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AIR HYGIENE FOUNDATION OF AMERICA, Inc.

FIFTH ANNUAL MEETING OF MEMBERS

PITTSBURGH, PENNSYLVANIA
NOVEMBER 12-13, 1940

01100

Note to Members:

The enclosed Proceedings of the Foundation's fifth annual meeting is in the nature of a "text book" on industrial hygiene. It is doubly important now when the health of workers is as vital as health of soldiers. The Proceedings is an "annual report" to your company on what the Foundation has done for industrial health conservation during the past year. Extra copies are available. Please put this information to work, passing it through the departments it affects, as:

Top Management	Legal Department
Medical Department	Public Relations
Industrial Hygiene Engineers	Personnel

Industrial Relations

Today's wars are won in mines and mills, emphasizing the importance of employee health conservation.

January, 1941

Air Hygiene Foundation
4400 Fifth Avenue
Pittsburgh, Pa.

01101

PROCEEDINGS
OF FIFTH ANNUAL MEETING
OF
AIR HYGIENE FOUNDATION OF AMERICA, INC.
NOVEMBER 12-13, 1940

AIR HYGIENE FOUNDATION

A National Organization
for the
Advancement of Industrial Health

01103

CONTENTS

REPORT OF THE PREVENTIVE ENGINEERING COMMITTEE

PROFESSOR PHILIP DRINKER, Chairman

During the past fiscal year of the Foundation, one fellowship has been maintained at the Harvard School of Public Health. The incumbent is Bernard D. Tebbens, who is a candidate for the Doctorate of Science Degree at Harvard. He has worked on two problems, one involving a study of particle size in relation to silicosis, and the other comprising a study of the gases and visible fume evolved in arc welding with coated rods.

I. For the studies on particle size and silicosis, dust samples of finely-ground flint, 99.3% SiO_2 were prepared as follows:

3.3, 1.7, 1.0, and 0.6 microns.

As stated in our 1939 Report, the object of Mr. Tebbens' work with sized particles was to study the relationship of size to potency (with respect to silicosis). It was generally admitted that the potency increased as the size decreased but, so far as we know, no studies had been made (and certainly none published) which attempted to answer this question with pathological proof. We are indebted to Dr. R. Z. Schulz, of the Department of Pathology, Harvard Medical School, for his cooperation in this work and wish to make clear that he will be joint author with Tebbens of a paper detailing the complete study.

For each size group, nine rabbits were used and each received 400 milligrams of dust in saline suspension injected in two equal doses at three-month intervals. No animals died from the injections. One animal from each series was sacrificed at about three-month intervals.

Histological sections of the liver of animals sacrificed early in the experiments showed differences in liver damage. After the first three months, animals receiving 0.6 and 1.0 micron dusts showed small though definite evidence of scarring. Microscopically, they had small areas of necrosis and fibrosis distributed particularly in the portal areas of the liver. On the other hand animals injected with 1.7 and 3.3 micron dusts showed no gross pathological changes. On microscopic examination, only numerous macrophages and giant cells were found scattered throughout the organ.

As the experiment progressed further, gross pathology became increasingly evident in the animals which had received the two finer sizes, and even to some extent in animals having 1.7 micron dust. This pathology was particularly evident as fine wrinkling of the liver surface, merging into clearly-defined nodulation and white areas of fibrosis. A gradual enlargement of liver and spleen was also noted.

In the last group of animals, such striking gross pathology was evident that there is no doubt as to the relative importance of these dust fractions in producing tissue damages. These animals had lived about 17 months since the original injections.

The animal receiving 3.3 micron dust had slight enlargement of liver and spleen with negligible other appearance of pathology.

Those receiving 1.7 micron dust had very noticeable enlargement of the organs, and the liver was coarsely nodular with white fibrosed areas.

The 1.0 micron animals showed complete fibrosis of about ½ of the liver, the remainder being distinctly nodular. The spleen was enlarged about three or four times normal, but was otherwise not remarkable.

The 0.6 animal functioned on about ¼ of the liver, which itself was ... coarsely nodular. The remainder of liver tissue had been completely ob... erated by fibrosis. The spleen was enlarged several times and showed wi... miliary tubercules over its surface. Other organs, such as lungs and bo... marrow, showed distinct evidence of fibrosis. Kidneys of all animals ha... appeared normal throughout.

Engineers are concerned with particle size in the causation of silico... because we, and not the physicians, are responsible for dust control and f... making estimates or counts of dust concentrations. From the results ... Tebbens and Schulz, it is evident that methods and apparatus designed ... abate dustiness or to estimate dust concentrations should be directed esp... cially against the particles well below and not above 1 micron in size.

II. A part of Mr. Tebbens' fellowship work has been devoted to a study of the nature and quantity of the fumes and gases liberated during ar... welding with coated rods. Not all electric welding today is done with coate... rods, but their use is increasing rapidly. In the case of alloy steels and o... non-ferrous metals, coated rods are the rule and not the exception. In general, the primary purpose of the coating is to shield both the arc and the hot metal from reaction with constituents of the air.

Time would not permit studying the full list of the coatings used on modern welding rods or the products they give off in the arc. With the help of welding experts from the General Electric Company, who supplied all the equipment needed for this study, we selected a number of representative rods, of which the makers generally furnished the approximate analyses. In those few cases where analyses were not available, it was not difficult to determine the essential ingredients and to check them against the products of combustion in the arc. Every effort has been made not to divulge any trade or manufacturing secrets regarding the exact nature of the coatings.

A. Low-Carbon Steel Electrodes: Seven different electrodes were studied in three sizes, 5/32", ¼", and 5/16". The coatings contained the following:

Ferromanganese, the Mn serving as a deoxidizer.
Various silicates, including asbestos, with small amounts of free silica.
Sodium silicate as binder.
Titanium dioxide as an arc stabilizer.
Carbohydrates to furnish a reducing atmosphere.

B. Steel Alloys and Non-Ferrous Alloys: The coatings of these rods all contain fluorides, usually carbonates, and some silicates. The coatings of rods for welding aluminum contained chlorides, while one alloy rod, a 96 Cu 4 Si rod, contained borates.

In general, all rods and all coatings gave off a visible fume or smoke of which the qualitative composition was predictable from the analyses of the rods. In addition, we looked for any volatile fluorine compounds, as well as carbon monoxide, chlorine or hydrochloric acid, and especially oxides of nitrogen.

Welding was done in a gas chamber equipped with a special exhauster and a hood placed near the welding work. Beads were welded across small metal plates and the gases and fumes from the arc were drawn into the hood by the exhauster. Samples of gas and of the visible fume were taken from the flue pipe and analyzed. From a considerable series of such analyses, it was possible to construct a table:

FUMES AND GASES
ARC WELDING — COATED ELECTRODES
Partial Summary of Data

Electrode	Diam. inches	Power V X A	NO_2 per Electrode mg.	Fumes per Electrode gm.	Constituents of Fume Per Cent SiO_2	Fe_2O_3	MnO_2	F-	Others	
A	5/32	4500	13.2	1.3	20.0	62.3	10.8			
	1/4	9900	14.8	2.3	19.4	52.7	12.2			
	5/16	18000	43.2	5.0	22.4	--	9.1			
B	1/4	10500	18.9	1.2	33.9	28.4	10.4			
C	5/32	4200	6.4	0.9	12.7	67.1	5.2			
A (Galv.)	5/32	4500	0.9	7.3	0.2	10.7	1.7		ZnO	65.2
Bare	5/32	3200	22.0	--	--	--	--			
Stainless	5/32	4200	--	0.5	5.8	10.1	2.8	17.6	Cr_2O_3	8.3
Alum.	5/32	3125	19.4	0.2	(Al_2O_3 12.5)			26.2	Cl-	34.6
Monel	5/32	2800	23.3	1.5	1.2	3.8	6.5	9.4	NiO	1.8

If welding was done at various distances from the hood it was possible to determine at what suction velocity, measured at the arc, all visible fume and gas entered the hood. It was also possible to predict, with fair accuracy, the gas and fume concentration after a definite time when welding under particular condition. It follows, therefore, that one may write specifications for the necessary ventilation to bring about any desired condition.

In general, we believe that modern electric welding is a safe job, providing that the worker observes certain simple rules:

He must avoid ultraviolet light from the arc and his skin, especially hands, face and neck must be protected. We have no fault to find with the conventional safety practices with regard to these items.

He should not weld in confined spaces, like tanks, unless they are well ventilated, or unless he is supplied with an air mask.

If the concentration of visible fumes is kept within reasonable limits by means of appropriate ventilation, there will be no risk of poisoning from NO_2. This does not mean that NO_2 gas and visible fumes are given off in any constant ratio. They are not. But low concentrations of visible fumes, such as 20-25 mgs./cu. m., preclude any possibility of harm from breathing gaseous impurities.

We had formerly a second fellowship, held by Leslie Silverman, but this was stopped at my suggestion because Mr. Silverman was appointed Instructor in this School and had to devote a large part of his time to teaching and not to the research in which the Foundation was interested. However, his original thesis work, made possible by the Foundation's generosity, will be published.

