a loaf rather than no bread, but Manfred Bowditch, of the Massachusetts Bureau of Occupational Hygiene, is unwilling to go higher than 75 ppm., and Yandell Henderson, of Yale, enters an emphatic protest against 100 ppm., saying that if 20 ppm. is feasible for hydrogen sulphide and carbon disulphide, he can see no reason why it is

Tentative Standards Adopted By Public Authorities For Maximum Allowable Concentration Of Silica Containing Dusts

Authority	Millions of Particles per Cu. Ft. of Air Free Silica Content		
	High	Medium	Low
National Silicosis Conference. 1936. Medical Committee.	5	10 to 20	
Ditto. Engineering Committee.	5	10 to 30	
U. S. Labor Department 1936 Division of Labor Standards	5	15	
New York Department of Labor. 1940	5 (Foundry (Free Silica) 25 Abrasive Sand Cleaning Operations)	10	100
Connecticut Department of Public Health. 1936.	5		
California Industrial Accident Commission. 1936.	5		50
Wisconsin Industrial Commission. 1932.	5	15	
Massachusetts Department of Labor & Industries 1940	5	10 (Granite)	100 25 Foundry Nuisance Dusts

impossible for benzol. Certainly it is to be hoped that ventilation engineering will progress to the point where a lower figure than 100 ppm. may be reasonably demanded for benzol users.

The standard for carbon monoxide does not need to be defended. This gas has been exhaustively studied by the Public Health Service, by State bodies, by industry and by toxicologists in many universities. Carbon disulphide and hydrogen sulphide are the two dangerous gases which must be dealt with in the manufacture of viscose rayon, and the maximum allowable concentrations given in the chart were established through careful studies made by the American Viscose Corporation, the du Pont Company and the Tubize-Chatillon Company.

When it comes to the other toxic substances I have listed here, we can say only that the figures given represent our best judgment at the present moment, but we admit that that judgment is often based on something not far from guess work. Take the one on which I happen to be engaged at the present time, tetrachlorethane. We know it is the most dangerous of the chlorinated hydrocarbons used in industry, and we have known since the first world war what its action is on the human body, for it was used by both the Germans and the British in airplane dope, and in both countries gave rise to cases of acute yellow atrophy of the liver; but, in spite of a voluminous literature and an impressive number of clinical cases, we find no instance in which the amount of poison in the air was determined. Therefore, the standard, ten parts per million, is based on Zangger's statement that this solvent belongs with

aniline and nitrobenzene in the most toxic group. We have little data with regard to the other chlorinated hydrocarbons, except CCl_4, about which we do know somewhat more. As to the others, our knowledge comes chiefly from the studies made by pharmacologists who sought a relatively non-toxic agent for cases of hookworm infection which is not the same thing as industrial poisoning from fumes.

To pass over to that very controversial field, silicosis, I need hardly emphasize the fact, so evident in the accompanying charts, that there is still much confusion and little agreement among the authorities as to the amount of dust containing varying proportions of free silica that may be permitted in a workshop. This is to be expected. Poisoning from gases and fumes, even if slow, does not take fifteen years to make itself evident; silicosis may. Moreover, other factors, aside from the actual proportion of free silica, affect the dangerous character of a given dust.

Take foundry dust. It may be rich in silica, running as high as the notoriously dangerous granite, yet, while consumption has for centuries been the occupational disease of stone cutters, this has not been true of foundry workers. Recent brilliant studies of this particular dust, notably the one by Hatch and Moke, have provided an explanation for the difference between foundry dust and other silicious dusts, for it has been shown the silica content of the former is chiefly in the large dust particles, and as the particle size decreases, the free silica falls, till it may reach, for particles under 2 microns, only one-fifth or even one twenty-fifth of the percentage in particles over 10 microns. This is, however, not true of the foundry dust caused by sandblasting and by chipping, jobs which everyone familiar with the industry knows to be dangerous. Steel foundries are the worst, probably because more chipping is needed on steel castings.

If our knowledge of the minimum dose required to produce poisoning needs amplifying, this is still truer of our knowledge of what constitutes dangerous dust concentrations. Here the Public Health Service is doing much valuable work. Their two monumental investigations, of the Vermont granite industry and of Pennsylvania anthracite mining, have carried us a long way forward, and, in cooperation with the experts of the Saranac Laboratory, they have found it possible to establish certain fundamental facts. For instance, the Laboratory is able to state that the percentage of tuberculosis to be expected among workers in an establishment where there is no special dust hazard, is 4.79 per cent. If the rate is higher, there is something wrong; of course, not necessarily dust in the air; perhaps low wages.

Donald Cummings, of Saranac, urges the adoption of two limiting concentrations of free silica; first, the "primary threshold," a concentration of dust in which a healthy man may be employed throughout his working life without a disabling injury; second, the "secondary threshold," a concentration in which a healthy man will "inevitably contract silicosis if regularly employed for several years." General experience shows that the first is at about five million particles less than ten microns in diameter; the second is probably at a level not higher than 100 million.

At the National Silicosis Conference held in Washington in 1938, this formula was suggested for determining the maximum permissible concentration of free silica in the air breathed. Multiply the percentage of free silica by the total small particle dust count by impinger. If the result is under 5 million, the condition may be looked on as permissible. If it is over 5 million, the condition is to be looked on as poor. For example, 10 per cent free silica with an average total dust count of 30 million particles would mean 0.1 times 30 million, or 3 million, good practice; while 30 per cent free silica and 50 million particles would mean 15 million, too much. This formula cannot be applied to a dust with less than 5 per cent silica.

Both New York and Massachusetts have made fairly recent studies of the foundry industry and of work with granite, such as quarrying and stone cutting, tunnel and foundation excavating. New York recommends for dust containing less than 10 per cent free silica, 100 million particles; for 10 to 70 per cent, 10 million; for 70 per cent and over, 5 million. Foundry dust, averaging 20 per cent free silica, should be kept down to 25 per cent; the dust from abrasive blast-cleaning, averaging 70 per cent down to 10 million. Massachusetts allows 25 million for foundries, 10 million for granite stone mills and 5 million for pure silica dust.

Source of Dust	Percent of Quartz (Free Silica)	Suggested Standard In Million Particles (Below 10 Microns) per Cubic Foot of Air.
Gold Mines, South Africa	86 to 95	3 to 4.5 million.
Metal Mines, Broken Hill, Australia	10 to 17	14 "
Tunneling, Sydney, Australia	90	Under 6 "
Gold Mines, Ontario	5 to 10 (2 Mines) 25 to 35 (" ")	8.5 "
Iron Mines, Canada	50	5 "
Granite Cutting, Barre, Vermont	(Rock, 26.5) Dust, Average 35	9 to 20 "
Metal Mines, Tri-State Area	90	5 "
Potteries, West Virginia	20 to 30	
Anthracite Mines, Pennsylvania.	(Rock 43% Coal, 6% Dust 90% Coal, 2-4% quartz) Rock Workers dust average 35	5 to 10 "
Subway and Foundation Drilling, New York City	Less than 1 to 84.	10 "

We must not forget that the adoption of any of these standards does not mean that there will be no silicosis among the workers. In the South African gold mines, the free silica in the air was brought down to 3 to 4.6 million particles per cubic foot by 1933, but cases have continued to develop, taking 14 years now, on an average as against an earlier average of 9½ years. In Barre, Vermont, the Public Health Service found cases appearing after only ten years at 6-9 million particles and after 9 years, at 20 million. They have placed the maximum allowable number of particles at 9 to 20 million, which is certainly not extreme.

I need not say anything more about the great need of careful studies of actual conditions under which damage from poisons and dusts occurs in industry. It is in our country that these studies must be made. The day is past when we could look to Germany as the source of knowledge of industrial toxicology. Now that authoritative position has passed to us. And this particular method of determining safety limits by means of a combination of experts in several fields, chemists, ventilation engineers, and physicians is peculiarly American. No other country has made anything like the contributions to this branch of industrial hygiene that we have. But we need a great deal more.

Occupational Disease

WEDNESDAY MORNING SESSION

October 9, 1940

The meeting on Occupational Disease, planned as a Panel Discussion, was called to order by the Chairman, Walter S. Paine, Manager, Engineering Department, Aetna Casualty & Surety Company, Hartford, Conn., who presided.

Panel Discussion

CHAIRMAN PAINE: At every meeting that I have attended in this Congress someone has raised the question of industrial disease. This is a subject which has been maligned in some cases and has been a fantasy on the part of others. This, however, should not in any way destroy the realities that are back of the industrial disease problem.

In the discussion of this broad problem, we have asked the medical profession and the law to help us this morning. Each phase of the problem will be discussed on an orderly basis. We have assigned as the first speaker for each of these subjects one of the men on this platform. After he has finished his preamble, we will ask the members on the floor to present any question they may have on the subject.

We have the following subjects to be discussed: "Health Hazards in Spray Painting." Dr. Eugene Walsh, Associate Supervisor of Medical Service, International Harvester Company, Chicago, will open this subject.

"Fatigue and Its Relation to Occupational Disease." This subject will be taken up by Dr. Norbert Enzer, Director of Laboratories, Mount Sinai Hospital, Milwaukee.

"The Toxic Limits of Common Solvents." This subject will be opened up by Dr. Elston L. Belknap, of Milwaukee.

"Lead in Industrial Processes." Dr. Belknap will also handle this subject.

"Skin Affections." This will be handled by Dr. M. J. Reuter, Dermatologist and Syphilologist, of Milwaukee, Wis.

The last, but one of the most important, will be "Should Occupational Diseases Be Considered as Industrial Injuries?" This will be handled by H. A. Nelson, Director of Workmen's Compensation Department, Wisconsin Industrial Commission of Madison.

We all regret that severe illness keeps Dr. Paul Brehm, Supervisor of Industrial Hygiene Unit of Wisconsin State Board of Health at Madison, from being present.

The panel will be opened on "Health Hazards in Spray Painting." Dr. Walsh.

Spray Painting

DR. EUGENE WALSH: Painting is a topic that has received considerable attention. There has been an increasing number of hazards imported into the trade. Most of these have received wide attention, particularly in the past few years. It has been incident to the wide use of the spray gun and of the lacquers, particularly. Many of the solvents and products used in the paint industry have known toxicities. Others are in the experimental stage, so far as use and so far as their hazards to health are concerned.

In the old days of brush or dip or flow painting, still widely used, solvents of rather low volatility were employed, and the hazards associated therewith were not as great. However, when we come to the spray gun, particularly in its use in the recently developed lacquers, we come into a host of new solvents.

The quantities used per year in lacquers and solvents in the paint industry in the United States reach astronomical proportions. Many, many tons are used.

In the paints we have the following hazards: first, the pigments. There has been a growing trend definitely away from lead-bearing pigments toward non-lead pigments. Many of the non-lead pigments have their peculiar hazards. Many of them are relatively innocuous and can be used with relative immunity.

So far as the fluid constituents of paints are concerned, in the order of volatility in general, we have, first, the solvents which are, largely, for paints; the paraffins; oils and varnish; varnish with some turpentine or some spirits of various and sundry types. The lower the volatility, of course, if it is a toxic product, the greater the toxicity.

The ordinary paints and lacquers can be considered together because the problem of the solvents, largely, is what we wish to deal with. The solvents used are relatively low boiling products which volatilize readily, such as acetone, ethyl acetate, butyl acetate, amyl acetate, and so forth.

Next, the paint must have a diluent. In the ordinary paints—oils, turpentine or spirits may be used. In the lacquers—alcohol, toluol, xylol, mineral spirits have been used. In the past, benzol was used. We are glad to say that there is a growing trend away from the use of benzol, which has cumulative actions in the human individual, resulting in the low grade chronic toxicities due to a depression of the blood-forming organs. There is a definite trend toward less toxic diluents.

The next essential part of the lacquers includes the driers, such as ethyl lactate or cellusolve, monomethyl ethers and various other chemical products of known and unknown toxicities. Also, there must be a flexilizer or a substance giving flow to the plastic. These are relatively high-boiling low-volatility products, triphenylphosphate, castor oil, camphor and others, again of known and unknown toxicity.

The toxicities of the various individual products are often known. It is not the toxicity of the pure product, however, that we are often concerned with. It is the product which is used commercially. It is often the impurities in minute quantities that add to the toxicity. For example, we may say there is, in the case of xylol, some question as to its inherent toxicity. However, since benzol is so frequently a contaminant, we do have some benzol toxicity to be considered when xylol is used. Also, when we have two substances which are used to make a paint or lacquer fluid, its toxicity, resulting from the combination of the two substances is not usually that of a summation of the individual toxicities of the two. That is, if we have two products (A and B), A of one-plus toxicity, if we wish to use that terminology, and B of one-plus toxicity, we may get a resulting toxicity of three-plus or something of that nature—just to use an arbitrary form.

The methods of control are: First, substitution of non-toxic products such as we mentioned in getting away from lead-bearing pigments in paints. We are fortunate to have alert chemists in the paint industry who are alive to this problem. They are constantly striving and working with the industrial hygienists and the physicians in developing non-toxic products, so far as both pigments and solvents are concerned.

Another factor that has been considered and is of importance today in reducing toxicity is the mechanical development of the spray gun. Many years ago, in its infancy, no precautions were taken. The engineers weren't particularly interested in it. A man would spray with one or another, spreading the toxic substances about. Today, however, with the development of lower pressure spray guns, with the development of engineering methods of control of vapors and fumes and dusts incident to the process, we have less toxicity.

If an individual corporation is alert in observing and controlling the hazards, very little toxicity need be encountered in most processes. This includes, of course, the precautionary methods for the individual when I speak of the industrial hygienist and the engineer's phase of the problem. If the individual is working in a place where engineering methods are not feasible to control the vapors and toxic products, then the individual must have his respiratory protection to prevent inhalation. Most of the solvents which are used have a narcotic action, and their action is by way of absorption through the respiratory tract. Taking away the fumes is the way of control.

I have very briefly outlined some of the major points. Do you choose to pursue the question?

CHAIRMAN PAINE: Discussion may be carried on from the floor and also

the viewpoint of the panel on this subject, namely spray painting?

EDWARD SMITH Malleable Co., Buffalo, N. was made about spraying

DR. WALSH: Sprayin is a common procedure, largely calcium, it is a re substance. It is irritating tree and may be follow cough. Not infrequently t iants in whitewash whic products in them but, washing is a matter that i week, and perhaps a mor done in most industries d exposure is relatively in hazards are not great.

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X-Ray Examir

DR. ROBERT M. BU State Medical Assn., St. there methods by x-ray time to time or from yea show progress of bronchi sult of these paint inhalatic

DR. WALSH: There would answer your questi tive. Most roentgenologist scoff at the idea of maki bronchiectasis by x-ray. T we see in the x-ray are la blood vessels and fibrous lung. It is true, however, using some paints which one paint that I did not m in the porcelain indus paint—that is another pr using a silica-bearing pair characteristic changes of

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AINE: Discussion will the floor and also from the viewpoint of the panel. May we proceed on this subject, namely health hazards in spray painting?