The purpose of Mr. Silverman's work was to study the factors affecting hood design for single-slot unheated tanks, such as those used for cold-plating, pickling, acid-dipping, alkali baths, and wash tanks. The effect of temperature and of solvent characteristics on ventilation requirements, together with the performance of double-slot hoods, is being studied further.

Ventilation requirements (Q) of unheated tanks with lateral exhausts were found to vary as follows:

Unflanged hoods — $Q = 2.3\ (\text{width})^{1.15}\ (\text{length})^{0.53}\ (\text{velocity})^{1}$

Flanged hoods — $Q_f = 1.9\ (\text{width})^{1.3}\ (\text{length})^{0.7}\ (\text{velocity})^{1}$

The optimum height of flange is that equal to tank width and the optimum flange inclination is the maximum allowable without interference with industrial operations.

Slot velocity or slot width are unsatisfactory criteria of hood performance. The important factor is the quantity of air handled.

The depth of liquid surface below the slot does not affect greatly the characteristics of hood performance, but may have an important effect on evaporation and entrainment of liquid.

I am assured that one fellowship and perhaps two will be continued at Harvard, and I should favor, in this case, departing from perhaps our straight engineering and including something that is a little more biochemical, particularly a problem on how the human system handles solvent vapors when exposed to a compound such as carbon tetrachloride. That is, if an individual is exposed to a certain concentration, how soon does his blood become saturated at that concentration, and if he leaves, how soon does he

become desaturated? The fundamental data on such matters now are badly lacking.

I am also informed that our Engineering Committee next year will be able to make the survey of exhaust systems which we projected last year and which we were unfortunately prevented from making purely by lack of funds. I am informed that such funds will be available this year, and we shall go forward with that as projected in our annual report of a year ago.

The Foundation has liberally continued its support of the Journal of Industrial Hygiene and I think I can say to the mutual advantage of the Journal and of the Foundation. From my standpoint, as editor of the Journal, it is a most desirable cooperative effort. We exchange data all the time and each helps the other materially in the abstract work, thereby considerably reducing the cost of the combined effort.

There is one item in which I would bespeak your support if it should come up. In the general field of industrial solvents, particularly of common organic solvents, new ones are being introduced continually. Every now and then a lawsuit such as this will arise: An individual, unknown to himself, has a tubercular lesion. He receives an exposure to some solvent vapor or perhaps an irritant gas as from electric welding. He does not know that he has tuberculosis at the time, but a month or two or three later he breaks down and is examined by a physician and is found to be in an advanced stage of tuberculosis. Human nature being what it is, he looks about for some one on whom he can pin the blame and he picks on the employer at that time. That is perfectly natural. You have experienced such suits. We may have our opinions about those things, but to prove them is a very difficult matter. I can number in my own experience two or three such occasions. The proof of the thing is highly embarrassing, even if the firm has worked in the utmost good faith and is of the opinion that it has kept working conditions as they should be....

The answers to vexing problems like that might well be obtained by research, especially at Saranac Lake, of the type that Gardner has done so successfully with dusts, namely, the treatment of experimental animals inoculated with tuberculosis virus in accordance with Gardner's technique: treatment of animals with known concentrations of solvent vapors such as we would permit our workmen to have in their ordinary work, and then seeing whether their condition is adversely affected.

You may say that legally you cannot draw inferences from animal experimentation. I'd like to say that you can and it is being done with great success in defending unjust claims. It is the one way you are likely to be able to answer the problem with even a shadow of success.

I think that the Foundation could very well interest itself in that problem. It is one which is most vital to the engineer, because sometimes we are called upon to take responsibility for a ventilation installation which we have put in in the utmost good faith. Later we find that the individual for whom it was designed, perhaps, is a little below par, tubercular, if you wish, and he breaks down and then they say, "Well, the engineers didn't ventilate this job. If they had done it the way they should this fellow should be all right." I don't think it is our fault, but nonetheless they hunt around for blame and they pin it on us, sometimes with a good deal of success.

I note in the program that at the conclusion of what I have to say, and we have reached that point, the meeting is open for discussion. If I or colleagues in the audience can answer questions on these matters, we will be glad to do so.

DR. MILES: When you discussed the effect of these fine dust partic on the liver, in what way did you say the dust was got into the rabbi Injected?

PROFESSOR DRINKER: Yes, into the ear vein. That is an old te nique that has been used for many years in the study of toxicity of vari types of dust. Of course you can produce fibrosis from silica in various pa of the body. Putting it in in that way it can be done with precision a with quantitative accuracy. The reason we didn't dust these animals by t air-breathing route is because the samples of 0.6 micron dust were measu literally by the milligram and not by the gram and we didn't have enou dust to permit inhalation experiments.

DR. WILKIE: I'd like to ask Mr. Drinker if he did find anything in t lungs.

PROFESSOR DRINKER: Not of any consequence. They showed slig evidence, macroscopically, of fibrosis.

DR. SELBY: If it is so difficult to make this fine dust does it actua exist in industry?

PROFESSOR DRINKER: Yes. That is a very good question and a ve practical one. The trouble is that fine and coarse particles are intimate tied together, and what we wanted were the individual sharply defined fr tions. In any place like a foundry or mine, all air-floated dust will conta plenty of the 0.6 micron dust. We wanted to get it by the milligram, gram if we could, and to get it and separate it from the other sizes extraordinarily difficult. It is very tenacious. They just stick together.

DR. MILES: In your conclusion, then, while you actually examined t liver you would say that there was an analogous change in the lungs. mean it would cause fibrosis in the lungs were it breathed in.

PROFESSOR DRINKER: I am sure it would.

DR. MILES: But there would be no liver change?

PROFESSOR DRINKER: If you breathe it in, the most importa damage will be in the lungs, whereas if you take it intravenously the impor ant damage is always in the liver.

MR. BAILEY: The inference is that the dust particles larger than 1 microns are harmless?

PROFESSOR DRINKER: That is certainly what I think—not total harmless but, compared to the others, yes, they are harmless.

MR. BAILEY: Isn't that something new?

PROFESSOR DRINKER: I don't think so. I think all we have don is put a hunch that everybody had on a slightly more quantitative basis.

DR. LEROY U. GARDNER: May I say a word about this?

This subject (toxicity of fine particles) that Professor Drinker ha reported this morning is one that we have investigated also. You confir the findings that we have already made with the exception that we felt tha if you allowed particles up to three microns in diameter to act long enou they would produce definite and progressive changes in the liver. I hav forgotten how long your maximum observation was.

PROFESSOR DRINKER: A year and a half.

DR. GARDNER: We found that in a year's time the particular quartz with which we worked was active. We set three microns as the upper limit of activity. Four microns to six microns were inert. We all agree, I think, that the smaller the particle the more active it is and the more rapidly it produces changes in various organs.

The liver that Professor Drinker has used as a test organ is one that we have used also. When you come to apply this knowledge to inhalation, however, you have got to consider whether particles under a micron or a half a micron, perhaps, are going to remain in the lung in sufficient quantities. We tried some years ago an inhalation experiment with some exceedingly fine silica, whose average particle size was said to have been of the order of 20 angstroms. This material was prepared and measured by a method that I am not in a position to discuss just now. The point I want to make is that when we made animals breathe this exceedingly fine silica we didn't get what we expected, that is, almost immediate death, within a few weeks. We found that relatively little of it stayed in the lungs. There was enough there to produce some changes in the phagocytes, the formation of giant cells, and what not, but no fibrosis developed. There was not enough of the very fine silica in the lungs to cause any death of tissue. While it is perfectly true that the finer the quartz particle or the silica particle the more active it is, it seems to me that our application of this finding must also be conditioned by what can be expected to remain in the lung after inhalation.

I quite agree with you that the engineer ought to be prepared to trap the finest dust that is in the atmosphere and that he needn't be much concerned about anything over three microns, even though the lung may contain some ten micron particles. Those three to ten probably are pretty inactive. Whether it is going to be 1.5 or 3, I cannot say, but I certainly feel that it is in that range that we are going to define the upper limits of activity.

PROFESSOR DRINKER: I think that engineers feel that we trap the fine dust, but I don't think that we feel that we measure it when we go out into a plant. I think if we catch the 1's and 3's we will catch the 0.5's too. But certainly, if we make estimates of relative hazards of one job and another, we don't come anywhere near laying the emphasis on the fine material that we ought to.

In defense of my having infringed on the field which Dr. Gardner has so well covered, I should like to say that we were guided in our choice by the fact that in the experimental work which he cited. I am distinctly under the impression that he stopped at three microns. He said that the others were more toxic, but his published data, quantitatively, are on the three microns and over. That may seem like splitting hairs to you gentlemen, but it isn't to me.

MR. FRAZIER: I should like to ask if you have actually correlated the different rates between the size particles on those particular animals you have experimented with. In other words, by 0.5 on up.