EDWARD SMITH (Jewell Alloy & Malleable Co., Buffalo, N. Y.): No mention was made about spraying whitewash.

DR. WALSH: Spraying with whitewash is a common procedure. As whitewash is largely calcium, it is a relatively innocuous substance. It is irritating to the respiratory tree and may be followed by a chronic cough. Not infrequently there are contaminants in whitewash which may have toxic products in them but, ordinarily, whitewashing is a matter that is done today, next week, and perhaps a month hence. It isn't done in most industries day after day. The exposure is relatively infrequent and the hazards are not great.

Likewise, most of the whitewashing procedures are done in large, open, airy rooms. That isn't true of some tunnels, of course. If it is done in closed rooms, tunnels and places with poor aeration, it is advisable, if the process is to consume any length of time, to have the individual protected in some manner. Just a simple draft of air or a mechanical dust type respirator probably would be usable if the exposure is to be of any length of time.

X-Ray Examinations

DR. ROBERT M. BURNS (Minnesota State Medical Assn., St. Paul, Minn.): Are there methods by x-ray examination from time to time or from year to year that will show progress of bronchial trouble as a result of these paint inhalations?

DR. WALSH: There are those that would answer your question in the affirmative. Most roentgenologists, however, rather scoff at the idea of making a diagnosis of bronchiectasis by x-ray. The shadows which we see in the x-ray are largely those of the blood vessels and fibrous structures of the lung. It is true, however, if an individual is using some paints which bear silica—and one paint that I did not mention is that used in the porcelain industry, silica-bearing paint—that is another problem. If you are using a silica-bearing paint, you will get the characteristic changes of silicosis.

However, for the ordinary pigment, so little is absorbed, so few of them produce progressive changes in the lung, they rarely show on the x-ray. Most of them are stopped by the mechanical aids in the nose and upper respiratory tract, that is the hairs, the cilia on the lining of the respiratory tract and, as such, are coughed up, rather than persisting in the respiratory tract.

However, if one is using paints over a long period of time, it is conceivable that a chronic bronchitis might result. However, here one is on thin ice. There are many other factors incident to the causation of chronic bronchitis that are equally as, or more, important than the products used in the paints, either the pigments or the solvents.

MR. CARR (Eastman-Kodak Company): In spray painting in rooms, using lead-free gasoline as a solvent, we have noticed as much as 450 parts per million of aromatics, and we find that many lead-free gasolines now sold contain a large proportion of benzol, xylol and toluol. I wonder if much attention has been given to that fact?

DR. WALSH: That is a problem, it is true. Certain fields of oil, particularly those bearing the naphthionic acid oils do, in their cracking processes, result in benzol in considerable portions, in some instances, in the gasoline. The straight paraffin oils may result in some benzol but not so much. It is a factor to be considered. It depends largely upon the amount of benzol present. In some instances it has been said to run as high as 10 per cent in certain gasolines. That is not usually true.

There is one factor to be considered in gasolines, and that is the difference between benzol, which is the ring-structure and benzene, which is a straight hydrocarbon, and relatively innocuous. It has some toxic properties, but they are transitory, largely.

Benzol has developed a chronic toxicity. This particular subject is receiving attention now, but very little has been produced to indicate that benzol is a definite toxic agent in the gasolines produced. But if the aromatics were up to about 450 parts per million, you certainly are on the borderline of toxicity. I think it should be reduced, if it is at all possible, by ordinary methods. At least the men should be watched very carefully for blood changes, so far as benzol exposure is concerned.

MR. CARR: In that connection, it has been necessary for us to go to a respira-

tory protection of the supplied air type, supplied also with a charcoal canister at the man's waist, so that when he breaks the connection temporarily to go back in the room, he still has the protection of the charcoal canister. The connection, when broken, automatically shuts off both the air supply and also the air connection, so that he cannot breathe the room air.

MR. WALSH: That certainly is a fine arrangement.

DR. ALICE HAMILTON (Division of Labor Standards, U. S. Department of Labor, Washington, D. C.): In the Division of Labor Standards in Washington some two or three years ago, when I was visiting automobile plants, I was told they were making a change in their coatings. I wonder whether that has proved to be true? They told me they were substituting synthetic resins for nitrocellulose and as a solvent were using hydrogenated petroleum products which had very low volatility. It seemed as if that were going to be a revolution in coating in automobile factories. I should like very much to know whether that change has been made.

CHAIRMAN PAINE: Can anyone answer the question of Dr. Hamilton?

DR. WALSH: I would like to say to Dr. Hamilton concerning the substitution of synthetic resins for nitrocellulose products in the automotive industry particularly, most of us receive all of our information concerning the newer development of hazards from no person other than Alice Hamilton. If anyone in the room can answer the question, I am sure that she can. She beautifully reviewed the entire literature in a publication that she produced in 1936 called "Recent Changes in the Painter's Trade" which is Bulletin No. 7 of the U. S. Department of Labor. It is a beautiful presentation, and it is worth while for any of you interested in the paint industry to read it.

Concerning the question at hand, I know of no particular changes at the moment that have been instituted in the industries that I have been acquainted with. I do know that such things are in progress. As Dr. Hamilton suggests, I think that they are changes in the right direction, in many instances.

DR. A. L. BROOKS (Fisher Body Division, General Motors Corp.): We do quite a lot of spray painting. I believe there has been no radical change in the solvents and diluents used in the lacquers which we are now employing. The process is very much the same.

There has been some change in the length of time required to dry these lacquers, but the constituents are the same as they were.

W. H. KRAUSE (Allis-Chalmers Mfg. Company, West Allis, Wis.): There have been recent changes in regard to the diluents used in paint. Quite a few of the larger oil companies are now producing hydrogenated naphtha. These do contain considerable quantities of benzol and toluol. I wonder if there had been any experiments made with regard to the toxicities?

Benzol Frowned Upon

DR. WALSH: I should have mentioned when we were speaking of toxicities of solvents, that the medical profession frowns upon the use of benzol as a solvent or diluent in any proportion. We just don't like benzol, regardless of its percentage, because it is so easy to get out of hand, and the chronic effects of its poisoning come on so slowly and so innocuously, they creep up on you before you know it. It requires rather close observation wherever there is any benzol or toluol.

So far as the hydrogenated naphthas are concerned, it has been said that the hydrogenation does not change the chemical structure of the naphtha, and it does not increase, particularly, its toxicity. If it is in relatively large quantities, 400 to 500 parts per million, then there is the problem of the toxicity inherent to the hydrogenated naphtha itself. But we would much prefer not to see benzol at all.

CHAIRMAN PAINE: Dr. Hamilton, do you wish to say anything else on the subject?

DR. HAMILTON: I think not. My study of the subject is four years old. I thought perhaps very important things had come to light since then. Apparently benzol is still a lurking danger which arises in all sorts of unexpected ways, especially as we use more cracked gasoline than we did before. I quite agree that it is something that should be avoided.

We always advise the substitution of toluol, when possible, for benzol, but I don't think any industrial physician would be willing to give a clean bill of health to toluol

We are beginning to look upon it with more suspicion. Dr. Gammon of the du Pont Company believes it is much more harmful than we have thought hitherto. I hope he will, as the years go on, accumulate more accurate knowledge about it so that we may perhaps encourage its disuse, also. I think that probably would be in the right direction.

MR. GUMMAR (Barrett Company): I would like to offer one minor observation. Commercial toluol, as sold in this country, does not contain any measurable amount of benzol.

CHAIRMAN PAINE: We will have to pass on to the next subject: "Fatigue and Its Relation to Occupational Disease." This subject will be opened by Dr. Norbert Enzer, Director of Laboratories, Mount Sinai Hospital, Milwaukee.

Fatigue and Occupational Disease

DR. NORBERT ENZER: At ten-thirty in the morning I generally have a very low level of activity and suffer from fatigue. Fatigue is a state of bodily ill health, of transient character. It is evanescent, as we understand it and as the term is commonly used but, so far as industry is concerned, fatigue is that state of the individual at work which makes it impossible for him to perform what may be considered an average good output.

It is characterized by certain symptoms. It is characterized under ideal conditions of investigation by certain objective findings which I will elaborate in a moment.

In the strictest sense of the term, I suppose an occupational disease is that condition which can be directly traced to some agent or circumstance in employment which can be labeled as the causative factor of the disease. But as our horizon widens and our concept of disease becomes broader, and as we interpret the word "disease" as it was originally used in the Greek sense, a state of variation from health, or dis-ease, then we must regard an occupational disease as any state of ill health which has been contributed to by circumstances of employment.

Under that definition there is hardly a disease which, under some imaginable state of affairs, couldn't be related to what the man is doing. Those are the two extreme definitions of the term "occupational disease."

Now, any disease will produce fatigue. It is almost synonymous that disease limits the output or the capacity for work of the individual who has been affected. What we must recognize today is that fatigue may also constitute a state of disease. In other words, that we recognize fatigue as a functional disorder which must be treated, which must be recognized and corrected, and its causes eliminated.

The mechanism of fatigue is grounded in a very complex explanation which is not yet clear, and probably won't be clarified until a great deal of investigative work has been done. But in the past few years a considerable amount of investigation has been done, most notably at the Harvard School of Industrial Hygiene under Dr. Dill; and in Europe, up to recent times, a great deal of work has been done, particularly in Germany and latterly in Russia; the object being, in the European investigations, to find out what circumstances of employment contributed to fatigue, to correct those circumstances of employment to prevent fatigue and, hence, to increase the output of the individual in terms of his occupation, without sacrificing any quality of his health.

Now, the thing that we lack at present, so far as its applicability to the practice of industrial medicine is concerned, is a yardstick of fatigue. A number of investigations are going on in that direction, and I think before long something will come of it.

Broadly speaking, two main channels of investigation are being carried on. One is a state of fatigue relative to the muscular capacity of the individual. In other words, we relate muscle strength to the load. The other is a form of fatigue which I think is going to be more important as time goes on, and is related to the nervous system. That arises out of the fact that fatigue is produced by agencies which impair the ability of the brain to send impulses to the peripheral muscles so that work may be performed.

Now, monotony of work may be the greatest contributor to fatigue. I use that as an illustration. For example, we found out, in one small investigation, that individuals employed at work which called for dexterity and nimbleness and a high degree of mental alertness fatigued in less than a twenty-

four-hour period. In other words, if a certain standard of measurement was applied in the morning before work began, the same standard applied at the end of the day, we could demonstrate in almost all individuals so tested that they had experienced fatigue, and that that experience was definitely related to the ability of their nervous system to regulate their muscular activity in coordination.

Now, in jobs which call for a stationary position, manipulation of instruments, or monotony of exercise, that kind of fatigue must be recognized by the industrial physician.

The occupational disease, conversely, may produce a fatigue which may not be recognized unless the individuals are subjected to critical examination. For example, we have noted that individuals with a mild type of hypertension, increased blood pressure, have in many instances a lower fatigue threshold than individuals whose blood pressures are normal. Or an enlarged heart, due to valvular disease, a leaky valve, will produce the same phenomenon; or individuals with diabetes or individuals that have a low basal metabolic rate, because of a thyroid gland disturbance, or individuals who are abnormally obese will all show variations in their fatigue thresholds.

The only way that aspect of the subject can properly be investigated is to approach it from the medical point of view first. Examination of the individual becomes the important thing. It is important to apply critical standards of evaluation, then to attempt to correct them by medical program.

The industrial factor of fatigue, which I mentioned first, calls for a recognition that certain kinds of work can produce fatigue, and then to apply methods which will relieve it. I want to illustrate just two:

If we recognize that a certain occupation produces fatigue, we have discovered things which will relieve the fatigue. A man whose job calls for standing at a bench, in more or less one position, doing the same job over and over again, will have fatigue which can be relieved if he can periodically sit down, because his oxygen consumption in the sitting position is greatly reduced over the standing position, and he performs virtually less work within himself. Consequently he has available greater reserves of energy for his performance.

It has been pointed out, and we have been able to show in some small investigations, that if it could be arranged that this job could be done in the sitting position, the output would be increased and the individual would suffer less fatigue.

It has been shown that, in jobs which call for a to-and-fro movement between more or less fixed positions, without much opportunity to rest, if the period is broken hourly or at hour and one-half intervals, and simple freedom of motion allowed to get out of the range of what he was doing, rest is instituted and fatigue is relieved.

We have been able to show that the application of bathing, in certain kinds of industries, will produce a diminution of fatigue and raise the capacity of the individual to resist his industry or his job. Those are some of the features of the occupational aspects of disease, or the industrial aspect of fatigue, rather. With that casual introduction we can go on and attempt to answer some questions.

Tests of Fatigue

CHAIRMAN PAINE: Are there any questions from the floor? This is an interesting subject.

DR. WALSH: I should like to ask a question in relation to various tests of fatigue. The psychologists have all had people putting round pegs in square holes and all that sort of thing, rapidity of processes. That is one method that is not my concept of the way to measure fatigue, but it brings up one point; namely, the relation of fatigue to the mental status of the individual. Again, the psychological yardstick may be applied. What is the relationship of the intelligence quotient of the individual to fatigue? Does the person using his mind in his work fatigue more readily than one doing physical activity in which no mental effort is required?

DR. ENZER: If I may restate the question, I think Dr. Walsh really wants to find out whether individuals of low I.Q., so to speak, experience the same phenomenon of fatigue under the same circumstances as an individual of high I.Q.

DR. WALSH: That is right.

DR. ENZER: I would say he does, though that is not generally recognized.

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BEN JOHANEK (Connor Lumber Co., Wakefield, Mich.): Do you not think a lot of this is caused through improper food?

DR. ENZER: We are now recognizing that nutrition plays an important part in nervous fatigue because we know there are certain elements in food that are important for the integrity of the central nervous system. But we have been able to show in some preliminary experiments and from some surveys that, in a general way, food intake, except in an extreme instance, is not important, but in acute instances it is. What do I mean by that? An individual who has been deprived of food for longer than he is normally accustomed, in other words, if his meals are separated by too long a period of time, fatigue will be produced. We showed that on ourselves. We took no food from early morning until late at night and were able to show increasing fatigue.

We were also able to show—and this is well known, of course—that there are certain kinds of food which give some temporary relief from fatigue, most notably the sugars, carbohydrates, and a good deal has been done on the proteins of diet. But in the sense that I have tried to lay the subject this morning, diet is a factor in fatigue because it becomes a disease state, and a disease state induces fatigue. Diet will produce a state of fatigue if we have some acute disturbance in the habits of diet.

Factor of Noise

MISS MARY LOU SCOTT (Jos. E. Seagram & Son, Louisville, Ky.): I would like to know the effect of factory noise in regard to fatigue.