PROFESSOR DRINKER: You mean correlated in a mathematical sense?

MR. FRAZIER: Yes.

PROFESSOR DRINKER: You can't. I don't think we would ever be able to. You can't do much more than get the results and just present them as you see them, either in gross preparations or histological size, and say

that one is so much worse than the other. To make any precise mathematical correlation I am afraid is beyond any skill that we have.

DR. FRARY: Does that not imply that the examination and dust counting should be done on a dark field rather than light field? I understand you can't get much under one micron on bright field. Some dusts which look very well if you look only in the bright field become very bad if you look only in the dark field.

PROFESSOR DRINKER: In defense of the present counting technique, which is light field and does pay particular attention to material that is bigger than one micron, the results of such counts, when they show high dustiness, correlate fairly well with such data as we have on incidence of silicosis and the like. After all, dust counting as it is done today is not to measure how much dust is in the air. It is to estimate what the silicosis risk is, and from the present way of looking at things the two haven't a whole lot in common.

From the standpoint of precision, I agree very warmly with Dr. Frary. From the standpoint of public health, perhaps the present counting technique will do.

DR. RUSSELL: Have the pathological effects of amorphous silica dust and diatomaceous earth been determined by experimentation?

PROFESSOR DRINKER: We haven't tried any. Dr. Gardner can answer that much better than I.

DR. GARDNER: We have tested the effect of diatomaceous earth by this method and found that it is capable of producing a progressive fibrosis that looks very much like that caused by quartz. That is the only form of amorphous silica that I know of that will produce this reaction.

It has been suggested that the reason for the greater activity of this particular form of amorphous silica is due to the tremendous surface exposed to contact with the animals. The diatom shells are pierced by tremendous numbers of exceedingly fine tubes and as these particles are brought into contact with the tissues the tubes' surface is added to that of the outer surface. All the other amorphous silicas that I know of are incapable of producing a progressive reaction. A few of them cause a little chronic inflammation, but that tends to subside rather than progress.

PROFESSOR DRINKER: A year or so ago, at the last Saranac Silicosis Symposium, Dr. King, from England, took me, among others, pretty severely to task for the unscientific way in which we made dust estimates. His language was diplomatic, but the implication was that we were somewhat stupid and that the British method of thermal precipitation, which caught very easily the fine materials of 0.5 microns, was superior to ours. Dr. King said that their technique deliberately ignored the bigger sizes, say above three, and took the finer ones, whereas the impinger did its best work beginning at about two microns and going on up to ten. He took us to task, and I think with reason, for the unscientific methods we were using. On the other hand, we can maintain with some justification that with such as we have we can control silicosis and measure the degree of our control fairly satisfactorily.

DR. MELLER: The next section of the program is in charge of Dr. Lanza, Chairman of the Medical Committee.

REPORT OF THE MEDICAL COMMITTEE

A. J. LANZA, M. D., Chairman

During the year we have succeeded in getting our studies on sick absenteeism in industry well under way. Fifteen firms are now cooperating with the Foundation and with the United States Public Health Service in putting into effect what will be a comprehensive record which will show the extent and nature of sick absenteeism in industry. The importance of this is self-evident when we consider the enormous economic loss involved. We will hear in detail of this activity from Dr. Gafafer, who is on our program today.

The Foundation has continued to sponsor research activities at the Saranac Laboratory and this very important piece of work will be described to you by Dr. Gardner, who is also on our program.

We also have continued to support the extremely interesting studies on the effects of toxic materials on the living tissue carried on at the University of Pennsylvania, which will be described to you in detail by Mr. Haagensen. As you know, the studies on x-ray which have been carried out at the University of Pennsylvania have been completed and are available to the members and to the public in bulletin form.

For the coming year we propose to carry on our studies in sick absenteeism, in cooperation with the United States Public Health Service and with those member firms who have requested participation in this undertaking. We also propose to carry on Dr. Gardner's studies at Saranac and Dr. Clark's and Mr. Haagensen's studies at the University of Pennsylvania.

I should like to discuss very briefly recent advances in the status of the industrial health program throughout the country.

The American Medical Association, through its Council on Industrial Health, is continuing its various efforts in this field. The Council not only carries on educational and informative activities at its headquarters in Chicago, but has inaugurated a broad campaign of education through the state and county medical societies all over the country, having in mind particularly the needs of small plants wherein, as you know, some eighty per cent of all our industrial wage earners are employed.

The activities of our own Air Hygiene Foundation are well described in the program of today and tomorrow, but I should like to emphasize for your attention that this Foundation is the only national association including in its membership all types of industries which is actively supporting and financing fundamental research in industrial health problems, without which continued progress in the field of industrial health is not possible.

A committee composed of Mr. Ahearn, Professor Drinker, Dr. Cranch and myself, representing the Air Hygiene Foundation, recently went to Washington and had an interview with Governor McNutt, who is at the head, as you know, of the Social Security branch of the Government. We went to see Governor McNutt because the Public Health Service, whose interest we were endeavoring to elicit, is now in that division of the Government. One of our objects in talking to Governor McNutt was to try to make the responsible people in Washington appreciate the importance of industrial health work in industry and to prevent as far as possible industrial physicians, industrial hygiene engineers, industrial safety men, and other technicians

being diverted from their activities in industry into some other form of ernmental service.

Furthermore, we also hoped that the industrial physicians, the indu hygiene engineers, the safety engineers, and others, who carry on enormously important work, which is just as essential a part of na defense as carrying a gun, would receive some credit or some recogn so that they might not feel that they had not been doing their part in general defense program.

I might say that Governor McNutt gave us a very sympathetic hea and I think some advantage may come out of our visit.

The National Association of Manufacturers is continuing its activitie stimulating and educating its member firms. This association also is pu its emphasis on the needs of small industries.

On the official side, we have the Division of Industrial Hygiene of United States Public Health Service. I don't have to talk to you about importance and far reaching activities of this division, which have been m ifested by the energy and thoroughness with which they have been inst mental in setting up divisions of industrial hygiene in some thirty s governments. Every phase of health in industry is covered in the activi of the Public Health Service.

The Department of Labor, through the Bureau of Labor Standards the Bureau of Statistics, is continuing its important activities in the fie industrial health, both through field work and educational publications.

Now comes a new factor in the field, and I am referring to the Cou on National Defense. Industrial health is a major portion of the nati defense program and is contained under the Health and Medical Comm of the Council. The Health and Medical Committee includes a sub-comm on industrial health, the importance and scope of whose activities cann overestimated. Two members of the Medical Committee of the Air Hyg Foundation are also members of this Industrial Health Committee of Council on National Defense, and one of them, Dr. Selby, is the Chairman the Industrial Medical Committee of the National Defense Council.

This committee is tied in with the National Research Council on the hand and with the military services on the other. Liaison officers have detailed to work with the committee by the National Research Council by the Surgeons General of the Army, the Navy, and the Public Hea Service.

At no time in our history has the health of the industrial work whether he may be employed in plants owned by private industry or in plan operated by the various departments of the national government, receiv so much careful consideration as at the present time. It is safe to say as a result of the national defense program and its coordinated activi industrial health and hygiene will be established on a firm and permane foundation throughout the United States.

In conclusion I wish to make just a few more comments on the ha phase of the national defense program.

Physical selection is an integral part of the program, applying to c ian as well as military forces. The present employment examinatio becoming more thorough and thereby more valuable for diagnosis and for therapeutic purposes. The importance of this, from the standpoi public health, is inestimable.

Many men with minor and even major defects must be utilized in

defense industries, both in the general interest and in their own interest. Men must work where work is available, and it is the function of industrial medicine to see to it that those who present special needs or problems secure the help and supervision that will enable them to keep on with their work. It is important that those who are concerned in the administration of private and governmental industries keep well in mind that the function of a physical selection program is not to bar men from work but to determine what kind of work they can best do and to provide adequate medical supervision while they are at work.

It is extremely important that those who are found unfitted for work—and I am thinking particularly of those who will be found to have tuberculosis and other general organic diseases—receive adequate care, care that will have for one of its objects as far as possible physical rehabilitation.

The defense program provides for our national security. As an essential part of that program physical selection and medical supervision must contribute a positive force for health that will serve to raise the general health standard of the entire industrial population. That is our goal, and we must strive toward it.

DR. LANZA: We will continue our program. The next paper will be by Dr. Gafafer, the Senior Statistician of the United States Public Health Service, who will speak on the sick absenteeism study.

DR. LANZA: The next paper is by Dr. Gardner, dealing with ai research at Saranac Laboratory.

INDIVIDUAL SUSCEPTIBILITY TO TOXIC DUSTS

DR. L. U. GARDNER*

I am going to talk to you this afternoon on one phase of the activities at the laboratory which I believe may have some practical significance. It is the one question as regards silicosis which I still believe needs further clarification.

We have a good deal of information now on this subject. If we could put that information to work in most places we would be able to control silicosis pretty effectively. However, where funds are not sufficient to institute all of the methods of control that are known, we are pretty apt to have some silicosis persisting.

For that reason I believe that it would be important for us to know whether there is such a thing as individual susceptibility to the action of silica dust, and, if it exists, whether we are able to detect it by any method. If this were true, it might be possible in the pre-employment examination to discover the individuals who are unduly susceptible to silica dust and its action and to place them in jobs where the exposure would not be as great.

There seems to be more or less evidence for thinking that reaction to silica dust, like reactions to other environmental conditions, may not be the same in every individual. It is pretty common experience that out of any group exposed to silica dust only a small proportion of that group will react. With the high silica exposures of hard rock mining, we find that pretty much the world over only about a quarter of groups, as a whole, develop demonstrable silicosis.

It is also true when we break down exposures for individual groups that we find that the longer men have worked the more of the particular group will be involved. It is also true that the higher the concentration of silica in the atmosphere, the greater percentage of the group will be involved.

Even with these reservations, we find that there are some men who have worked all their lifetimes under conditions that seem to involve pretty severe exposures and yet they have escaped. It is also true that there are some individuals who seem to be exposed to relatively slight amounts of silica dust and yet in a very short period of time, long before any of their fellow workmen, these individuals develop advanced stages of the disease.

Slide No. 1—Classical nodular silicosis in the lungs and lymph nodes of a granite cutter employed in Barre for 12 years.

Slide No. 2—No evidence of silicosis in the lung but some nodulation in the lymph nodes. This subject had worked for 28 years in the same industry with apparently equal opportunities for inhaling granite dust but no pulmonary disease developed.

Slide No. 3—A higher magnification of the lung shown in slide 2 to indicate the localization of the dust cells in the walls of the bronchioles and blood vessels.

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* Director, The Saranac Laboratory.

Slide No. 4—A still higher magnification from the same lung to demonstrate the quantities of dust in cells collected in a blood vessel wall. The shadows of such perivascular accumulations are responsible for the pattern of exaggerated linear markings seen in an x-ray film of the lungs.

Slide No. 5—The usual picture of the modified silicosis seen in iron miners who have drilled in rock formations. History of such drilling for 30 years.

Slide No. 6—Minimal silicosis, too slight to be demonstrable in ante mortem x-rays, in an iron miner employed in the same mines, at the same job, for 30 years. Most of the reaction consists of foci of pigmentation without fibrosis. Only 2 or 3 minute nodules of silicotic fibrosis are seen in this entire section.

Slide No. 7—A localized focus of tuberculosis in another part of the same lung shown in slide 6. Here the presence of preexisting tuberculous reaction has caused the retention of unusual amounts of inhaled silica and iron resulting in localized silicotic nodulation. The silica, acting on the latent infection, has caused it to progress after a time. The section demonstrates a focus of active tuberculosis which is about to break down and form a cavity. During life an x-ray picture would demonstrate only the overshadowing tuberculous changes.

Slide No. 8—Another instance of the effects of inhaling silica dust into a lung with a latent focus of tuberculosis. Occupation: sand miner for 15 years. Death from tuberculosis which progressed fairly rapidly after once it had become reactivated. Evidence of silicotic nodulation largely confined to parts of lungs about the old focus of infection.

Slide No. 9—Another section representing conditions in other parts of the lung whose apex is shown in slide No. 8. Here the changes are very largely due to tuberculosis and only a few of the smaller nodular areas are silicotic in origin. An associated lymph node reveals extensive silicotic changes. Presumably there was insufficient fine, silica dust generated in this occupation to have affected a normal subject. The individual illustrated retained effective quantities only in the region of a partially healed tuberculous focus. In this location, however, the dust was dangerous because it presumably caused a latent infection to become progressive.

Slide No. 10 shows the complete absence of silicosis in the lung of an individual exposed to dust containing calcined gypsum and quartz. There is a slight amount of pigmentation about the lymphoid tissues within the lungs but no fibrosis. In the lymph nodes at the root of the lungs are clusters of silicotic nodules. In spite of a theoretical silica hazard which caused us considerable concern in surveying the plant where the man had worked, no effect is apparent in his lungs. He has no scars of old tuberculosis and his lymphatic system has drained away such silica as he has inhaled. Only his lymph nodes show evidence of reaction and in this location the effect is harmless.

Slide No. 11 comes from the lung of a man with a history of exposure as a sandblaster for 7 years—part of the time with improper protection. He had been previously employed for 6 years in a railroad shop and before that had worked 8 years as a coal miner. An ante mortem x-ray of his chest showed no evidence of nodulation. This section, typical

of the rest of his lungs, reveals only minute areas of black pigmentation without fibrous reaction. There is no evidence of silicotic nodulation. Apparently his previous exposures had conditioned his lungs to the silica subsequently inhaled in sandblasting so that they did not react by forming fibrosis. While there had been some dyspnoea the man was well along in years and it is doubtful whether inhaled silica was the cause.

The slides just shown are examples of the absence of generalized reaction in the lungs of individuals exposed to concentrations of silica dust that are theoretically hazardous. In most cases the exposures are known to have produced silicosis in other men exposed. In these cases they have either caused no silicosis or only localized reactions about areas of infection. In the last two cases the influence of other minerals in the dust were probably responsible for inhibition of the silica, in No. 9 the effect was probably chemical inside the lungs and in No. 10 atmospheric phenomena presumably prevented silica from being inhaled.

Slide No. 12 is an example of the reverse where silicosis develops rather rapidly in only an exceptional member of a group. This young man, still alive, had worked for 5 years in several talc mines where evidence of typical silicotic nodulation is never found. He then began drilling in a hard rock mine but was not examined until 3 months later. At that time his chest film showed a well developed generalized silicotic nodulation. It is true that the drilling in hard rock might readily have produced silicosis but not after 3 months' exposure. Apparently the previous exposure to talc had conditioned his lungs so that they reacted with surprising rapidity to subsequently inhaled quartz. One might also imagine that his tissues were unduly susceptible to silica for others in his location with similar histories have not had the same experience.

There are various theoretical explanations which have been offered for these appearances. It is generally considered to be the case that these individuals who do not develop disease, in spite of their apparent exposure, have worked in parts of the plant where they do not get as much dust as their fellows. Sometimes that may be the case. Sometimes it may be true that the modifying action of other minerals associated with the silica in the atmosphere may have prevented these individuals from getting as much dust. But it is pretty hard to understand why these protective actions, about which we know a little, should be exerted on one individual and not on all members of the group.

The other possibility that we have to think of is modifying host factors. Lehmann, as you recall, proposed the hypothesis that some individuals had better noses than others. In some instances the nose would filter out a good deal more dust than another and prevent its being inhaled. He made some rather elaborate measurements of the amount of filtering action of the upper respiratory tract and thought he was able to correlate the amount of pulmonary reaction with the effectiveness of the nose as a filter.

Professor Drinker and his students made a similar study among granite cutters. They were not able to confirm these findings of Lehmann's as fully as they would like to have done.

Another possible host factor is that of coincident infection of a chronic nature. We all know that scars in the lung of old tuberculosis tend to hold and retain a good deal more dust than normal lung tissue does. It is a very

familiar experience to all pathologists to find in the top of the lungs old scars that are deeply pigmented by the soot and coal that most city dwellers inhale. It is also common experience to find variations in the amount of dust that is eliminated from the lungs by the lymphatic drainage system and deposited in these lymph nodes.

Presumably, then, infection may have two effects. It may hold the dust in the lung locally. The infection may also disturb the function of the lymphatic drainage system and prevent effective elimination.

Then there is, finally, a third general possibility. That is that the tissues of some individuals are basically more able to react to the stimulus of silica than the tissues of other individuals.

The Saranac Laboratory, during the past year, has been carrying on a more or less detailed investigation of these possibilities to find out whether any of them are capable of absolute proof. We have in the laboratory, as you know, dusting chambers where we can control pretty well the atmospheric concentration of dust to which all members of the large group of animals are exposed. We are making dust counts at very frequent intervals in different cages and in different parts of these rooms, so that we know just about what the dust exposure is. In spite of those controlled conditions we find that normal animals exposed for the same periods of time do vary in their capacity to react. This is especially true of animals with long tracheas, like the rabbit. It is not so true of the smaller rodents, like white rats and guinea pigs.

In the next slide which you will see I want to show you the variation in the reaction in rabbits after four and a half years' exposure to a concentration of one to three micron quartz particles averaging 144,000,000 per cubic foot. The amount of dust varied from time to time, but the concentration throughout the different parts of the room was essentially so similar that I don't think the differences were significant. We will see in the animals' lungs differences in reaction.

I will say that there are thirty rabbits in this experiment and these represent the variations in the picture which one sees. They all have been exposed to the same quantity of quartz under as nearly identical conditions as we could produce.

Slide No. 13—This one has a heavy generalized discrete nodulation throughout his lungs. Another on this slide has a slighter degree of the same reaction, and another has nothing that I could identify as silicotic.