DR. ENZER: Noise is a factor in fatigue. We demonstrated that objectively in a scientific way in a laboratory, in this manner: We tested individuals for the flicker phenomenon rate which, as I said, constitutes our base line. Afterwards, we stimulated their ear with a sound of a regular intensity, rhythm and rate, the object being to see whether we could fatigue one center of the brain and in that way effect fatigue in another center of the brain, or whether we could distract one center from the other.

We found that this investigation brought to light two rather interesting things, or I would say three, perhaps. One is that there are, constitutionally, individuals who are sensitive to sound, because we could pick out from a group of individuals people whose flicker phenomenon was greatly depressed when their ears were stimulated; which suggests, perhaps, that such individuals work best in a quiet atmosphere. That is a speculation.

Another type of individual constitutionally wasn't affected at all. His flicker phenomenon was exactly the same rate.

The third individual had his flicker phenomenon raised; in other words, he was stimulated by sound. I think casual examination amongst yourselves will probably bring to light illustrations of the individual who can concentrate on reading a book while the radio is going on and understand both of them perfectly satisfactorily.

Now, when we fatigued these individuals by their routine work—in other words, we conducted the same test in the morning and got a certain base line value; then they did their day's work, and we tested them at the end of the day—we found that fatigue accentuated the direction of their natural constitutional tendency. In other words, the individual to whom stimulation of the ear depressed the flicker phenomenon found that depression increased at the end of a day's work.

PAUL M. HARRIS (Western Austin Co., Aurora, Ill.): Does the human system have the ability in any way to build resistance to this monotony, so it doesn't affect fatigue other than the infusion of glucose or the increasing of the oxygenation?

DR. ENZER: That is a phenomenon known as adaptation in a physiological sense and, in the social sense, it is a question of getting used to your job; but the unfortunate part about getting used to your job is you are not the same today as you were five years ago. In other words, the individual today can't get used to the things he was used to five years ago. Age is an important factor in that direction. The longer the individual is at a fatigue-producing job, sooner or later there will come a time when his fatigue becomes manifest.

We have found that our so-called fatigue testing standards showed no important factors, so far as age was concerned; but the older individuals had a lower threshold to the thing that produced the fatigue than the younger individuals. In other words, it is

not entirely true that experience with a job is a guarantee against fatigue.

CHAIRMAN PAINE: In order that you may not be fatigued, I am going to change the subject. I am asking Dr. Belknap to take the two next subjects, 3 and 4: "The Toxic Limits of Common Solvents" and "Lead in Industrial Processes," and dwell on those subjects as one.

Toxic Limits of Solvents

DR. ELSTON L. BELKNAP: Mr. Chairman, I know there may be several distinguished guests but I am sure that we in the medical profession always feel peculiarly honored when Dr. Hamilton is in the audience. If there has been anyone who has been responsible for bringing to the attention of American industry and the medical profession the importance of toxicology in industry, I think it is Dr. Hamilton.

I will emphasize a few of the toxic limits of the common solvents, so-called thresholds. While these limits have been more or less agreed upon by authorities in this field, particularly such as Doctors Drinker, Haggard and Hamilton, there is no threshold that is arbitrarily perfect, and the actual decision of whether or not the solvent has been toxic is the observation of the patient who has been exposed to that solvent, by a physician who must determine whether or not there has been a temporary intoxication and whether or not there has been a residual.

Dr. Walsh has really very ably covered the subject of the hazards of spray painting, and solvents which are the chief hazards of spray painting, but I will mention a few which I have grouped in increasing order of toxicity.

As an example of probably the least toxic solvent—I say least toxic because no solvent is not potentially toxic; in fact, some go so far as to say that all good solvents are toxic—there is gasoline. It has been stated that 1,000 parts per million is a safe limit for continued exposure. The matter of contamination by other substances such as benzol in 5 to 10 per cent in certain gasolines, certainly would alter that safe threshold.

The next group of solvents which are slightly more toxic, are the amyl and butyl acetates and ether which are allowable in 400 parts per million. Some authorities still state that they belong in the 1,000-part-per-million class. The most recent report classes them as 400.

The third group of 200 parts per million include the following: Methanol or wood alcohol which, as you know, may have a serious effect on vision and cause blindness; toluol and xylol, tetrachlorethylene, trichlorethylene and turpentine.

The fourth group, which has dropped to 100 parts per million, include carbon tetrachloride and ethylene dichloride.

Then the fifth group of 75 parts per million, benzene and dichlorbenzene; and finally nitrobenzene, which we, fortunately, actually use very rarely as a solvent, is 5 parts per million.

I think it is important to remember that these solvents, if in sufficient concentration, may kill. This is in contradiction to the other subject that I have been given, which is lead, which very rarely kills. These solvents kill, first of all, by simple suffocation, if they are used in an enclosed space, such as in a vat. But the sinister element of the solvent, of the toxic solvent, is the fact that it affects the central nervous system because, as you know, these solvents are fat solvents and our nervous system is largely made up of fatty tissue or lipoid tissue, so the nervous system is very easily affected. We have involvement, therefore, of the eye, one of the most highly developed nervous structures. Then we have central nervous system involvement, paralysis, inability to walk. Also, we must realize that some of these solvents actually damage the liver and the kidney, causing an acute Bright's disease. It is thought that some of them may cause cirrhosis of the liver.

Fortunately, one of the best solvents and one of the most dangerous is not included in the list at all now, the tetrachlorethylene group used in the airplane industry in the past. That had the peculiar ability to attack every part in the body, including the heart muscle. That, too, is said to be a contaminant of some of our other solvents. It has been mentioned that possibly the former contaminance in trichlorethylene was really the reason why trichlorethylene had originally had a rather bad name.

I think one of the most important things to remember in dealing with the control of a good solvent is that we are dealing with a potentially dangerous substance and not make any effort to conceal that fact from

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anyone in the factory. We of the medical profession have always felt that we like to put our cards on the table. We know that it is a great deal simpler to follow the ancient axiom and tell the truth. So that I admire, for instance, the benzol company which puts on its barrels, the word "Poison." I am not in a position to know whether they more or less did that because they were told to, but I know they do it.

I think there are many other companies that should put the same label on their products. In this connection I would like to tell of a case that I was asked to see, where a young boy, in the middle of the summer, working in a shop which made motors, suddenly became very sick, had severe headache, was dizzy, and had to stop work. He was working with a substance which he regarded as harmless.

When I first heard of the case I insisted I would want to see the process and see the plant and get my information firsthand rather than attempt to talk to some foreman by telephone. (The medical profession as a whole is learning that it can only fulfill its responsibility by demanding the right to know all about the exposure of the patients that come into their hands.)

When I went into the shop I found that this worker was using a large paint brush, dipping it into a large bowl of liquid, which he then slapped on the motor to get the grease off. On this particular hot day he evolved the idea of setting a fan, which ordinarily, perhaps, blew the fumes away from him, directly opposite the bowl of material and blew the fumes directly in his face. He got cooled, and then he got sick.

I asked the foreman what was in that material, and he said he didn't know exactly; it was stock material, perfectly safe, as far as he knew. I said, "Well, I would like to know what it is." So there was considerable upsetting of routine. We finally went out into a shed in the yard, completely dark; we finally got a flashlight, and we found a barrel of benzol marked "Poison."

Now, no one in that shop knew what they were doing. There were a dozen other workers each doing the same thing but who, fortunately, had not gotten this brilliant idea of the fan.

The curious thing about this particular shop was that in another department, not more than thirty feet away, they were doing spray painting, and they were doing it properly. Instead of exhausting the spray overhead and bringing it right back in the worker's nose, they had their suction underneath the objects to be sprayed and took advantage of draft as well as suction. They had good engineering equipment and staff, but they simply didn't know what they were using in this particular instance.

I had another experience with a man who was a so-called neurotic. I think most of you can spot the neurotic, if you have had any shop experience. This man certainly was. He had been to half a dozen doctors. One told him he was going to die, and another one said there was nothing the matter with him.

Again I demanded to see the shop. We found he was spraying an aluminum paint with a harmless solvent, a diluent, with good suction and a good spray gun. However, on further talking with him and confirmation from the foreman, I found he was the one man in the shop who was delegated to clean the spray guns at the end of the working day. He would sit down and squirt material, which he called chloride, in and out of the spray guns, and lean over the vat he was working with. Finally, he noticed he began to get happy and whistle and sing, and he began to look forward to the experience at the end of every day. However, finally he noticed he was getting weak, he was dragging one leg, and his heart was going very fast.

Well, this illustrates one other sample. This was a large corporation. Yes, they were using chloride. Finally, after great difficulty and having to go to the treasurer of the organization and going into the files we found that this was a material which they had bought from a supply house in another city, and they did not know what was in it. We finally found out it was ethylene dichloride.

The course of that individual is interesting, in that we put him in the hospital. It eventually cost that company quite a lot of money. I was in this to find out all I could learn about the action of that particular substance. I put the man in the hospital three or four weeks and did a number of delicate tests on him.

Well, he came through, and interestingly we found that he had an opportunity to de-

velop a cold once or twice. Every time he did, he developed jaundice. Then, of course, we did liver function tests and found reduction of that. We found he had a weakening of the heart muscle. We found a definite loss of the reflex in one ankle-joint, showing a definite involvement of the central nervous system.

We were able to get all the physicians to agree that this man was considerably disabled, that it was due to his occupation. You will understand, I was seeing him not for himself but for the company. I think a good many of them thought I had been a little generous to this malingerer. This man went ahead and got a large settlement. I don't know anything further about the case except that I did hear that the man died and that the death certificate said "cirrhosis of the liver and heart failure." Unfortunately, we didn't have an autopsy to confirm it.

In my opinion, the toxic solvent is one of the most dangerous substances and it may seriously disable and kill.

Lead in Industry

As a preface to the subject of lead in industry I would like to say that I am not specializing entirely in industrial medicine. I am one of that increasing group who feel that industrial medicine is not a specialty in itself but is merely a division of internal medicine or diagnosis, the large field of medicine which in private practice corresponds to or is correlated with the field of general surgery. I am glad that a large proportion of my practice is still private practice. I am also consulting medical director to one of our industrial concerns in Milwaukee, with branches over the country, so that I have an opportunity to see lead and other toxic materials.

When you see a man who has said, "I have lead poisoning," do not forget he may have some other disabilities and diseases, such as acute appendicitis, gallbladder, kidney colic and so forth. In my private practice my interest has been sharpened so that I always make an effort to take a very careful occupational history on a patient, even though he may come to me purely as a heart case, lung case or what-have-you.

In this particular plant in Milwaukee, which began by having and still has, as a large proportion of its work, storage battery plants, when I came there about ten years ago we were having an incidence of about 24 lead cases per 100 employees per year, which corresponds pretty well with the percentage of 15 to 16 reported by the Public Health Service in work about that time.

In five years, by cooperation with the medical department, the engineering department and management, we dropped that to an incidence of 2 per 100, and for the past five years we have had no cases of lead poisoning at all. We are still handling lead, and we are still handling it in large amounts.

I think that in the storage battery industry, probably, men are exposed potentially, at least, to the greatest concentration of lead for absorption directly into their bodies than in any other industry. I am glad to report that, having followed this particular group of workers of about 80 for this period, at the end of ten years they are certainly just as healthy and, in many respects, I think healthier than the general run of the population. Of course, we have reduced their lead absorption, but at times it will break down the physical resistance and respiratory protection. They occasionally have large doses of lead. Many of these workers were persons who had had lead poisoning or serious lead absorption in the period of observation.

Lead is one of the most widely used so-called poisons in well over 200 industries. I think there is no doubt but in the large industry the hazard is well controlled. First of all, it is recognized and then every effort is given. No expense is spared to study the men and to reduce the continuance of any exposure. However, in the small industry, which is the major portion of industry in this country, lead is continually cropping up, and even silicosis, which got the center of the stage in recent years, certainly in Wisconsin, has now taken a back step, and lead has come back into its own, together with the ever increasing use of solvents.

I would like to stress again the danger here is one of non-recognition, that we have our trouble in the small plant, not in the large plant. I will rapidly run through the types of industry where I have seen lead poisoning occur and in which it is an important factor.

The most important is the storage battery industry. Then the streamlining of our automobiles and trucks where, after welding, of

course, as many of you know, they were at first smoothed off by lead paste which, after it cooled, was then ground by machine grinder and produced a great amount of finely divided, inhalable dust. This brings up the fact that the most important hazard in any lead industry is the point of inhalation. Remember that lead dust inhaled is much more dangerous than swallowed, and that very rarely are lead compounds absorbed through the skin, perhaps with the one exception of tetrachloride fluid which is an organic and fat soluble substance.

It is rather amusing that many plants, smaller plants, particularly, have had to give up machine grinding of welded joints. Here is a return of modern industry to the horse-and-buggy stage. They are going back to hand filing where, of course, the particles are heavy and are too large to be inhaled. The problem can be licked, however, even by machine grinding, if one uses respirators.

It has always been said that lead poisoning was very common in painting. My experience in ordinary painting nowadays is it is very rare, particularly on inside painting where lead has been substituted by other substances, such as titanium, zinc and so forth, which are harmless. On the outside, however, lead is still used, in many instances, because it is the one substance that will stand the weather.

Here we come again to the question of finely divided, inhalable dust where men burn off old paint in private homes; not in the large installations, of course, or sand, either by machine sanding or by hand sanding. There we have the question of finely divided dust which is inhaled. You must remember that dust when inhaled passes directly into the system and immediately circulates throughout the entire body.

I make a recommendation, therefore, that any worker that has gotten his lead by burning off or sanding, be given a respirator for that purpose, even though he is an outside worker.

In the wrecking of old buildings and old ships, of course, there is the constant presence of lead absorption when the steel has been coated with several coats. It ideally forms a fume which is so fine as to be almost invisible and is absorbed directly into the system. The only way that a worker doing that can really protect himself, in my opinion, is to use an air-line respirator. I have seen a great many men in one particular type of ship industry, cutting up old ships outdoors in mid-winter, and I have never seen men leaded up so rapidly. Of course, this is an old story. The Navy found this out twenty years ago when they scrapped many of the old ships. Unfortunately, some people in industry, as well as doctors, are not very good on keeping up on their own work, by reading, and experiments have to be made again.

In the smelting of lead or zinc ore and scrap metal, lead poisoning occurs. In pottery making where a glazed mixer is used, one most usually is exposed to lead. In the printing industry we have heard a great deal of lead poisoning.

I had an opportunity recently to make a survey of a group of printers with the Wisconsin Industrial Hygiene Unit. They went into an analysis of the air, and I examined the men. To be sure, our list was not large, some 30 men in fourteen different printing establishments, but it was striking that all of these men were in excellent health and, as far as I could see, there had been no great deal of turnover.

However, there are one or two spots in the printing industry that are danger spots; that is, of course, the man who remelts the old type metal or who saws it, if he uses a machine saw. I think it is interesting, going through our records of printers in the State of Wisconsin, for a great number of years past, we have had only three cases, one of which was really a serious case, and that man was one who remelted—he was the forgotten man, worked down in the basement on a kettle, where nobody paid any attention to him, and there was no protection.