Slide No. 14—We determined the amount of quartz in these animals' lungs, using x-ray diffraction, which we have been doing recently, and we found that the amount of quartz is comparable to the amount of reaction present. Here is the first animal, with the two quartz lines showing very strongly, corresponding to the heavy reaction. The second animal has a smaller amount of quartz. The third shows just a faint suggestion of the heavier quartz line. It is very, very slight in amount.

Some animals vary in their response even though the concentration of dust in the atmosphere is pretty constant. We find that the amount of variation is greater as the quantity of silica from the atmosphere decreases. We found with our mixture of calcined gypsum and quartz that not more than a quarter or perhaps a third of the animals developed any silicosis.

This type of observation seems to indicate that host factors rather than environmental factors must be responsible for this variation. In the animals

that I have just shown you the factor of infection has been excluded. I haven't discovered whether there is any variation in the structure of the upper respiratory tract in these three rabbits or others like them. It is a pretty difficult thing to do. We have tried to find out, but without much success.

We have thought of the influence of differences in the efficacy of the lymphatic drainage system of different animals. We are attacking this problem by comparing the relative amounts of silica in the lungs and lymph nodes of animals exposed for the same period of time under practically identical conditions. We have also considered the possibility that the difference in the amount of disease may be due to variations in fundamental capacity of tissues to react. I shall show a few illustrations of our work in these fields.

In Slide 15 you see an example of the effect of a healed tuberculous scar in retaining dust within the lungs. The scar, which extends from the pleura to the root of the lung, is heavily pigmented with iron dust. Similar effects have been illustrated by the cases with more active infection just shown.

I have indicated that possibly such retention is due to interference with the function of the lymphatic system. We can see in this case that the fibrosis involves the lymph nodes draining the pigmented part of the lung. Possibly the central obstruction in the node or the pressure on the delicate lymph vessels inside the lungs has prevented elimination of the inhaled dust.

These effects all have to do with chronic infections but the acute ones are also puzzling. One frequently sees very little dust in the lungs of animals dying of an acute pneumonia at the end of a dust exposure of 2 or 3 years' duration. The infection has apparently lasted only a few days and could have exerted no effect upon the dust inhaled previously. Was the infection present in latent form from the time when the animals were placed in the dust room, or did these particular individuals possess some peculiarities which prevented dust from accumulating in their lungs?

Slide No. 16 illustrates the effect of subacute bronchitis in white rats in excluding dust from the lungs. It demonstrates the effect of exposure to quartz dust in four of these animals. Two with no trace of infection present well-developed silicotic nodulation; two more with subacute purulent bronchitis show none. Apparently the secretion in the bronchi has prevented effective quantities of silica from entering their lungs. In view of such observations in animals it is possible that the chronic bronchitis of which human silicotic subjects complain may be a protective mechanism.

We have also considered the possibility of variation in innate capacity of tissues to react to silica. To make certain that the same quantity of quartz particles reached the tissues of every animal we have employed the intravenous injection technique mentioned by Professor Drinker this morning. We have substituted guinea pigs for rabbits because we could afford to use a much larger number of animals. First we determined the minimum effective dose of quartz that would produce reaction by this pathway. Then we injected 100 of these animals with a measured quantity of a suspension of carefully graded 1 to 3 micron quartz particles. All of them will be killed after one year and their spleens, livers and lymph nodes will be analyzed individually for their silica content and degree of tissue reaction.

Slide No. 17 shows the degree of variation in the amount of silicotic fibrosis observed in three of the guinea pigs used in the preliminary test to establish a proper dosage. Reaction is illustrated in the livers 8 months after the injection of 50 mg. of 1 to 3 micron quartz. One of them has barely visible foci of reaction scattered through it. The second has somewhat larger ones and in the third the nodules are very obvious. Chemical analyses indicate that these quantities of silica in all three livers are apparently adequate to have produced a maximum reaction. Confirmation of these results in a much larger series of animals would indicate that more subtle variations in capacity to react to silica are responsible.

We have tried to find out wherein these differences might lie. We have felt that possibly the diet of the animal might affect its capacity to react. It has been claimed that in the presence of a vitamin C deficiency scar tissue forms less readily than on an adequate diet. As a consequence we have put some animals on a C deficiency diet, exposed them to quartz, but we have found no difference in their capacity to react. For this reason, I think we can exclude C deficiency.

Then we tried vitamin B and its effect. We did this because some one had shown that feeding high vitamin B diets would prevent the development of cirrhosis of the liver by various irritants. We were getting cirrhosis of the liver by injecting quartz into the blood stream, as Professor Drinker showed you this morning. Feeding vitamin B did not do very much. It prevented the most widespread and destructive changes in the liver, but it did not prevent the formation of these nodules in the liver, such as you have seen here.

This work is still in progress. It looks to me now as if the variations which animals and human beings undoubtedly do manifest were pretty largely due to host mechanisms rather than environmental factors. It looks to me as if possibly the effectiveness of the upper respiratory filtering mechanism were very important in determining whether one individual got more dust than another. I also feel, however, that the effectiveness of the lymphatic drainage mechanism has to be taken into account, that old healed infections may alter the effectiveness of drainage and allow more dust to stay in the lungs.

Just what this last observation will eventually turn out to mean I don't know. We are going ahead and as soon as these hundred animals have had a year's contact with quartz we will make our analysis and draw our conclusions. Whether we will be able to apply any of this work in pre-employment examination I do not know. But it does suggest that respiratory infection, even though it is inactive, may be of importance.

I have illustrated one line of research which has been going on with your assistance at the Saranac Laboratory. There are many other subjects that I could have chosen to discuss. Perhaps some of them would have interested you more. We have work progressing in the field of asbestosis, which I think is going to be of definite significance as to the etiology of this disease.

The fourth year of this program is just about completed. There is still more to do.

We have been studying the effect of carbon arc ash of low concentration, similar to that to which motion picture booth operators may be exposed. Nothing has happened, as one might anticipate.

We have been working on the effect of inhaled glass wool. Apparently

even the finest textile glass set up as an atmospheric dust is not inhaled in appreciable concentrations and does not produce any dangerous effects upon the lungs.

As Professor Drinker indicated this morning, I believe there is a real field for investigation of the effects of various respiratory irritants upon tuberculosis. I have been so impressed myself, as probably most of you have, with the growing importance attached to tuberculosis in industry that we have proposed this year, in place of the regular silicosis symposium which we have held from time to time in Saranac Lake, to offer a symposium on this subject. There seems to be a very considerable interest in the problem and I think we can assemble a group of speakers who would make a definite contribution.

The symposium might be developed along the following lines:

An introductory review of the fundamentals of tuberculosis and its diagnosis; the routine methods of control; a discussion of the known factors influencing tuberculosis susceptibility, theoretical as well as practical.

Then a presentation of conditions in different industries where statistical evidence indicates that the tuberculosis rate is high; an analysis of those industries to discover whether industrial factors are responsible.

Finally, a discussion of the subject of control from insurance and compensation angles.

If there seems to be a sufficient interest for such a program the Saranac Laboratory probably will offer it some time during the early part of June.

DR. LANZA: We will now have a discussion of this paper. I do hope that you will ask Dr. Gardner such questions as may appeal to you.

DR. DAVIS: I should like to ask if in the development of this work there does appear to be another illustration of what Jerome Alexander has spoken of as the "optimum colloidality." He points out that a great many phenomena, when plotted against particle size, as you go down in particle size go up in intensity. As I understand the discussion this morning, you get a marked increase in the silicosis effect when you reach a particle size of three microns or less. Then away over on the other side you have practically none at all. I wonder if that might represent a method of attack or if it has already been proved or disproved.

A further factor that seemed to occur this morning is this. I wonder if there is an item of crystallinity in our silica. We know we have silica occurring in different forms. Cristobalite is the only one I happen to remember now. I was wondering if the effect had been associated with one or other or is it independent of the crystalline forms of the silica presented. Are there manifestations that that may be a suitable attack?

I personally would be greatly interested if you could outline, if possible, those materials which when added to quartz, such as quartz and asbestos which you mentioned, give silicosis reaction and some similar materials which do not give it.

I hope I haven't covered too much ground.

DR. GARDNER: The first question dealt with variations according to particle size, such as Professor Drinker discussed this morning. I think that is unquestionably true. The smaller the particle injected into the tissues the more active it becomes. As far as inhalation is concerned, presumably there is an unestablished lower limit of particle size beyond which we do not

have to be concerned, because the particles smaller than this minimum size will be exhaled again about as fast as they are inhaled. But in the inhalation experiment which I was discussing I don't believe that the matter of particle size was responsible, because we have made size frequency determinations of the dust in the atmosphere in these rooms and we find a sufficient quantity of fine particles at all levels in our dust house to produce disease. The cages are not over five feet above the floor in any place and the lowest one is about eighteen inches above the floor. Furthermore, the animals are moved around from time to time, so that they have exposures at different levels. I therefore feel pretty strongly that it is a difference in host factors that is responsible for the variation in the animals' lungs that you saw rather than any difference in the atmosphere or in any dust in the atmosphere.

All this dust has been ground first and all of it is agitated in a hopper. The room is not over eight feet square and eight feet high. The chances for variation in atmospheric concentration are pretty slight.

Does that answer that phase of the question?

DR. DAVIS: Yes.

DR. GARDNER: Would you mind repeating your second question?

DR. DAVIS: Crystallinity.

DR. GARDNER: We have tried a great variety of free silicas to see what their effect might be by injection techniques that already have been discussed. We have found that normal quartz, the cryptocrystalline free silicas and the vitreous silicas, cristobalite and the other inversion form, tridymite, are all equally active; that of the amorphous silicas only diatomaceous earth produces a silicotic type of reaction. Opal, which is now reclassified as cryptocrystalline rather than amorphous, as they used to tell us, will produce a moderately severe type of reaction when ground. All of these tests were made with materials ground to one to three micron particle size and all injected in the same dosage.

As far as I can see any inversion forms of quartz are just as active as the normal quartz itself.

Then you had one other question.

DR. DAVIS: Are any of the silicates—

DR. GARDNER: None of the silicates, by the injection tests, have given a significant degree of reaction. A few of the clay minerals will produce a chronic inflammation but this is not generally a severe one and does not progress to the formation of scar tissue. I think there is, however, a very real and urgent excuse for doing an inhalation experiment upon the clays, because clays have such a varied industrial use and because so many roentgenograms come to us of men who have been exposed to clays either in refractories industries or in potteries. These roentgenograms show shadows. The roentgenologist is quite sure that the industrial exposure is responsible. There have been no autopsies reported on human beings exposed to clay alone. We don't know the basis of these shadows. In many instances the exposure has been not only to the clay but to flint and various other forms of free silica. We may be seeing the effects of combined exposures to quartz and clays.

I am quite sure that were it possible to do inhalation experiments with pure clays, both of the refractory group and those used in the potteries, we

would have some valuable information. I think probably what would h pen, as the result of our experience with injections, would be that we w see in the lungs evidences of slight amounts of chronic inflammation ; that these chronic inflammatory changes would not be associated with d nite fibrosis; they would not give rise to conditions which were disabli However, this needs verification and needs proof. The only way I know getting it is to do it by inhalation rather than by injection.

Of course we do have too the effects of mixtures of the substanc mixtures of free silica with other materials, like clays and many oth things, that will modify the picture. I was talking at lunch this noon to group interested in iron oxide and recounted an experience which we had h We injected one group of animals with a mixture of equal parts of hemat and quartz ground together dry in a ball mill. Injection of a saline suspe sion of this material caused no reaction whatsoever. In the second expe ment we ground the two substances separately, then mixed the suspensio of particles together and injected the same quantities. We now got progressive silicosis.

The same amount of quartz was there in both instances, but the meth of treatment in the first case had neutralized the quartz. It had simp plastered the surface of every quartz particle with a thin layer of iro oxide, which had prevented reaction.

It is obvious that effects like this may occur in the process of grindi in industry and that inactivation of quartz does occur, so that the story isn as simple as just having a given amount of quartz particles of a given siz and a host exposed to it.

MR. ROUTSONG: I was very much interested in the slide which had t do with the sand blaster's lungs and his previous industrial history. Yo said that the previous history had conditioned the lung to resist silicosi Could you expand that a little for us?

DR. GARDNER: I wish I could. I was trying to cover my ignoranc with a phrase. I don't know what happened. All I know is that that lun had, by chemical analysis, a very appreciable and what I felt was significan amount of quartz in it but there was no evidence of specific reaction t that quartz. Now it might be that through the mechanism of the body cel the coal dust became applied to the surface of the quartz grains and th quartz no longer reacted.

Years ago Dr. Haldane called attention to the same thing. He had see a few cases where men were exposed first to coal dust and then to quartz and nothing happened. He thought that the effect was due to stimulatio of phagocytes which carried the quartz out of the lung, because the coal had stirred up these dust cells to carry the stuff out. The effect isn't as simple as that, because we know the quartz is in this lung. Chemical analysis shows it is there, and yet no reaction has developed.

It may be that we have something taking place in the body comparable to that coating of the quartz that I just described by mechanical grindin We know that the body tends to bring inhibitory substances into the same locations as the quartz, which prevents irritation. We have done quite a bit of work, which I have discussed here before, with aluminum hydroxide as inhibitor. We can put quartz in the body by one pathway and aluminum hydroxide by another and, provided the pathways are such that the two substances get into the same organ, inhibition results in no silicosis what-

soever. I presume that something like that has conditioned this sand blaster's lung.

MR. KUMLER: Dr. Gardner, do we have to change our counting technique in the case of silica in order to more accurately estimate these three to one-half micron sizes?

DR. GARDNER: I doubt whether we will. As Professor Drinker said this morning, our method of control seems to work. Probably it is sufficiently accurate to establish what we need to know about dust in the atmosphere. If these very fine particles which can only be seen by dark field or some other technique are actually retained in the lung the scientist would like to know how many of them there are there, but the engineer probably doesn't need to know because his present rules of thumb seem to take care of the situation. We know, as a general rule, that rather constant ratios exist between dark field and light field counts. Generally the dark field count is three to four times that of the light field count. We still use dark field counts for our experimental work and in some other places. If we find a disturbance in that ratio and there are many times more dark field particles than those observed by light field, we think there is sanitary significance to the variation. But I doubt whether it is necessary to change.

DR. HAYTHORN: I should like to add one comment. I have seen a number—I wouldn't say a great number, but I have seen several lungs from the coal mines around Pittsburgh which show practically the same picture that we have just discussed. That is, there is plenty of pigment there—coal pigment, I think, largely. We incinerate the sections and put them under the petrographic microscope. There are plenty of silica crystals there and yet there is no nodulation in the lung.

That has caused me to wonder what is the difference—the actual difference—between silicosis that is prominent, as Dr. Lanza himself has shown in his surveys, in the anthracite mines in the east and the bituminous mines that we have in this district. We do get the silica inhaled and we do get the carbon there in large quantities. While I have seen more of it since they have been sending in a good many lungs on account of compensation than I did previously, the actual silica nodulation is not common in this district in coal miners' lungs, as compared to those that are reported from the eastern part of the state.

On the other hand, we just had a sand blaster's lungs, within the last two weeks, that is as typical a case of silicosis as I have seen. There is something in connection with that soft coal dust-inhalation and the presence of silicosis in the lungs and its effect.

DR. GARDNER: I suspect, Dr. Haythorn, that some day we will have a method of determining the physical relationship of the silica to the other minerals and perhaps we will find a way to discover whether coating effects actually exist or not. I really believe that is the explanation. How it comes about I don't know.

DR. LANZA: Are there any more questions?

Thank you very much, Dr. Gardner, for your paper and your discussion.

The next paper is the account of the research that has been carried on in the University of Pennsylvania, by Mr. Haagensen.

FURTHER DEVELOPMENTS OF THE OBSERVATION OF THE REACTION OF LIVING TISSUE TO SILICA GRANULES IN THE LIVING MAMMAL

ELIOT R. CLARK, M. D.* and DARROW E. HAAGENSEN, M. S.*

During the past year additional work on the observation of the reaction of living tissue to silica granules in the Sandison-Clark rabbit ear chamber has been carried out along two lines.

First—A more detailed study of cellular reactions has been recorded, particularly with respect to macrophages and giant cells.

Second—An additional sample of silica obtained through the courtesy of Dr. L. U. Gardner, Director of the Saranac Laboratory, has been used. This sample was 100% SiO_2 of particle size 3μ or less in diameter.

The results may be illustrated by a number of slides considered to be representative of the large number of photographic records obtained in the past 12 months.

Series A

This series of slides at a low magnification illustrates two points:

Slide 1—The vascular pattern was growing downward. This exposure was taken immediately prior to insertion of the silica particles in the chamber.

Slide 2—Four days later the blood vessels and the connective tissue have grown downward leaving one of the silica fields in a vascularized area.

Slide 3—Nine days after the insertion of the silica particles the blood vessel pattern had started to grow through the large central field of silica. This field of silica had been inserted purposely in a lower region containing only extravasated material, the result of the original insertion of the chamber in the ear.

Slide 4—On the 18th day, the large field was above the growing edge. However, as the first point, the growth of blood vessels and connective tissue had not been influenced by the presence of the silica in this central field. If the silica particles had through some mechanism caused rapid connective tissue growth the growing edge would have been uneven in the central region. This was not the case.

Slide 5—On the 65th day the chamber connective tissue bed had a clear space because of the retraction of the tissue near the growing edge.

Slide 6—On the 93rd day this space was completely filled in by the production of connective tissue fibers through the activity of the fibroblasts normally found in the living tissue. For the second point of interest, while the rapid tissue growth took place to fill in the space, there was no indication of unusual connective tissue growth in the region of the large central silica field during the same period of time.