In passing, I will simply name soldering, and that occurs very frequently even in the evaporated milk industry out in the country; tempering of metals, brass foundries, ink making, insecticides. This potentially may occur in leaded steel when it is scrapped. Then I would class as the final one, the risk which says they have no lead hazard. That is the place you should look particularly.

As I said, the important portal of entry is through the nose. If we bear that in mind, the problem of control of lead poisoning is really not difficult. If, of course, your engineers reduce the exposure to 1.5 milligrams, I don't think you will have a case of

lead poisoning. However, as a matter of practical interest, I think it is very difficult to get there in ordinary industry, and I would more or less formulate 3 as a safe level. I think it has also been found by other workers. However, if you get over 3 you certainly have the possibility of serious lead absorption.

I want to emphasize that mere lead absorption does not mean the man has lead poisoning. He may have a lead line, may have lead in the urine, .08 milligrams per liter, but that does not make lead poisoning. That is merely evidence of absorption. He must have, in addition, clinical symptoms of one of the three types of lead absorption.

As a final word in regard to the role of the medical profession in its cooperation in industry, it seems to me that the medical man should first of all be responsible to the highest man in authority in the plant, so that he can go direct to him with any problems that arise, and before any new procedure is used or any new chemical, have a conference with the physician to see whether there is any possible danger, whether there are volatile solvents or lead, so that the danger to the men may be prevented before it happens.

I am not making a plea for absolute substitution for and avoidance of the use of lead. I think industry cannot get along without the use of lead, cannot get along without the use of a number of these solvents; but if industry is forewarned, and if the worker is informed correctly so he will cooperate in the use of protective devices and, finally, the medical profession is allowed to see the patient as often as they see fit, perhaps every two or three weeks, then there need occur no occupational disease in industry.

CHAIRMAN PAINE: Are there any questions on either one of these subjects that you would like to ask?

MR. HUTTER: What is the effect of the smell of kerosene on the worker? Is that a permanent effect or just temporary?

DR. BELKNAP: As far as I know, there is no ill effect from the inhalation of kerosene.

R. S. SKYRM (Employers Liability Assurance Corporation, Boston): Would Dr. Belknap recommend the direct venting of lead pots on linotype machines where the pots are electrically heated and thermostatically controlled so the temperature of the lead does not exceed 500 degrees?

DR. BELKNAP: That is a good practical question. As we know, lead melts at a much lower temperature when volatilized. In my experience, lead does not really volatilize until around 1,000 or 1,100 degrees.

In this group of workers that I am following, some years ago, large lead pots, two or three feet in diameter, were unhooded because of certain changes in technical procedure, and the plant never got around to the hood. I disapproved of that and repeatedly said they would have cases of lead poisoning there if they didn't hood. They said they would just as soon as it was practical. It has never been practicable, and I am here to say we have had no lead cases develop to the point of disability, and we have had very little lead absorption result from that. The same men have worked there for ten years, dipping lead out of the pot, incidentally, at temperatures ranging usually from 850 to 950 degrees. In this group of linotype workers we found no evidence of lead absorption in some of the shops where they had no suction. I would not say in my own experience that hooding is necessary at 500 degrees.

MR. CARR: I wonder if there is any evidence to indicate the toxic limit for butyl alcohol could be raised above that given? We had experience over some twenty years with the same group of men, working in concentrations from 200 to 400 parts per million, and under careful examination they have shown no indication of trouble.

One other question is whether much work has been done on toxic limits of dioxene?

DR. BELKNAP: I think Dr. Hamilton will have to state if she recalls whether we have toxic limits established for dioxene.

DR. HAMILTON: Dioxene is the trade name for diethylene chloride. No toxic limit has been established for that. We considered it a fairly safe solvent until some very disastrous cases occurred in England from over-exposure, causing five deaths. Since then we have looked upon it with a good deal more suspicion. There has not been enough work done on it to establish any maximum allowable concentrations.

DR. BELKNAP: In regard to butyl alcohol, the butyl radical acetate is much less toxic than ethyl or methyl, particularly. So I would be interested in your point of view.

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MR. GUMMAR: I want to endorse completely the principle of maximum clearances of safety standards in engineering, but they should be based on factual data. As regards toluene, there have been enormous quantities used in the lacquer industry since spray painting came into use. It is sprayed into the air as fine mist, and even though it evaporates at a slower rate than benzol, if you inhale the mist, you get severe effects, if it is toxic.

I have employers in four states, New York State, New Jersey, Ohio and Connecticut, who for the past sixteen years have not had any reported cases of chronic toluene poisoning; yet Ohio in the past sixteen years has had over 69 cases of poisoning from gasoline in the petroleum industry.

As to the experimental results, I can find no experimental data indicating the toxicity should be such a low value. Some of the confusion arises from the failure to distinguish between chronic and acute poisoning. It is a fact that in extremely high concentrations, toluene and xylene are more toxic than benzol; that is, if you go into a chamber where there are 20,000 parts per million, you would be unconscious quicker from toluene and xylene than you would with benzene, but that is an entirely separate phenomenon from the slow chronic poisoning.

CHAIRMAN PAINE: The next topic will be "Skin Affections" and that will be handled by Dr. M. J. Reuter.

Skin Affections

DR. M. J. REUTER: When we consider to what extent the skin of the worker is exposed to injuries, the action of chemical irritants and the various infectious agents, it is not surprising that occupational skin diseases are a major problem in industry. The United States Public Health Service estimates 69 per cent of occupational diseases are skin diseases. They carried out a study in 100,000 workers and found, in the course of a year, that one per cent of these workers were affected with some skin disease as a result of their work. This did not include burns, splashes of acids and alkalis, and the ordinary pus infections of scratches or abrasions.

The problem confronting the physician is to determine the relationship between the skin disease and the occupational environment, to discover the nature of the offending agent and to adopt measures designed to cure the condition and prevent its occurrence.

There are many predisposing causes of occupational dermatosis, such as uncleanliness of workrooms, of clothes and of person. Previous skin diseases as, for example, eczema, are potent factors, because individuals with eczema react seven times more frequently to external irritants than non-eczematous individuals. The skin of the young and old is more susceptible to irritation and infection than the skin of persons in middle life. Dry skin is more vulnerable to irritants than oily skin.

The skin of certain parts of the body is especially sensitive, for instance, that of the eyelids and genitalia. Anything which lowers the normal threshold of resistance of the skin may act as a predisposing factor in the production of dermatitis and of infectious lesions. These factors include bruises, marked sweating and maceration, prolonged exposure of the skin to water and especially to alkalis and repeated scrubbing of the skin. These agents tend to remove the normal protective coats of the skin, that is the outer horny layer, and thus expose the sensitive epidermal cells directly to irritating material. A point fairly recently emphasized is that individuals with common athlete's foot are much more prone to occupational dermatitis of the hands. Apparently this infection of the feet acts as a sensitizing factor.

The actual causes of occupational dermatoses are mechanical, physical and biologic agents and chemical irritants. Mechanical agents produce bruises and lacerations, as well as friction and pressure resulting in various inflammations and thickenings of the skin.

The physical agents which may produce an occupational dermatosis are heat and cold, either dry or wet, electricity, ultraviolet rays, either from the sun or artificial, and x-rays or radium.

The biologic agents are the common bacteria, fungi and parasites.

For the most part, however, occupational affections of the skin are the result of contact with irritants of a chemical nature,

usually acids or alkalis producing simple inflammation of the skin.

As a subject for discussion, I might say that probably cleansing agents used by the workers are one of the most potent factors in the causation of occupational dermatitis.

CHAIRMAN PAINE: This is a subject in which there are a number of men interested. Will you expedite your questions?

DR. ENZER: I would like to ask Dr. Reuter to say a work about skin cancer in occupational exposures.

Skin Cancer

DR. REUTER: Ewing has set forth five postulates which must be fulfilled before accepting trauma as the cause of given cancer. Ewing states: "First, the authenticity and adequacy of injury must be established." That is, the patient's statement regarding injury must be substantiated, and there must be actual tissue damage.

"Second, the previous integrity of the wounded part before the injury must be established.

"Third, the cancer or tumor must arise at the point of injury.

"Fourth, there must be a reasonable time limit between injury and the development of the tumor." That is, if the tumor develops within two or three days after the injury, it isn't reasonable that in that short a time a tumor could have developed.

"Fifth, the positive diagnosis must establish that there is an actual tumor present and the nature of the particular tumor."

CHAIRMAN PAINE: Dr. Enzer, do you want to speak to the point?

DR. ENZER: I thought Dr. Reuter might say a word about the increasing attention now being given to highly complex chemical agents which have the property of inducing cancer in the experimental animal. We know of certain agents which have that property in the human. The commonly known one is tar cancer. We know also of certain aniline products which would produce cancer of the skin, of the urinary bladder, and so on. I think that subject is highly complex, but I think it should be brought to your attention that in the dermatology of today we deal not only with dermatitis but also deal with tumors.

DR. REUTER: I have had very little occasion to pass on a question of cancer of the skin arising from alleged contact with some carcinogenic agent.

C. T. DENSMORE (Koppers Co., St. Paul, Minn.): I would like to hear a little about the effects of coal tar and creosote oils in relation to cancer and what-not and also the inhalation of the creosote vapors.

DR. REUTER: Regarding the development of cancer from coal tar and creosote I have had no experience. We do know that coal tar is the nearest to the skin and does produce an inflammation about the hair follicles called folliculitis. It is undoubtedly due to the plugging of the follicles with the tar and perhaps some stimulation of the epidermal layers of the skin from the tar, which sets up this inflammatory reaction around the hair follicles. I think the same is true with creosote. But, regarding the actual development of hypertrophic lesions of the skin, particularly cancer, I have had no experience.

DR. BELKNAP: As for creosote, I have had no actual cases brought to my attention. As far as I know, the fumes would be no more irritating, possibly, than causing an irritative cough. I know of no cases, really of acute chronic bronchitis or other carcinogenic results.

CHAIRMAN PAINE: We will leave any other questions to the end of the session and hear Mr. H. A. Nelson, Industrial Commission of Wisconsin, on the question: "Should occupational diseases be considered as industrial injuries?"

Disease Vs. Injury

H. A. NELSON: Mr. Chairman: The question which I have been assigned is susceptible of treatment in several ways. We can treat it in a highly technical way and quibble about the court construction of the word "injury" or we can treat it in a general way and ask whether occupational diseases should be compensated. That is the real crux of the question today.

It sometimes astonishes me that thirty years after the passage of compensation acts we are still disputing this question of whether we shall compensate a condition which may cause disability and which arises

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out of industry just as clearly as does an accidental injury. There are various ways of meeting the problem. One, of course, is to do nothing about it and that is what about eighteen states in the Union are doing. They are not compensating occupational diseases at all. Out of the other states there are a few which are compensating all occupational diseases, and some of them are compensating by schedule.

Now, schedules are usually designed so that there are a great many diseases which are named, but very generally you will find that they leave out the one occupational disease which causes the most disability. For example, silicosis is left out of a great many of the state laws which have schedules. Therefore, silicosis, which is a serious occupational disease is not compensated.

When we go back to the prime principles of workmen's compensation, we wonder why the compensation fathers didn't face the question at the time the compensation acts were passed. That was the time to do it. Either through inadvertence or, in some cases, through design, occupational diseases were left out of the laws pretty generally, but accidental injuries were to be compensated. I think that was largely because of the rather violent split from the old common law system and leaving out of consideration the question of negligence. It was a rather violent split, but it is surprising that in all of these years occupational diseases still remain uncompensated in so many states.

When we consider the nature of an occupational disease, which is usually a slow, insidious process, over which the man himself, the employee, has no control, but over which the employer, as these physicians have told you this morning, has the entire power of control, there seems to be one hundred times more reason why the employee who cannot protect himself in any possible way should be compensated for occupational disease rather than for accidental injury.

I suppose there is no question but that the real crux of the matter is that of expense to the employer, and that comes because of the so stated accrued liability. Here has been an accrual of dosage which finally eventuates in disease, and this accrual is such that the disease may come at a given moment.

If the employer has a large number of employees who have this accrual of dosage, it may be that in periods of depression the expense to him will be rather great for the time being, and that must be met in various ways. Employers are humanitarian. Most of them would like to do these things but they are also in competition with each other, and that means there must be laws and regulations for the compensating of occupational disease if the job is to be done in a satisfactory way.

Another reason that is often given is that industrial commissions and accident boards are unable to decide the question of occupational disease, as to causation, between the process and the final condition which results in disability. I think that is a rather fatuous objection, in view of the development which has been made in medical science within the last few years.

I think those questions are no more difficult to decide than the questions of aggravation of pre-existing conditions which are clearly under the law, if we may ascribe them to accidents, and also to the obscure conditions which are ascribed to accidents and as to which compensation boards and commissions have to make decisions every day.

Can Stamp Out Disease

But the most glorious thing that has been accomplished by occupational disease laws is the fact that employers have become conscious of the fact that they can stamp out occupational disease. I am glad to say that in Wisconsin we have practically stamped out silicosis, that in a few years more it is going to be practically a negligible thing. Dr. Belknap has shown you how lead poisoning can be almost entirely eradicated. That is the glorious thing which we have achieved through the passage of occupational disease laws. I want to give a very few figures showing the drop in silicosis in the State of Wisconsin since 1933 to 1939. These figures are the percentages of compensation which were paid in silicosis cases as compared with the total. In 1933 the figure was 9.4; 1934, 8.8; 1935, 7.9; then 5 and 2 and .9 per cent, and, in the year 1939, 1.3 per cent which we figure is getting about on a level.

I think there is no question but that the public is demanding that occupational dis-

case be compensated. The trend is coming on. I believe that industry and society will be gratified when the problem has been solved by whatever methods may be adopted and suited to the different states.

CHAIRMAN PAINE: Are there any questions you want to ask Mr. Nelson before we go into the general discussion?

ADOLF AMRAM (Hamlin & Co., New York City): We passed a compensation law in New York State some two or three years ago. Perhaps you recall that first we passed a most illogical law, the all-inclusive act which acted most disastrously not only to industry itself but to the workmen. You are familiar with the history and the recall of that act. I dare say in a number of states it is absolutely right and proper the compensation act should include silicosis, but it should include silicosis on a schedule basis, as I think is the case in Illinois.

MR. NELSON: All states are not as fortunate as some other states. I think we are showing generally throughout all of the states that the problem can be solved.

Protection for Welders

CHAIRMAN PAINE: We have a few minutes. Anyone in the audience has the privilege of asking this panel of doctors any questions they want to raise on these main subjects.

J. S. GREEN (Edgewater Steel Co., Oakmont, Pa.): I would like to ask to what extent an ordinary welder in a small plant should be protected? Once in a while there will be a hardening material which has poisonous results. Other times there will be flame cutting of paint on a product which will raise some noxious poison. To what extent would a welder or a cutter have to be protected?

CHAIRMAN PAINE: The question is, how are we going to protect, and what is the exposure to welders on new material or on old material that has been painted? I will ask Dr. Walsh to answer that question.

DR. WALSH: Within the past couple of years we have had occasion to go over a group of about 1,000 welders. Of that group some 300 have been welding for ten or more years. They were gone over from the clinical point of view. X-rays of the chest were taken. Disability due to sickness over the ten-year period was calculated and seen for each individual man. There certainly was no marked difference in sickness rates and in disability rates in the welders as compared to the general run of employees working at their side in the same plant and in the same economic situation.

When you are considering welders who are cutting painted materials, lead-bearing paints, or who are welding in confined spaces, you have a different problem; but for the ordinary welding group, working in a large, airy room, with ordinary precautions and ventilation, their health is just as good as any other group of workers. If they are cutting paint or toxic coatings of any kind or lead-bearing steel, or if they are working in a confined space, then additional respiratory protection is necessary.

So far as the question that was rampant a few years ago, that is the development of so-called fibrosis of the lungs due to welding, we have not found this to be the case. Dr. Enzer and Dr. Sander at Milwaukee have had occasion to examine at postmortem a few of these individuals who have been welding for long periods of time in confined spaces, usually. They did find there was an increase of iron in the lung but no fibrosis causing harm.

Recently, we also have examined another group of welders, that is a variant of the welding trade, scarfers, in the continuous production of steel. These scarfers use a long torch and blow out the defects in the billets rather than chiseling them out. This group also, while it is an infant in the group of welders, apparently have no health hazards of any consequence in their trade. In other words, I think if ordinary precautions, so far as ventilation and so forth are concerned, we need not be concerned other than if we are using lead or chromium or some of the toxic materials.

CHAIRMAN PAINE: Dr. Enzer will speak for a minute on the subject and then Dr. Belknap will follow him right up.

DR. ENZER: I want to seize the opportunity to make some comments relative to the general attitude of this panel discussion. It is most important that in any approach to the problem of industrial health we remember that a statistical analysis of groups is not a true criterion of the health of the individual. In other words, just like the sur-

goon who might boast of a 99½ per cent recovery rate from a specific operation, the individual who happened to belong to that ½ per cent constitutes 100 per cent mortality for the individual. There is a very sharp contrast there.

Now, when we talk about safe limits of toxic solvents, safe limits of toxic agents, safe limits of an industry, we must remember that the problem is not going to be solved by a blanket generalization of a problem, but by a careful and meticulous analysis and study of the individuals exposed to the industry. I know of no other way that this can be done except that industry realize that the profession of medicine forms the keystone to the solution of these problems.

DR. BELKNAP: I simply wanted to amplify what has already been mentioned, and that is when a welder or cutter works on material that has been painted or coated with lead, he, of course, probably will acquire lead absorption in a serious fashion about as quickly as, if not quicker than, in any other way.

It is very simple now, with the respirators we have available, for a shop welder, whenever he is doing that type of work, to use such a respirator. In my own particular series of cases one of our cases of lead absorption was a welder who had been repairing some ovens containing a large amount of lead.

Also, I had occasion recently to see half a dozen cases of cutters who were cutting up tanks in a large chemical plant. These steel tanks had been lined with lead. They could not understand why the men were getting lead. They went through the lead with a torch 3000 or 4000 degrees and they absorbed lead exceedingly rapidly. It is not a difficult question to solve at all but it is a matter of having perfect confidence in what the work is for and preparing for it.

MR. CARR: In connection with welding, we have had instances of zinc chills from work on galvanized iron. We have also had nitrous fume poisoning from the use of coated electrodes.

I would also like to call attention to a relatively new development, that of the flame cleaning of metals before painting, by the use of a special oxy-acetylene torch. That includes previously painted metals as well as those which simply have mill scale on them.

R. J. WOODHOUSE (Continental Casualty Company, New York City): I had a case a short time ago that I was called upon to investigate. The man worked in a small printing plant where he was assumed to have contracted lead poisoning. After making impinger tests which proved negative, an award was given by the commission to the widow in the amount of $13,000. The lead was melted around 650 to 700 degrees and the experts proved that toxic fumes were not liberated.

I wonder what yardstick the referees used to measure those cases, in the face of such expert testimony.

MR. SKYRM: I would like to know the relation of hemlock poisoning and poison ivy to skin disease.

DR. REUTER: Poison ivy, of course, is the most common plant that causes dermatitis. Hemlock is occasionally the cause. Poison ivy is an intense irritant to most people's skin. It is a question of whether in itself it is not an irritant to all people's skin.

We used to think we knew the cause of poison ivy dermatitis, but more recent work has tended rather to disprove that we do know the actual cause of what it is in the plant that produces dermatitis. Apparently it is a water-soluble fraction rather than an oil.

Its relation to occupational disease depends on what the individual is doing. If he is exposed to it in his occupation, for example, as a farmer, WPA worker, lumberman or any occupation that keeps an individual out-of-doors, and he contacts poison ivy, it certainly would be an occupational disease. Poison ivy dermatitis is apt to be a much more violent dermatitis than that from hemlock.

CHAIRMAN PAINE: Mr. Gumaer, will you repeat your question?

P. W. GUMAER: The question was, what is the basis of the comparatively low maximum concentration of toluol in the absence of any reported occupational disease cases, and are there any experimental data to warrant such a low concentration where such experimental data deals with chronic poisoning and not with acute poisoning?

this ventilating current be interrupted for a period of 24 hours and with the same amount of methane being liberated, there would be 5,760,000 cu. ft. of explosive mixture. Should this be contained in an entry 10 ft. square, the length of the entry would be approximately 11 miles.

Methane detectors are now available which will automatically indicate and record percentages of methane. One type may be located in working sections on cutting and loading machines, in the last breakthrough and along haulage roads. This can be adjusted to give a warning by a signal light or other means at any predetermined methane concentration. For example, at 1 per cent or any specified percentage between .5 per cent and 3 per cent, it will operate special electrical circuits through suitable relays to cut off the power in a section or give warnings in other parts of the mine or to the outside. Methane recorders for continuously indicating and recording methane in main returns particularly have been perfected recently and are designed to be located in fan houses or other convenient places.

There is no relation between the volume of methane given off in a coal mine and the chemical composition of the coal, as both bituminous and anthracite mines produce methane, and methane has been found in metal and other mines when tunneling through carbonaceous shales. The common occurrence and explosibility of methane are responsible for numerous mine disasters and every precaution should be taken to keep it within safe limits. This can best be done by the use and guidance of methane detectors.

Pulmonary Diseases in the Mining Industry

By DR. R. R. SAYERS

Director, United States Bureau of Mines, Washington, D. C.

Although the attention of those interested in diseases peculiar to mining has been focused recently on one dust disease—silicosis—other respiratory diseases may cause more suffering and economic loss. The increased incidence of these diseases usually is attributed to exposure to abnormal air conditions underground, such as sudden changes from high to low temperatures, excessive dust, and noxious gases. The principal respiratory diseases to which the miner is subject are bronchitis, influenza and pneumonia, pulmonary tuberculosis, anthracosilicosis, and silicosis. Although the mortality statistics in some of the tables included in this paper are old they have been corroborated by later, less complete data.

Bronchitis

Bronchitis is an inflammation of the bronchial tubes due, among other causes, to the inhalation of irritants such as dusts (organic and inorganic). Occupations that entail exposure to atmospheres polluted by irritating gases carry with them the risk of bronchitis. According to Collis,[1] it cannot be distinguished clinically from bronchitis occurring among the general population and its association with the inhalation of dust or gases usually is unrecognized.

During middle life bronchitis causes much recurrent invalidity and incapacity. Mortality records compiled by Collis[2] indicate that it is of a chronic traumatic origin associated with irritating atmospheric conditions, rather than of an infectious origin.

Statistics for bronchitis for the general population and for workers exposed to dusts in Great Britain[3] for 1921-23 show the role played by dusts in the increased incidence of the disease in certain industries. The comparative mortality per thousand (ages 26 to 65) for those in occupations exposed to siliceous dust was 214, for those exposed to nonsiliceous dust 59.5, and for the general population 49.6. (See Table 1.)

In this connection it is interesting to note the mortality statistics per thousand (ages 26 to 65) for coal miners of counties of Wales (in which most of the coal mined is anthracite) compared with those for two other groups in England and Wales.[4] The figures for coal miners are 112 for all underground workers and 139 for hewers and getters compared with 46 for males in the same social class and 50 for all occupied and retired males. (See Table 2.)

In a study by the United States Public Health Service[5] 35 cases of chronic bron-

Table 1.—Mortality per 1,000 from Bronchitis in Dusty Occupations and in the General Population, by Age Groups

	Ages at Death					Comparative Mortality Figure
	20—	25—	35—	45—	55—	20—65
Occupations exposed to						
Siliceous dust	1.1	1.0	13.7	44.6	153.7	214.0
Nonsiliceous dust	....	....	4.9	12.8	41.8	59.5
General population	0.5	1.7	5.4	13.3	28.8	49.6

bitis were found among 344 employees of two anthracite mining companies examined. In the study conducted by the Bureau of Mines and the Public Health Service at Picher, Okla.,[4] the highest incidence of silicosis or tuberculosis, or both, was found among men who reported having had bronchitis, pleurisy, and asthma.

Table 2.—Comparative Mortality Figures for Bronchitis per 1,000 of Certain Groups in England and Wales

	Bronchitis
Coal miners[1]	
All underground workers	112
Hewers and getters	139
Males in same social class	46
All occupied and retired males	50
Ratio of rate for hewers and getters to rate for same social class	302

[1] In three counties in Wales.

Table 3.—Influenza-Pneumonia Mortality among Bituminous Coal Miners—October to December 1918

Age Period	Annual Death Rate per 1,000	
	Bituminous Coal Miners	All Industrial White Males
All Ages[1]	50.1	22.3
15-25	29.5	17.5
25-45	62.1	32.6
45-65	44.4	11.7

[1] Fifteen years and over.

Influenza and Pneumonia

Figures of mortality available for 4,700 miners insured in the group department of the Metropolitan Life Insurance Company[5] show that in the last quarter of 1918 during the influenza epidemic 64 of these miners died of influenza or pneumonia. This is equivalent to an annual death rate of 50 per 1,000. Among all occupied males aged 15 years and over, the rate from influenza-pneumonia in the same 3 months was 22 per 1,000 or less than half as high. In the age period 45 to 65 years the rate among bituminous coal miners was about four times as high as among all occupied males in the same age group. (See Table 3.)

Influenza and pneumonia, as reported, were responsible, in the years under study, for 31 per cent of the mortality in the group of bituminous coal miners at ages 16 to 70 in Indiana, Missouri, and Wyoming compared with 19 per cent among both farmers and "all other males" at these ages in the same counties. (See Table 4.)

In 1918, the year of the great epidemic, influenza and pneumonia deaths constituted 55 per cent of total mortality exclusive of accidents among the bituminous miners in contrast with 27 per cent among the farmers and 34 per cent among all other males. These percentages were not as high as the figures for Wilkes-Barre. The fact that the soft-coal counties under consideration are largely rural areas may have made some difference.

In the epidemic year 1920, the proportion of deaths from influenza and pneumonia was smaller than in 1918, but, as was found for the anthracite miners, the bituminous group showed a wider difference in the pro-

Table 4.—Percentage of Deaths from Influenza and Pneumonia (After Deducting Accidents) Among Bituminous Coal Miners Compared with Farmers, and With Other Males in the Same Areas of Several Soft-Coal Producing States[1]

Male Groups Under Comparison	Ages					
	16-70	16-30	31-40	41-50	51-60	61-70
Bituminous coal miners	31.1	49.4	44.7	25.1	19.7	10.4
Farmers	18.7	38.4	32.4	19.7	12.4	10.8
All other males	18.6	30.0	29.0	16.0	11.3	10.8

[1] The death records for soft-coal miners were obtained from 15 coal-producing counties of Indiana, 1916-20, inclusive, 8 coal counties of Missouri, 1917-21, inclusive, and the 6 principal coal-producing counties of Wyoming, 1919-23, inclusive. The record for farmers and for "all other males" was obtained from the above-mentioned coal-mining counties, and also from 3 agricultural counties of Wyoming, 1919-23.

portion of deaths from this cause compared with "all other males" than in 1918. The percentages in 1920 were 29 for the soft-coal miners and 16 for "all other males." In this epidemic the bituminous miners at ages 40 to 60 suffered heavy excess mortality from influenza-pneumonia, but the differential mortality was not quite so great as at ages 16 to 30.

Among hewers and getters of soft coal in England and Wales, the death rate from influenza and pneumonia, 1921-23, was about 12 per cent greater than among males of comparable social status (social class 3). The difference was found to be 5.8 times the probable error of the difference which is certainly a significant variation.[3] Thus, an abnormal mortality from influenza and pneumonia is consistently indicated among miners of coal—both anthracite and bituminous—in England as well as in America.

The pneumonia mortality figures in 1921-23 for occupations in Great Britain exposed to siliceous and nonsiliceous dust compared with those for the general population were given by *Collis*[3] as 134.3, 76.0, and 85.3, respectively; similar figures for influenza were 47.1, 49.6, and 36.5, respectively. (See Table 5.)

According to Brundage,[4] an outstanding feature of the statistics of death for hard-coal miners in the United States is their extraordinary mortality from influenza and pneumonia not only during influenza epidemics but at other times as well. In a study by the Public Health Service at Wilkes-Barre, Pa., in 1915-23 and 1906-25[4] it was found that influenza and pneumonia were responsible for 40 per cent of the total deaths compared with 25 per cent among males at ages 15 to 65 who had been engaged in pursuits other than coal mining in Wilkes-Barre and vicinity. The percentage of deaths from influenza and pneumonia for these two groups by ages for the years mentioned was 19.3 for anthracite miners compared with 13.3 for males in other industries. (See Table 6.)

Tuberculosis

In 1925 Collis[3] said that for coal miners generally the tuberculosis rate apparently is low although miners at the coal face suffer from the disease more than other coal miners underground and workers above ground. Results of more recent investigations seem to contradict the assumption that tuberculosis does not occur among coal miners. An investigation by the Welsh School of Medicine, reported in 1937,[9] indicates that this supposed exemption of coal miners from tuberculosis may be due to a mis- or partial diagnosis. This investigation revealed a high incidence of tuberculosis (35 per cent if the "probable" cases are included) among the pneumoconiotics of the South Wales coal fields and supports the theory that coal dust modifies the tuberculosis, leading to failure to recognize the disease or to the mistaken diagnosis of "bronchitis."