Series B

However, this series of slides at a much higher magnification illustrates tissue response to a relatively small field of silica.

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Slide 7—In this slide the particles were seen 7 days after placement in the chamber. They were widely dispersed, mostly within the phagocytic cells.

Slide 8—On the 24th day the grouping effect of these silica-laden cells was seen. This point has been checked a number of times in other chambers.

Slide 9—On the 96th day the field of cells was dispersed. The connective tissue pattern was not influenced by the presence of the silica field and there was no indication of a concentric arrangement of connective tissue fibers.

Series C

Slide 10—This series illustrates the tissue reaction with a larger field of silica in the same chamber. In this slide the field was on the 7th day fairly evenly distributed.

Slide 11—On the 45th day the field was in the stage of aggregation of the living cells with their contained silica.

Slide 12—On the 95th day the field was again in the dispersed phase. There was no indication of a specific response on the part of the fibroblasts.

Series D

For contrast this series illustrates the tissue reaction to a comparatively very large field of silica particles varying widely in size, however, of which 87.5% were under 6μ in diameter.

Slide 13—Fifty days after insertion the connective tissue bed along the periphery of the field was uniform from left to right.

Slide 14—On the 71st day the field appears relatively stable.

Slide 15—On the 105th day the connective tissue formation in this region was not greatly changed. The silica field was dispersed.

Slide 16—The same day another exposure in a region free of silica was taken. The connective tissue differentiation from left to right appeared similar to the field of the previous slide.

Series E

Slide 17—For purposes of comparison 2 slides are shown of the reaction which followed the use of the 2 silica samples. This slide from a chamber in which silica sample A was used was taken 151 days after insertion of the silica.

Slide 18—This slide from a chamber in which we used silica sample B obtained from Dr. Gardner indicates the tissue response 153 days after insertion.

Series F

For a period of 8 weeks the movements of a large multi-nucleated giant cell containing a number of small silica particles were recorded.

Slide 19—On the 46th day the cell was rather faintly visible with the previously ingested small silica particles.

Slide 20—On the 71st day the cell was larger in diameter and the silica particles were more clearly defined. The cell had moved upward over a large venule.

Slide 21—On the 82nd day the cell showed signs of degeneration. The boundary was indistinct and a number of macrophages were sticking to the periphery of the cell.

Slide 22—On the 108th day the cell was obliterated. The silica particles which had been ingested 3 weeks previously were then either deposited on the connective tissue bed, or taken up by phagocytic action of macrophages. There is no evidence of any irritating residue left by the degeneration of the giant cell.

Motion Pictures

Several references in the literature on silicosis indicate the sticking of leucocytes to the endothelial cells lining the blood vessels in regions where silica particles are present. The film about to be shown was taken of a silica field in the living tissue of a rabbit ear chamber. The incorrectness of that conclusion reached from dead tissue studies of rabbit ears is evident from the film. The silica had been placed in the chamber several weeks previously. The last part of the film was taken of another chamber by Dr. Clark. This illustrates sticking of leucocytes caused by artificial means and not by the presence of silica particles.

Conclusions

It may be concluded from observations for a period as long as 370 days following a single insertion of silica granules in a series of rabbit ear chambers that the following points are evident:

The endothelium of the very extensive complete vascular system found in various chambers does not show increased growth or differentiation into epithelioid cells.

The granular leucocytes do not show a specific response to the silica particles.

Neither the lymphatic capillaries nor their contained cells show a specific response to the presence of the silica particles in the living connective tissue.

Visible reaction to the silica has been almost exclusively confined to macrophages. As described last year, these cells take in or phagocytize the silica granules in varying numbers, and retain them for weeks or months. They become very sluggish in their movements and show a pronounced tendency to form permanent groups in the tissue spaces. Such groups have been followed for nearly a year without showing any significant changes.

During the first few weeks, before grouping has occurred, and in lesser degrees in later stages, there are seen many giant cells. They do not develop until after the macrophages have taken up most of the silica, and are probably formed from macrophages, either by coalescence or by nuclear division, as W. H. and M. R. Lewis have described. While they may contain large numbers of silica granules, the evidence indicates that these granules have been taken up by the individual macrophages, and not by the giant cells themselves. Giant cells may persist for days or weeks, during which time they may manifest a very slow amoeboid movement. They may then disintegrate, the remains being taken up by new macrophages, or the silica granules may be left in the tissue or incorporated in connective tissue fibers. Following the disintegration of giant cells there is no visible evidence of a deposit of any irritating residue.

It should be pointed out that giant cells are invariably observed in the

chambers in the rabbit's ear whether or not silica granules are present. It should also be mentioned that the supply of macrophages is probably inexhaustible, since they undoubtedly come from the large mononuclear leucocytes of the blood stream whose number remains constant normally, with replenishment to excess following depletion.

The pattern of the connective tissue growth in the neighborhood of the silica injections shows no noticeable difference from that in other regions, or in chambers without silica. Whether the groups of silica-containing cells are large or small, nothing has been seen, either in our living specimens or in those of our preparations which have been fixed and stained, which indicates even a tendency toward the appearance of the typical silicotic nodule.

DR. LANZA: This very interesting paper will now be discussed by Dr. Eliot R. Clark.

DR. CLARK: The results of these experiments are not considered by us to be necessarily in conflict with the results of the injection experiments of the many investigators who have described the formation, in rabbits and guinea pigs, following intravenous (Gardner, Simpson and Strachan), intraperitoneal (Sayers and Miller) or subcutaneous (Fallon and Banting) injections of silica* of characteristic nodules closely resembling the silicotic nodules found in the lungs in cases of silicosis; and who failed to find nodules of similar character following injections of many other types of granules.

Rather it seems to us that the absence of the typical reaction in the particular set-up of our experiments furnishes an unusual opportunity to investigate the various possible accessory factor or factors which apparently must be added to the simple long-time presence in the tissue of silica granules, in order to produce typical silicotic nodule formation.

That the complete explanation has not yet been found is evidenced by the variety of unproven hypotheses which have been proposed. These include among others: (1) the possible slow dissolving of the silica with production of colloidal silica which may have an effect upon the tissues; (2) some sort of slowly developed direct toxic action of the silica which eventually injures the cell; (3) the production by the cell of a phospho-lipid which becomes the irritating substance; (4) the suggestion that the silicotic nodule is produced not in reaction to silica alone but to silica along with something else: living bacteria such as tubercle bacilli or their products, gaseous substances, or various other chemical substances. Each of these hypotheses is held by one or more active investigators or group of active investigators at the present time. All of them are in need of crucial testing.

In anticipation of the desirability of having available an arrangement by which, in addition to having access to the living tissue for the insertion of granular substances, we might have a container, open to the tissues, we have developed a combination chamber which makes this possible. Without mechanical disturbance to the tissue, we can place in the container any desired chemical substance, can leave it, as long as desired, in contact with the tissue, and can then recover the contents of the chamber for chemical analysis. The device, which is an adaptation of one in successful use for some years in our laboratory in the hands of Dr. Abell, solves the problem of a way to subject tissues to the prolonged action of fluid substances, which

* (Fallon and Banting describe nodules formed in the ears of rabbits six months after single subcutaneous injections of silica, and caution against the use of too large a dose—in which case the granular masses seem to act only as foreign bodies.)

cannot be achieved satisfactorily by subcutaneous, intravenous or intraperitoneal injections.

With this new chamber we can, for example, subject to a new test the question of the reactions of living cells and tissues in the living animal to a combination of long-retained silica granules and freshly introduced colloidal or dissolved silica; we can study the reactions to phospho-lipids; we can observe the effects, on the cells with long-retained silica granules, of living or dead tubercle bacilli or their extracts—to mention only a few of the possibilities. A rabbit with this new chamber installed is on demonstration at this meeting.

There is reason to hope and to expect that, building on the foundation already laid in the studies which have been reported today and at last year's meeting, we may be able to make further contributions toward an understanding of some of the puzzling problems presented by the reactions of living tissues to silica dust.

DR. LANZA: Is there any discussion now or any question on either of these two authors' remarks?

DR. GARDNER: I show this slide to illustrate different stages in the reaction to direct injection of larger quantities of 1 to 3 micron quartz particles. The early phase consists of a tubercle-like collection of macrophages or dust cells filled with quartz of the same size used by Dr. Clark. After about 2 or 3 weeks the center of this mass breaks down with the liberation of fatty material demonstrable by special stains. After 6 months the center of the area has become soft and semi-fluid so that it has fallen out in preparation of the section. Surrounding it, however, is a definite layer of more or less concentrically arranged fibroblasts (connective tissue cells). One year after exposure the whole area has been replaced by a mass of silicotic fibrosis, so dense that the tissue is torn in preparing the section.

I cannot remember the exact dosage in our experiments but they were of the order of 0.1 cc. of a 1 per cent suspension, which means an enormous number of particles of the size employed.