Studies by the United States Public Health Service[10] revealed that 6 per cent of hard-coal miners examined had clinical pulmonary tuberculosis. The average prevalence in the adult male population of the United States is about 2 per cent. The prevalence of pulmonary tuberculosis among hard-coal mining employees at ages below 35 was slightly less than that found through studies among male adults in the general population. In the age group 35 to 44, however, the prevalence of tuberculosis was

Table 5.—Mortality per 1,000 from Pneumonia and Influenza in Dusty Occupations and in the General Population

	Pneumonia					
	Ages at Death					Comparative Mortality Figure
	20-	25-	35-	45-	55-	20-65
Occupations exposed to						
Siliceous dust	3.3	14.4	30.6	47.2	38.7	134.3
Nonsiliceous dust	....	9.6	13.7	28.7	23.9	76.0
General population	4.3	11.5	19.6	24.2	25.7	85.3
	Influenza					
Occupations exposed to						
Siliceous dust	1.1	7.7	11.3	8.7	18.3	47.1
Nonsiliceous dust	5.4	4.8	13.7	10.6	14.9	49.6
General population	2.0	5.2	7.7	9.7	12.0	36.5

about twice, at ages 45 to 54 about 5 times, and for ages above 55 it was about 10 times the rate found in the general population. The highest prevalence of clinical pulmonary tuberculosis occurred among the rock workers (men who had been employed in rock loading or rock extraction for more than 2 or 3 years). After 20 years' service, of which more than 2 or 3 years were in rock work, 37 per cent presented evidence of pulmonary tuberculosis. In a group of 135 completely disabled former anthracite workers, which did not include any known cases of tuberculosis, 10 per cent proved positive for pulmonary tuberculosis. These findings would appear to corroborate those of the Welsh National School of Medicine that indicate that the supposed exemption from tuberculosis of coal miners is erroneous due to incorrect diagnosis. There is evidence that the above increased incidence of tuberculosis is associated with exposure to silica dust.

The incidence of tuberculosis is much higher in metal mines than in coal mines, due no doubt to exposure to dust containing a higher concentration of silica. An investigation in 1914 by the Bureau of Mines in cooperation with the Public Health Service of pulmonary disease among the zinc miners of the Joplin, Mo., district, revealed a high death rate from pulmonary tuberculosis. The mortality rate for tuberculosis for Jasper County was 200.8 per 100,000 for 1911 and 1912 and 229.7 for 1913, according to statistics of the State Board of Health.[11] On the other hand, according to the Jasper County Antituberculosis Society, during the calendar year 1912 there were 720 deaths among miners who had worked 2 years or more underground in Jasper County. Not all

Table 6.—Percentage of Deaths from Influenza and Pneumonia among Anthracite Miners and among Males in Occupations Other Than Mining in Wilkes-Barre and Vicinity

Groups Under Comparison	Ages					
	15-64	15-24	25-34	35-44	45-54	55-64
Anthracite miners	39.8	45.2	67.9	42.9	31.8	19.3
Males in other industries	25.3	28.7	46.2	31.0	16.8	13.3

Table 7.—Mortality per 1,000 from Pulmonary Tuberculosis in Dusty Occupations and in the General Population

	Ages at Death					
	20-	25-	35-	45-	55-	20-65
Occupations exposed to						
Siliceous dust	25.6	63.4	141.0	211.6	150.6	592.2
Nonsiliceous dust	13.6	26.3	46.1	56.4	29.9	172.4
General population	20.6	37.6	43.6	39.0	22.7	163.5

Table 8.—P

Outdoor and i	
Indoor industr	
Coal trade	
Alcohol trade	
Heat and fume	
Dust of	
1. Animal or	
2. Vegetable materials	
3. Silica	

of these 720 deaths occurred in that county but they had contracted the disease there and some of them had moved away. Of 93 miners in the Joplin District examined by Lanza in 1914,[11] 64 showed definite symptoms of pulmonary disease and 39 of the 64 had the classical symptoms of pulmonary tuberculosis.

In a study by the Bureau of Mines and the Public Health Service[12] in Butte, Mont., during 1916 to 1920 the death records of the State Board of Health were examined. These showed that during 1915, 122, during 1916, 126, and during 1917, 169 Butte miners died of tuberculosis. The significance of the death from tuberculosis of 169 miners in 1917 is indicated by comparison with the records of other regions. In 1917 approximately 14,000 men were employed underground in Butte mines; with 169 deaths from tuberculosis for the year, the rate per 100,000 was 1,207. The tuberculosis death rate of Michigan for the 10-year period 1910 to 1920 was 97.4 per 100,000; hence the tuberculosis death rate of Butte miners was nearly 13 times as great as that of Michigan.

The part played by dust, especially dust containing silica, in the production of pulmonary tuberculosis is shown clearly by figures for Great Britain for 1921-23.[3] The death rate per 1,000 for ages 20 to 65 was 592.2 for those exposed to siliceous dust, 172.4 for those exposed to nonsiliceous dust, and 163.5 for the general population; for ages 35 to 44 the figures were 141.0, 46.1, and 43.6, respectively; for ages 45-54 the figures were 211.6, 56.4, and 39.0, respectively; and for ages 55 and over the figures were 150.6, 29.9, and 22.7, respectively. (See Table 7.)

According to Collin[3] the prevalence of bronchitis, pneumonia, and tuberculosis is high among those exposed to silica dust but the incidence of these diseases is not always the same under other conditions such as exposure to other dusts, heat and fumes, or working indoors. (See Table 8.)

Anthracosilicosis

From 1931 to 1937, 87 per cent of the total deaths in Great Britain due to silicosis occurred among the coal miners of South Wales; two-thirds of the deaths and disease occurred in the Swansea Division; and 89 per cent of the medical certificates granted in the Swansea Division related to anthracite collieries.[13]

In the United States the chronic disease among miners due to breathing air containing dust generated in the various processes involved in the mining and preparation of anthracite is called anthracosilicosis. This is a descriptive title for the form of pneumoconiosis commonly called miners' asthma. The disease was found in about 23 per cent of 2,711 employees of three anthracite mining companies studied by the United States Public Health Service in 1933. About 63 per cent of those having anthracosilicosis showed evidence of disability, compared with less than 10 per cent in the control group. Moderate and marked degrees of disability handicapped aproximately 21 per cent of those with anthracosilicosis compared with less than 2 per cent of the men serving as controls.

Wide differences were found in the extent of physical impairment in the several occupational groups. The highest rate of disability from all causes combined was found in the group of men that had changed more than 5 years before the date of examination from very dusty to relatively non-dusty occupations in the industry. In a

Table 8.—Prevalence of Bronchitis, Pneumonia and Tuberculosis Under Certain Conditions of Exposure

Exposure	Bronchitis	Pneumonia	Tuberculosis
Outdoor and indoor life	low	low	low
Indoor industries	low	low	high
Coal trade	high	high	low
Alcohol trade	moderate	high	high
Heat and fumes	high	high	low
Dust of			
1. Animal origin	low	low	low
2. Vegetable husks and debris, certain inorganic materials	high	medium	low
3. Silica	high	high	high

number of instances the change in occupation probably was made on account of physical condition. Miners at the coal face, rock workers, and men who had had appreciable exposure to harmful dusts in other industries also showed evidence of much greater physical impairment than was found in the control group.

Moderate or marked physical impairment from any cause was found in 1.7 per cent of the control group, in 9.9 per cent of the regular miners, and among 12.6 per cent of the rock workers.

There was a marked tendency toward increase in the proportion having physical impairments with increase in the number of years of employment in anthracite. This tendency is manifested whether one considers disability of any kind, disability in which pulmonary infection was contributory, or disability due largely to tuberculosis. In each case the occupational groups arrayed themselves according to extent of physical impairment as follows: (1) Rock workers; (2) regular miners; (3) all others exposed to more than 100 million dust particles per cubic foot of air; and (4) men in haulageways except rock workers.

The pathology of anthracosilicosis as observed in these cases is essentially a deposition of coal dust in the lungs, accompanied by an extensive fibrosis, both diffuse and nodular, with associated functional and degenerative changes, (silicosis modified by coal dust).

That excess mortality from respiratory diseases as a whole is experienced by American hard-coal miners is clear from the studies made by the Public Health Service.[4] The anthracite miners' proportionate mortality from all respiratory diseases was about 1½ times that of the other adult males in the same region. When proportionate mortality was computed for ages 16 to 65 in Wales,[4] it was found that respiratory diseases accounted for 53 per cent of the deaths of anthracite miners compared with 38.2 per cent among males in the same social class. The proportion among hewers and getters of anthracite therefore was 1.4 times that of men of the same social status.

Silicosis

There are various definitions of silicosis but all agree that the cause of the disease is the inhalation of silica or quartz dust.[5] Silicosis is a well-recognized clinical and pathological entity at least as characteristic as that of any cause of death known to epidemiologists.[6] The definition of silicosis, adopted at the International Silicosis Conference in 1930 and reaffirmed at the 1939 Conference and generally accepted is:

"Silicosis is a pathological condition of the lungs due to inhalation of silicon dioxide. It can be produced experimentally in animals."

The first investigation of silicosis in the mining industry in the United States was made in 1914-15 by the Federal Bureau of Mines in cooperation with the United States Public Health Service in the Joplin (Mo.) mining district.[7] As only a few men were examined at that time, it was decided to examine a larger number to gain a more accurate idea of the prevalence of consumption among the miners of this district. From

Table 9.—Mortality from Certain Respiratory Diseases of Those Exposed to Siliceous Dust, to Nonsiliceous Dust, and the General Population—by Age Groups[1]

Groups	Respiratory Diseases																					
	Bronchitis					Influenza					Pneumonia					Tuberculosis					
	20-	25-	35-	45-	55-	20-65	20-	25-	35-	45-	55-	20-65	20-	25-	35-	45-	55-	20-65	20-	25-	35-	45-	55-	20-65
Siliceous dust	1.1	1.0	13.7	44.6	153.7	214.0	1.1	7.7	11.3	8.7	18.3	47.1	3.3	14.4	30.6	47.2	38.7	134.3	25.6	63.4	141.0	211.6	150.6	592.2
Nonsiliceous dust			4.9	12.8	41.8	59.5	5.4	4.8	13.7	10.6	14.9	49.6		9.6	13.7	28.7	23.9	76.0	13.6	26.3	46.1	56.4	29.9	172.4
General population	40.5	1.7	5.4	13.3	28.8	49.6	2.0	5.2	7.7	9.7	12.0	36.5	4.3	11.5	19.6	24.2	25.7	85.3	20.6	37.6	43.6	39.0	22.7	163.5

[1] Made up from tables 1, 5 and 7.

May 15 to December 31, 1915, 720 miners were examined: 45.7 per cent had silicosis and tuberculosis and 5.3 per cent had tuberculosis.[16] A later report (1917) of this same investigation by Lanza and Childs[17] included the results of the first detailed X-ray studies of silicosis made in the United States.

Investigations of mining conditions by Harrington and Lanza[18] in Butte, Mont., in 1921, revealed that 42.4 per cent of 1,018 miners examined showed definite signs of lung damage due to dust.

From July 1, 1927, to June 30, 1932, the Metropolitan Life Insurance Co., the Tri-State Zinc & Lead Ore Producers Association, and the Federal Bureau of Mines operated a cooperative clinic at Picher, Okla.,[19] to demonstrate to industry a workable method for diagnosis of silicosis, to educate workers in preventive measures, and to serve as a model to industries presenting a dust hazard. Of 27,553 individuals examined during this period 5,366 had silicosis, 742 silicosis plus tuberculosis, and 320 uncomplicated tuberculosis.

The chief cause of disability in silicosis is tubercle infection. The silicotic lung appears to be a more favorable medium for the growth of the tubercle bacillus than the normal lung, not because it is fibrotic, nor because its lymphatic drainage is interfered with, but because it contains silica.[19] Apparently silica dust possesses this characteristic of increasing the liability to pulmonary tuberculosis. That this is true is indicated by the statistics quoted in this paper which indicate also that silica dust increases the liability to other respiratory diseases. (See Table 9.)

The results of virtually all studies are in agreement regarding the high mortality rates from tuberculosis for industries in which large numbers of men are exposed to a silica hazard. These rates are so high that they leave no room for doubt of the cause-and-effect relationship between the hazard and the high mortality.[20]

Joint studies made by the Actuarial Society of America and the Association of Life Insurance Medical Directors[20] revealed that there were 65 deaths where 6 were expected (about 11 times the expected number) from tuberculosis in 1925-1936 among a group of underground miners, most of

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whom were employed in metal mines. Tuberculosis deaths were fourteen times the expected among copper miners, nine times the expected among gold and silver miners, and eight and a half times the expected among iron miners. In an earlier study[20] covering the years 1915-1926, there were 18 times as many deaths as expected among lead and zinc miners.

An analysis by the field division of the Saranac Laboratory[21] of the roentgenograms of 5,000 white miners exposed to the inhalation of harmful concentration of silica dust shows that the incidence of pulmonary tuberculosis lesions increases as the degree of reaction to dust progresses. In this group of miners tuberculous lesions were found in approximately one-fourth of those with first stage silicosis, one-half of those in the second stage, and 65 per cent of those in the third stage.[21]

Prevention of Dust Diseases in Mining

The data given in this paper point to dust as the greatest contributor to the excessive mortality from pulmonary diseases experienced in the mining industry and to silica dust as the immediate cause of the disease silicosis. Therefore, the principal method of preventing these diseases is to eliminate as far as possible or reduce to harmless concentration the dust in the breathing zone of the workers. Experience has shown that the properties of a dust that determine its capacity to produce injurious effects are its composition, the quantity suspended in the atmosphere breathed by the worker, and its particle size.

Although any dust may be harmful to breath under certain conditions, as indicated by the data presented here that show a higher incidence of pulmonary diseases in nonsiliceous dusty industries than in the general population, physiological studies reveal that, in mining the harmfulness of a dust is proportional to its content of free silica or quartz, in particles small enough to enter the finer spaces of the lungs, and in high enough concentration to overcome the normal defenses of the respiratory system.

The prevention of dust diseases is the function of the physician and the engineer, working together to determine the extent of the dust hazard in industry and methods for its control.

The first step is to determine what dusts are harmful and in what concentrations. The procedure is somewhat as follows: It is observed that respiratory diseases appear more frequently or with greater severity among workers in certain industries or in connection with certain operations within an industry. The physician attempts to determine the extent of the injury to the health of those exposed and the cause. If the cause appears to be exposure to excessive dust, the engineer samples the air to determine the kind, amount, and size of the dust present. The physician and the engineer can correlate the results of their investigations to determine allowable concentrations of dust, exposure to which is not likely to cause harmful physiological reactions. The engineer is then in a position to devise measures for the reduction of the dust to a harmless concentration. The aim, of course, should be to eliminate the dust entirely or to reduce the dust to a concentration that will not cause disability during the working lifetime.

Allowable Concentrations of Dust in Mining

Investigations conducted over a period of years show that exposure to certain concentrations of harmful dusts for a certain time causes varying degrees of respiratory disability but that below these concentrations disabling pulmonary diseases do not occur within a working lifetime. The classical example of this is the experience in South Africa with the reduction in mortality from silicosis that has followed the reduction in air dustiness in the mines of that country.

The reduction in the incidence of silicosis in the South African mines attests the effectiveness of the control measures employed and the practicability of using a definite dust concentration as a guide in setting allowable limits of air dustiness in the prevention of pneumoconiosis in dusty industries. The production rate of silicosis in the scheduled mines of South Africa[22] in 1932-33 was 9.59 per 1,000 compared with 21.95 in 1917-18. (See Table 10.)

The incidence rates for silicosis uncomplicated by active tuberculosis among South African miners in 1936-1937 was between 8 and 9 per 1,000 per annum.[23] The liability among the whole body of miners to contract the disease in 1936-37 was less by 65

Table 10.—Average Number of Miners Examined Annually and Production Rates for Silicosis in Each Triennial Period Since 1917-18

Period	Average Number of Miners Examined Annually	Production Rate of Silicosis per 1,000	Period	Average Number of Miners Examined Annually	Production Rate of Silicosis per 1,000
1917-20	14,070	21.95	1926-29	14,624	21.11
1920-23	13,260	19.16	1929-32	16,774	13.87
1923-26	12,523	33.28	1932-35	20,139	9.59

per cent than it was in 1920-23. Among the "new Rand miners" who entered the industry by way of initial examination since 1916 and who have been exposed solely to the progressively improving occupation conditions that have obtained since that date, the liability to contract silicosis was less by 87 per cent than the similar liability among all miners working for an equal period in 1920-23. These "new Rand miners" constituted 77 per cent of all working miners examined. The group of pre-1916 miners, who in 1935-36 constituted less than 17 per cent of the total number of working miners contributed nearly 78 per cent of the new cases of silicosis.

In the study by the Public Health Service[6] of anthracosilicosis among hard coal miners silicosis was not found in a control group of mining employees whose dust exposure averaged less than 5 million particles per cubic foot of air.

The prevalence of anthracosilicosis among the entire group of employees was found to be about 23 per cent.

Among all except rock workers, less than 2 per cent of the men developed anthracosilicosis when the duration of employment was less than 15 years, regardless of the amount of dust in the air.

Among men exposed 15 to 24 years to dust containing less than 5 per cent free silica, 14 per cent of those who had worked where the average dust count was 100 to 199 million particles per cubic foot, 29 per cent of those exposed to 200 to 299 million particles, and 58 per cent of the men who had worked for this period in more than 300 million particles per cubic foot, developed anthracosilicosis.

Among men employed for more than 25 years in dust containing less than 5 per cent free silica, the proportion of persons found with anthracosilicosis under different concentrations of dust was as follows: 5 to 99 million particles, 7 per cent; 100 to 199 million particles, 54 per cent; 200 to 299 million particles, 71 per cent; 300 or more million particles per cubic foot, 89 per cent.

With the exception of miners, their helpers, and rock workers, about 25 per cent of all the men employed underground developed anthracosilicosis after a working period of more than 25 years. This group was exposed to dust having a quartz content of about 13 per cent.

The prevalence of anthracosilicosis among persons who had been exposed for more than 2 or 3 years to dust of which about 35 per cent was free silica, varied from 10 per cent among those who had worked in concentrations of less than 200 million particles per cubic foot for less than 15 years, to 92 per cent among those who had been employed for more than 25 years in dust concentrations exceeding 300 million particles per cubic foot, more than 2 or 3 years of which time was spent in rock work.

Age per se appeared to play a minor role in the development of anthracosilicosis.

Analysis of the data for the purpose of determining safe limits of dust exposure indicated that employment in an atmosphere containing less than 50 million dust particles per cubic foot would produce a negligible number of cases of anthracosilicosis when the quartz content of the dust was less than 5 per cent. In the gangways where the silica content of the dust was about 13 per cent, a safe limit appeared to be 10 to 15 million

Table 11.—Pneumoconiosis Cases Occurring Among 798 Workers Exposed to Mixed Dust, Classified by Average Dust Concentration, Length of Employment, and Number of Workers Exposed

Average Dust Concentration (Million Particles per Cubic Foot)	Years in Industry				
	0-4.9	5-9.9	10-14.9	15-19.9	20 and Over
0-9:					
Number of pneumoconiosis cases	0	0	0	0	0
Number of workers exposed	81	56	29	13	8
Percent with pneumoconiosis	0%	0%	0%	0%	0%
10-24:					
Number of pneumoconiosis cases	0	0	0	2	4
Number of workers exposed	123	94	66	29	23
Percent with pneumoconiosis	0%	0%	0%	6.9%	17.4%
25-49:					
Number of pneumoconiosis cases	0	0	3	3	6
Number of workers exposed	43	45	31	11	18
Percent with pneumoconiosis	0%	0%	9.7%	27.3%	33.3%
50 and over:					
Number of pneumoconiosis cases	0	4	5	4	4
Number of workers exposed	45	44	25	8	6
Percent with pneumoconiosis	0%	9.1%	20.0%	50.0%	66.7%

particles per cubic foot. The limit of tolerance for rock workers was set tentatively at 5 to 10 million particles per cubic foot of air. Similar results were obtained by the Public Health Service in an investigation of pneumoconiosis among mica and perinatite workers. (See Table 11.)

The relation of the average dust concentration and duration of exposure to the percentage of workers found to have pneumoconiosis was as follows: Of those who had worked in an average concentration of 50 million particles of dust per cubic foot of air, 9 per cent had pneumoconiosis in 5 years, 20 per cent in 10 years, 50 per cent in 15 years, and 66 per cent in 20 years. Of those who worked in an average concentration of 25 million particles 10 per cent had pneumoconiosis in 10 years, 27 per cent in 15 years, and 33 per cent in 20 years. Of those who had worked in an average concentration of 10 million particles of dust, 7 per cent had pneumoconiosis in 15 years and 17 per cent in 20 years. (See Fig. 1.)

The data obtained in other studies of dusty trades, anthracite mining, asbestos textile manufacture, and the pottery industry have been treated in a similar fashion and have yielded similar, although usually less regular, results. The position of the threshold value below which no cases were found and the magnitude of the trends between per cent affected and both time and intensity of exposure varies in different sets of data, depending, to a large extent, on the chemical nature of the dust to which workers were exposed and possibly on the regularity of employment.

It is tempting to seek to summarize the data of table 12 and figure 1 into a simple mathematical relation such as the following: With each twofold increase in average dust concentration above 10 million particles per cubic foot, the probability of finding cases of silicosis (in men exposed for comparable lengths of time) is approximately doubled. Application of the standard methods of curve fitting would yield more refined descriptions of these data. Nevertheless, it does not seem to be advisable to set up any mathematical treatment because, of all the men who entered the industry, only the survivors have been studied. There is no adequate way of finding out whether or not men who dropped out of the industry were in a better or worse state of health as far as silicosis is concerned than men who remained in the industry, and thus there is no way of introducing a correction for this element of selection into a mathematical

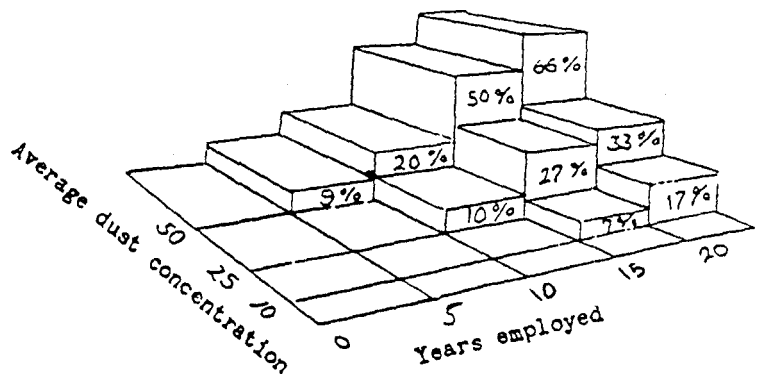

Figure 1.—Relation of average dust concentration and duration of dust exposure to the percentage of workers found to have pneumoconiosis. Based on data presented in Table 11.

treatment. It seems to be preferable to present the facts as simply as possible, together with the precautions to be kept in mind in studying them, and to emphasize that the principal use of these data is to show the degree of improvement in health to be expected in the future if dust formation and dust dispersal are brought under control in these and in similar industries.

Methods of Dust Control

After the physician and engineer have learned by observation and experimentation what kinds of dust are harmful and in what concentrations, it is the function of the engineer to evaluate the hazard in a particular industry, plant, or mine and devise measures for its elimination. The first step is to become thoroughly familiar with the industry being studied to determine the dusty occupations, the number of persons in each, and the various activities and time devoted to each activity in any one occupation.[21]

The amount of harmful dust created by various activities can be determined by taking samples of the atmosphere in their vicinity and counting and identifying the dust particles. Sampling of air is also a means of checking the efficiency of any control measures that may be adopted.

Several devices are available for collecting and counting dust samples. The impinger, however, is the instrument used most frequently in the United States. The Bureau of Mines and the Public Health Service have issued publications describing the method of sampling dust by the impinger (as well as by other methods) and procedures for counting and identifying the particles.[24, 25]

Dust sampling is complicated by the fact that air dustiness fluctuates rapidly from moment to moment. A single dust count represents only the amount of dust in the atmosphere at the time and place of sampling and gives no indication of the average exposure of the worker over a period of years, especially if he has not worked continuously at one job. The ultimate lung injury that he may suffer will depend upon the integrated effect of the dust over a long period.[26] A proper measure of dust exposure requires a picture of the variations in dustiness. The best procedure is to determine the dustiness during different stages of an operation and, with the aid of a time study, break down the worker's day according to the time spent in each step; from these data the average dust exposure of the worker can be calculated.

Drilling.—Experience in South Africa[27] with new methods of sampling have revealed that more dust is produced by rock drills

Table 12.—Period Worked at Face by First-Stage Silicotics

Job	Average Number of Years	Number of Men	Job	Average Number of Years	Number of Men
Shovelers' helpers (bruno men)	6.8	8	Machinemen (drillers)	11.4	348
Shovelers	8.4	428	Powdermen	11.5	24
Drillers' helpers (dummies)	8.4	79	Shovel bosses	15.8	21
Trackmen	9.8	52	Ground bosses	19.0	54
Roof trimmers	11.3	26			

than had been suspected, the difference consisting entirely of dust too fine to be detected by the older methods. The importance of control of drilling dust as a measure of the prevention of silicosis is demonstrated by the fact that the incidence of silicosis has remained consistently and considerably higher among machine drillers than among those who have never worked machines, and the excess incidence among machine drillers has been in direct proportion to the amount of time spent upon rock drilling.

Until relatively recently drilling of holes for blasting was a negligible producer of dust or air dustiness in coal mines especially other than anthracite mines. The hand-operated auger drill produces very little fine dust, but with the extension of mechanized mining power drills are becoming more and more essential and some of them are heavy producers of finely divided dust; this is especially true of reciprocating drills of the jackhammer type drilling dry and using compressed air in the clearing of holes either in coal or in roof or floor material.

Blasting.—The exceptionally heavy incidence of silicosis among rock drill miners in the early period of the gold-mining industry in South Africa[25] was due to the exposure of the rock-drill miner not only to drilling dust but to high concentrations of dust from blasting, and frequently to the inhalation of varying amounts of the irritant fumes produced by blasting, owing to the methods of blasting then in use. Blasting produces large quantities of very fine dust, the finest fractions of which tend to remain suspended in the mine air for a long time, unless removed by ventilation.

Shoveling or Mucking.—In the Picher, Okla., study,[7] it was difficult to show the relationship of the duties of the men to silicosis because of frequent change of occupation. All examined were divided into two groups—those exposed to large amounts of dust (those employed at the face) and those exposed to relatively small amounts (those away from the face). In the order of exposure to the dust hazard, the men at the face included shovelers' helpers, shovelers, drill helpers, machinemen, trackmen, roof trimmers, powdermen, shovel bosses, and ground bosses. According to analysis of the occupations of face workers the hazard was greater for shovelers' helpers and shovelers than for machinemen if length of time required to produce first-stage silicosis is a criterion. It is noted from table 13 that the average number of years worked by shovelers' helpers and shovelers was 6.8 and 8.4 years, respectively, while that for machinemen was 11.4 years.

Loading and Hauling.—Under some conditions hand loading of coal throws into the air of the working place considerable quantities of dust, but the amount is about one-tenth that caused by some types of mechanical loading. The greatly increased occurrence of dust from mechanical loading is due not only to the stirring of the dust into the air by the loading machines themselves but also to the greater rapidity of breaking down and taking coal from the face regions which increases other dust-producing processes, especially blasting, cutting, and hauling.[26]

Poorly constructed or poorly maintained pit cars or over-topping of coal in cars allows fall off or fine material to sift through to the tracks to be ground under the wheels of rolling equipment or the hoofs of mules and then to be thrown into the air by speeding cars or shuffling mules or men.[26]

Where conveyors (either the shaker or belt type) are used they form potential sources of dust clouds at the points where the coal is dropped or thrown which occurs at the discharge ends only. Also, where conveyors are used conditions at the face differ from those experienced with the older methods of working in that the miners are closer together, there is a tendency with the more systematic working for a larger number of the men to perform dust-raising operations at the same time, and the air speed at the faces is higher.[30] These conditions, if not controlled, tend to increase the dust exposure to the face workers.

Machine coal cutting.—When coal-cutting machines came into the picture early in the present century to supersede in large part both hand cutting and blasting off the solid, the really heavy producer of coal dust that the worker has had to endure was at hand, and this was especially true of the chain machines operated by electricity. Top cutting is the worst offender in so far as impregnating the air with dust is concerned, shearing occupies an intermediate position, and undercutting is the least offensive in this respect. However, undercutting machines under some conditions (such as cutting dry in friable coal with dull bits in a confined place with little or no circulating air) may produce air dustiness so dense as to reduce visibility almost to zero with available types of mine lighting and to cause between 100,000,000 and 500,000,000 (or even more) dust particles to be carried per cubic foot of air of the face region.[30]

Methods of Control

The principal measures for controlling dust in mining operations are ventilation, wet methods, proper blasting practice, the use of personal respiratory protection, and physical examination.

Ventilation.—The more or less generally accepted conclusion that coal miners suffer less from miners' consumption than metal miners, who either die early in life or are incapacitated in middle age, is due almost entirely to the superior working conditions in coal mines as a result chiefly of ventilation.

Approximately in order of their importance, the main features that affect air in metal mines are (1) movement, (2) temperature, (3) relative humidity, (4) gases, and (5) dusts. In coal mines the order of importance would probably be (1) movement, (2) gases, (3) dusts, (4) relative humidity, and (5) temperature.[31]

The matter of movement of air from the surface, through the working faces or places, and then back to the surface is by all means the most important consideration in effecting adequate ventilation of mines and in protecting the health and forwarding and maintaining the working efficiency of the underground workers. If adequate quantities of pure air are directed to the places where men work, most of the requirements of health and many of the demands of safety will be well served.[31]

Ventilation probably is the most effective dust-control measure although under some conditions it ranks second to wet methods. Where ventilation can be applied effectively, dust can be removed from the air or its concentration can be so diluted as to render it almost harmless.[29]

Wet methods.—As dry drilling is the dustiest operation in mining, wet drilling will keep air dustiness within safe limits if used with care and judgment in collaring wet, using plenty of clean, dirt-free water, and keeping the drills in good repair with adequate regulation of the proportion of water and compressed air flowing through the drill steel.[29]

In tests by the Bureau of Mines[32] the dust concentration in the air during actual drilling decreased as the water flow through the drill increased. The water flows for stopers was 0 to 3 gallons per minute, and that for drifters from 0 to 1.02 gallons per minute. The rate of decrease for stoper drills was greater for water flows from 0 to 1.3 gallons per minute than for higher water flows. The rate of decrease for drifter drills was similar to that for stoper drills at corresponding water flows and rather constant for water flows of 0 to 1.02 gallons per minute.

The results of this study shows that as water flow through the drills was increased the dust concentrations in the air decreased, but they do not show whether the decrease was caused directly by the increased water flow, by some condition influenced by variation in water flow, or by a combination of these factors.

In most pneumatic rock drills air passes

through the drill steel with the water. Ordinarily some of the high-pressure air supplied to the drill for its operation escapes into the back end of the drill steel, even when the operating throttle is not in the blowing position, and travels up to the face of the hole being drilled. The amount of air escaping is affected by the water flow through the drill steel; as the water flow through the drill steel increases the air flow decreases. Mining officials in South Africa concluded that such air leakage, although probably facilitating removal by the water of the cuttings from the hole, was responsible for entrapping fine dust and blowing it into the air during drilling.[31] Consequently they have devoted much attention to designing drills that permit the minimum practical air leakage through the drill steel and have established regulations on the use of such drills. Canada is also doing much work along this line, and recent reports state that such drills are practical and produce about one-fourth the dust concentration of corresponding drills without the "dustless-head" features.[31]

The results of this study show that operators can reduce dust concentrations resulting from drilling by increasing the water flow through the drill steel, either by increasing water pressure or by the use of water tubes of larger inside diameter.

Tests also have been conducted by the Bureau of Mines[32] on the effectiveness of spraying water along the outside of the drill steel compared with that of standard wet-drilling practice. The tests show that spraying water along the outside of the drill steel should not take the place of regular wet drilling. However, if a method is developed by which the device may be used on holes of any inclination, it might prove to be a valuable accessory to regular wet drilling in collaring and drilling the first 2 feet of a hole. Tests show that the greatest amount of dust is thrown into the air of a working place in drilling the first 2 feet of a hole.[32]

Wet drilling standards have been adopted in New York State for drilling in various classes of rock. The requirements for drilling in Class II rock (rock containing more than 10 per cent free silica) in confined areas, which include the use of mechanical ventilation, are quoted below:[34]

1. "Wet drilling shall mean the continuous application of water through the central hole in the hollow drill steel to the bottom of the drill hole. The central hole in the steel shall not be less than ¼" diameter.

2. "The water flow shall be maintained continuously whenever the drill is in operation, including the period of collaring or starting the drill hole. The valve in the water line shall be of such design that it has only two fixed positions, namely, closed and completely open. Six months after date of approval, all wet drills shall be equipped with a combination air throttle and water valve of such design that the required rate of water flow through the drill steel will be insured whenever the air throttle is in the operating positions, half speed as well as full speed.

3. "The water shall be clean and bacteria free and shall be delivered to the drills under pressure from an adequate source of supply. Portable water tanks for individual drills shall not be used unless approved by the Industrial Commissioner.

"Wet drilling in Class II rock in confined areas shall be used with mechanical ventilation, or in confined areas under decking by natural ventilation, if approved by the Industrial Commissioner and subject to the following provisions:

1. "The rock surfaces for a distance of 30 feet from the heading or other drilling area shall be maintained in a damp condition.

2. "Hand-held drills (maximum piston bore, 3") minimum rate of water flow through drill steel, 0.75 gallon per minute; maximum rate of air flow through drill steel at full speed, 2.0 cubic feet per minute; maximum rate of air flow through the drill steel at half-speed operation, 1.0 cubic foot per minute; minimum rate of general ventilation in the drilling area, 500 cubic feet per minute per drill and shall be increased if found upon tests by the Industrial Commissioner that the dust concentration is not in accordance with the requirements of the Code.

3. "Drifter-mounted and wagon-mounted drills (maximum piston bore, 4") minimum rate of general ventilation per drill in the drilling area, as shown in the table 'Limitations in Air and Water Flow Through Steel.'

4. "In the case of positive-pressure ventilation, the air must be well distributed over the drilling and must not create drafts

at the various drilling positions, in a manner satisfactory to the Industrial Commissioner. The intake for the ventilating system must be located at a point free from contamination with dust or provided with an efficient filter.

5. "In the case of exhaust ventilation, the location of the intake pipe or pipes must be such as to insure the removal of air from the entire drilling area in a manner satisfactory to the Industrial Commissioner.

6. "The required ventilation may be obtained by means of a recirculating air-cleaning apparatus provided the dust concentration in the air at the breathing zone of the drill operators is maintained at a level below that required in Rule 33-2.3 and the apparatus is otherwise acceptable to the Industrial Commissioner.

7. "The above rates of ventilation shall be increased whenever necessary to reduce dust concentration to the amount permitted by Rule 33-2.3."

Proper blasting practice.—Methods of controlling dust and fumes in mines by proper blasting practice have been determined by members of the Bureau of Mines staff by observation and experiment.[3, 4] 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 should be removed or thoroughly diluted before the men return to work. Ventilation is an essential agency in cleansing the air where blasting has been done. Results of investigations by the Bureau of Mines[3] show settling rate of dust after blasting under the following conditions:

1. The atmosphere was bratticed off for 3 hours while the dust samples were being taken. In this period, under these conditions, the dust concentration dropped from approximately 5,000 million particles per cubic foot to 50 million particles per cubic foot.

2. The atmosphere was bratticed off until 26 minutes after blasting when the brattice was raised and natural ventilation (which was virtually negligible except for eddy currents was allowed to dilute the dust-laden air. It required 2 hours and 20 minutes after blasting for the dust concentration to drop from approximately 5,000 million particles per cubic foot to 50 million particles per cubic foot, and 2 hours and 50 minutes to drop to 15 million particles per cubic foot.

3. The atmosphere was bratticed off until 37 minutes after blasting, when the brattice was raised and 10-inch canvas tubing installed to about 50 feet from the face. At 46 minutes after blasting, about 1,000 cubic feet of air per minute was forced into the face. It required 66 minutes after blasting for the dust concentration to drop to 50 million particles per cubic foot, and 70 minutes to drop to 15 million particles per cubic foot.

The accompanying table shows the time, in minutes, required for the dust concentration to drop from 825 million particles per cubic foot to 50 million and to 15 million particles per cubic foot under the three different conditions:

Ventilating Condition	Time, in minutes, to drop from 825,000,000 particles per cubic foot to—	
	50	15
No ventilation	121	181
Natural ventilation	105	135
Mechanical ventilation	20	24

In addition, the place (walls, floor, roof or top, and timbers) should be wetted thoroughly before blasting is done, and a water blast should be used during and after the blasting. The region should be wetted thoroughly when the men return to the face region after blasting, and the muck pile should be kept well wetted at all times while it is loaded out, as the water not only lays the dust but also either absorbs or otherwise aids in diluting or eliminating harmful or poisonous gases that follow blasting and that usually cling to the blasted material.

Tests[5] on the effectiveness of a spray of water mist in reducing the concentration of dust in the air after blasting and during mucking of the blasted material were conducted in three headings in medium hard to hard rock. The use of the spray apparently made it possible to reduce the concentration of dust generated and thrown into the air during blasting more than 99 per cent from the amount present when the spray was not used. Dust dissemination during mucking of material sprayed several hours before mucking was begun was much less than during shoveling of material thoroughly sprinkled just before and during mucking; and, of course, the dust present after use of either the spray or the sprinkling method is

far less than if the mucking had been done dry.

In general, the larger the quantity of explosive used, the more finely divided the blasted material, the more heavily the air will be impregnated with dust and smoke, and the greater will be the quantity of poisonous gases left at the place; hence, methods should be used that will tend to reduce the quantity of explosives used and of gases and dust produced. In this connection it will be found that the use of stemming or of some types of blasting plugs now available will confine more definitely the explosive, hence, increase its efficiency and reduce the quantity of explosive used and also reduce the amount of poisonous gas produced and possibly reduce the violence of the blast and its tendency to raise dust and disseminate it into the surrounding air. It would be well for users of explosives to investigate types of explosives that are known to give off minimum quantities of harmful gases on detonation and to use them; also, the discontinuance of the use of fuse and the substitution of electric blasting would aid in reducing the amount of poisonous gases from blasting. Explosives that have been held in storage too long or that have been stored under unfavorable conditions as to moisture, temperature, etc., are likely to give off maximum quantities of harmful gases, if they detonate at all; hence, utmost care should be taken in the storing of explosives to see that they are not held too long before being used.

Personal respirator protection.—This method of control of air dustiness is unsatisfactory in underground mining except for temporary use in emergencies. Although dust respirators give adequate protection against dust, they afford the wearer essentially no protection against toxic or asphyxiating gases, and are too cumbersome and uncomfortable for constant wear.

The Bureau of Mines has established a schedule of minimum requirements for mechanical respirators, one type of which is for protection against dust generated by disintegration as in drilling and blasting. About 30 respirators have been approved under this schedule.

Physical examination.—Although strictly speaking physical examination is not a dust-control measure, it is an important means of determining the efficacy of the methods employed to reduce the dust hazard. If workers exposed to dust are examined periodically increased incidence of respiratory disease can be discovered and measures taken to reduce the amount of dust in the air or remove any affected workers to a less hazardous environment.

In South Africa all white applicants for underground work are examined and certified to be physically fit and free of any disease of the respiratory organs and then reexamined every 6 months to determine and report all cases of simple silicosis, tuberculosis with silicosis, and simple tuberculosis that may have developed. In this way comparable records can be obtained for measuring the degree of pneumoconiosis and the extent of impairment of working capacity among employees from year to year. Comparable records will not be obtained unless periodic examinations are conducted in a standardized way, preferably by a permanent medical board composed of physicians specially trained and having adequate experience in the diagnosis of pulmonary diseases.

Summary

1. The principal pulmonary diseases to which the miner is subject are bronchitis, influenza and pneumonia, pulmonary tuberculosis, anthracosilicosis, and silicosis.

2. Statistics indicate that much higher morbidity and death rates from pulmonary diseases are experienced in dusty than in nondusty industries, especially where silica dust is used or produced.

3. Investigations by the Public Health Service reveal that hard-coal miners suffer a high mortality rate from influenza and pneumonia not only during influenza epidemics but at other times as well. In 1915-23 and 1906-25 influenza and pneumonia were responsible for 40 per cent of the total deaths among hard-coal miners compared with 25 per cent among males at ages 15 to 65 who were engaged in pursuits other than coal mining. Six per cent of hard-coal miners had pulmonary tuberculosis compared with 2 per cent in the adult male population of the United States. The rate for metal miners is still higher.

4. Anthracosilicosis was found in about 23 per cent of 2,711 employees of three anthracite mining companies studied by the United States Public Health Service in 1933.

About 63 per cent of those having anthracosilicosis showed evidence of disability, compared with less than 10 per cent in the control group. Moderate and marked degrees of disability handicapped approximately 21 per cent of those with anthracosilicosis compared with less than 2 per cent of the men serving as controls.

5. The occupational groups in anthracite mining, arrayed by the Public Health Service according to extent of physical impairment, are (1) rock workers, (2) regular miners, (3) all others exposed to more than 100 million dust particles per cubic foot of air, and (4) men in haulageways except rock workers.

6. The principal pulmonary disease in metal mines is silicosis and the chief menace in silicosis is tubercle infection. Apparently silica dust alone increases the liability to pulmonary tuberculosis.

Joint studies made by the Actuarial Society of America and the Association of Life Insurance Medical Directors[19] revealed that there were 65 deaths where 6 were expected (about 11 times the expected number) from tuberculosis in 1925-1936 among a group of underground miners, most of whom were employed in metal mines. Tuberculosis deaths were fourteen times the expected among copper miners, nine times the expected among gold and silver miners, and eight and a half times the expected among iron miners. In an earlier study[20] covering the years 1915-1926, there were 18 times as many deaths as expected among lead and zinc miners.

7. As dust is the greatest contributor to the excessive mortality from pulmonary diseases in the mining industry, the principal method of preventing these diseases is to eliminate as far as possible or reduce to harmless concentrations the dust in the breathing zone of the worker.

8. The aim should be to eliminate the dust entirely from the air breathed. Where this is not always feasible it may be possible to reduce the dust to a concentration that will not cause disability during a working lifetime.

9. Analysis of data collected by the Public Health Service to determine safe limits of dust exposure indicate that employment in an atmosphere containing less than 50 million dust particles per cubic foot produces a negligible number of cases of silicosis when the quartz content of the dust is less than 5 per cent; when the silica content is about 13 per cent 10 to 15 million particles appears to be a safe limit.

10. The amount of harmful dust created by various activities in mining can be determined by sampling the atmosphere in their vicinity and counting and identifying the dust particles. Sampling of air is also a means of checking the efficiency of dust control measures.

11. The impinger is the dust-sampling instrument used most frequently in the United States.

12. The dustiest operations in mining are dry drilling, blasting of dry rock or ore, shoveling or mucking, loading and haulage, and machine coal cutting.

13. The principal measures for controlling the ill effects of dust in mining operations are ventilation, wet methods, proper blasting practice, the use of personal respiratory protection, and physical examination of workers.

14. The efficiency of these measures in the reduction of the incidence of dust diseases is attested by the experience in the mines of South Africa where among the whole body of miners the liability to contract silicosis in 1936-37 was less by 65 per cent than it was in 1920-33. Among the "new Rand miners" who entered the industry by way of initial examination since 1916 and who have been exposed solely to the progressively improving occupational conditions that have obtained since that date, the liability to contract silicosis was less by 87 per cent than the similar liability among all miners working for an equal period in 1920-23. These "new Rand miners" constituted 77 per cent of all working miners examined.

Conclusion

The mortality and morbidity from pulmonary diseases are excessive in the mining industry as a result of exposure to large amounts of dust in the air breathed. Therefore, control of the dust hazard is necessary to prevent these diseases. No single measure of control is applicable to all dusty operations and processes, therefore, all the means of prevention must be practiced to insure success in the solution of the problem.

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ADJOURNMENT