I believe that the fact that so many observers have obtained silicotic reactions on injecting excessive amounts of silica directly into the tissues while Dr. Clark has failed to do so with smaller quantities indicates an indirect mechanism as the cause of the injury. Fallon proposed the hypothesis to which Dr. Clark has referred, i. e., that in silicosis the primary irritant was silica. This substance acting within the phagocyte poisoned it, liberating non-specific phospho-lipids. Phagocytosed tubercle bacilli might have a similar effect and likewise liberate phospho-lipid. It was the latter substance, non-specific in character, which then acted upon the connective tissues to produce the nodular type of reaction which characterizes both silicosis and tuberculosis.

We are testing this hypothesis at the present time. Dr. Clark has an exceptional opportunity in the method that he has developed to study the further stages of the reaction to silica and to test the Fallon hypothesis. I shall watch with great interest the further developments of his work.

DR. HAYTHORN: I should like to produce at this time an unpremeditated hypothesis.

Repair and reparative processes, and I think that the nodule of silica is a reparative process, depend on some injury or destruction of the circulation. Dr. Gardner's dosage, aside from considering the quantity of silica,

was large enough to injure the local circulation. We can see from the doses used in the ear, in these motion pictures, that the circulation was still intact. It might not amount to anything, but I should think there was a chance that those, when introduced, should injure some of the vessels.

DR. GARDNER: I think, Dr. Haythorn, that you are undoubtedly right. I think that there is an effect on the circulation, but I think it is a specific one, because the same dose of the same size particles of aluminum oxide, for example, will not have this effect. Nor will the nodule be formed if you coat your quartz particles with aluminum hydroxide or with iron or with any of the other known antidotes. I think the injury is the result of soluble silica continuously deposited on the surface of the quartz grain and that that injury manifests itself on the cytoplasm of the phagocytic cells, dust cells.

I quite agree with you that there is an effect on the circulation, because we see the vessels swell out with these big doses.

DR. HAYTHORN: On the other hand, if we make a cut with a razor that destroys the circulation, we will get a scar at that point.

DR. GARDNER: Surely.

DR. CLARK: I should like to say in connection with Dr. Gardner's discussion that we have been very much disappointed that we didn't have nice silicosis nodules develop with our method. We thought that here was a chance to watch the development of the nodules, perhaps add something to the mechanism. Then if we could get them produced we would have a chance to experiment with substances that might prevent the development of the nodules.

I think that we should go on and use more massive doses, although, as you saw from some of the pictures, for the amount of tissue involved some of our doses were pretty large. I still think that the fact that silica granules, in the doses that we used, can remain in this environment for months without producing any specific response does furnish a basis for a series of experiments in which we may see what it is when added to that silica—maybe it is more silica that may be the answer—that does lead to the result of this specific reaction in the connective tissues.

DR. MELLER: This is where we have planned to stop for the day. I want to call your attention to the symposium on industrial health defense scheduled for tomorrow, which will be intensely practical. I am sure none of you will want to miss it.

The meeting is adjourned until tomorrow morning.

WEDNESDAY SESSION

NOVEMBER 13, 1940

The Wednesday meeting of Air Hygiene Foundation of America, Inc., was called to order at 10:00 A. M. by Dr. H. B. Meller.

DR. MELLER: Mr. Waters is detained in court this morning so we will ask Mr. J. Dewey Dorsett to be kind enough to read Mr. Waters' paper.

REVIEW OF RECENT OCCUPATIONAL DISEASE LEGISLATION

THEODORE C. WATERS*

The maintenance of the health of workers engaged in industry has become an essential part of the program for national defense and, during the past year, there has been extensive activity on the part of Federal, State and private agencies to perfect the methods for health control. These programs have created new responsibilities for employers, and I have been requested to submit, on behalf of the Foundation's Legal Committee, a report dealing with current developments relating to the legal liability of employers for health injuries sustained by employees. For this purpose I have selected the following subjects for discussion:

I. State Legislation
   1. Occupational Disease Compensation Acts introduced in State Legislatures in 1940.
   2. Legislative Resolutions authorizing the appointment of Recess Committees to study the problems of occupational disease, and submit reports to the 1941 State Legislatures.
   3. Activities of State Bureaus of Industrial Hygiene.

II. Important Court decisions construing compensation statutes.

I. STATE LEGISLATION

1

Occupational Disease Compensation Acts Introduced in State Legislatures in 1940

No additional States, during 1940, enacted new occupational disease compensation statutes and, therefore, there are now twenty-four[1] States and the

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* Member Legal Committee; Chairman, Maryland Occupational Diseases Commission.

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1. Arkansas-1939
   California-1913
   Connecticut-1927
   Delaware-1917
   Idaho-1917
   Illinois-1926
   Indiana-1917
   Kentucky-1914
   Maryland-1902
   Massachusetts-1912
   Michigan-1912
   Missouri-1931
   Minnesota-1913
   Nebraska-1913
   New Jersey-1911
   New York-1920
   North Carolina-1935
   North Dakota-1925
   Ohio-1921
   Pennsylvania-1915
   Rhode Island-1936
   Washington-1911
   West Virginia-1935
   Wisconsin-1911
2. District of Columbia-1928

District of Columbia where occupational diseases are specifically compensable by statute. The State of Arkansas, included in this number, enacted its occupational disease law in 1939, but this law will not become effective until approved by referendum at the time of the general election this fall.*

During the past year, bills that would have made occupational disease injuries compensable were introduced into the State Legislatures of Louisiana and Virginia, but failed of enactment.

In New Jersey, several bills were offered to make the pneumoconioses compensable, but up to the present time none of these bills have been favorably acted upon by the Committees to which they were referred.

New York State enacted an important amendment to the section of the statute providing compensation for the dust diseases. Prior to the 1940 amendment, compensation for partial disability from these diseases was denied, and a limitation of monetary liability of an employer was established in the amount of $500.00, accounting from June 1, 1936, with progressive increases at the rate of $50.00 per month until a maximum of $3,000 was reached. That maximum would have been reached in August of 1940, but this amendment increased the maximum limitation of liability to $5,000, the progressive increases at the rate of $50.00 per month continuing until the maximum is reached in December, 1942. This amendment also provides for the appointment by the Industrial Commission of a board of expert consultants on dust diseases, consisting of three members, who are required to examine claimants in all controverted claims. The findings of this board of expert consultants are then filed with the Commission and become presumptive evidence upon the hearing of the claim.

During the coming year, the Legislatures of all the States will be in session, except those of Alabama, Kentucky, Louisiana, Mississippi and Virginia, and it is highly probable that bills will be offered to make occupational diseases compensable in some of the States where they are not at present compensable, while in other States efforts will be made to liberalize the laws and extend the benefits of compensation thereunder.

I would suggest, therefore, that the members of the Foundation follow carefully these legislative programs in the respective States in which they are engaged in business.

2

Legislative Resolutions Authorizing the Appointment of Recess Committees to Study the Problems of Occupational Disease, and Submit Reports to the 1941 State Legislatures

During the year 1940, legislative resolutions authorizing the appointment of Recess Committees for the purpose of studying the compensation laws and recommending revisions thereof have been passed in the following States:

Louisiana	Oregon
Michigan	Tennessee
New Hampshire	Texas
Utah	

In Louisiana, House Resolution No. 614 and Senate Resolution No. 387 provide for the appointment of a Commission to study the workmen's compensation law and to draft a new Act for that State. To date this Commis-

* This law was approved in the November, 1940 election.

sion does not seem to have been active nor to have prepared any report.

In Michigan, House Resolution No. 52 and Senate Resolution No. 38 provide for the appointment of a Committee to study the Workmen's Compensation Law. No personnel was appointed, and the Standing Committees terminated upon the adjournment of the Legislature. The State Bar Association of Michigan appointed a Committee upon Legislation and Law Reform to study the proposed revision of the Michigan Workmen's Compensation Law. This Committee has been active and, while its final report has not been submitted, it has prepared and submitted a progress report which includes the following reference to occupational diseases:

> "Your Sub-Committee has under consideration and has tentatively agreed upon the following general proposition, detailed recommendations as to which will be included in our final report: . .
>
> "We believe the law should cover accidental injury which may be definitely located as to time when and place where the accident occurred, as well as occupational disease clearly due to the nature of the employment. We recommend that the schedule of occupational diseases be eliminated from the statute and that the term 'injury' or 'occupational injury' be clearly defined by statute in such manner as to include accidental injury as indicated above and occupational disease peculiar to and resulting from the employment. Such definition should clearly exclude disabilities common to members of the public generally, except where it is clearly aggravated by the employment."

In New Hampshire, Joint Resolution No. 64 provides for the appointment of a Commission of nine men to study occupational diseases. This Commission has been appointed by the Governor but to date has not prepared its report.

In Oregon, House Joint Resolution No. 13 provides for the appointment of a Committee of five members to study the Workmen's Compensation Law and the advisability of including occupational diseases under the act. This Committee is now actively engaged, but to date has not prepared its proposed report.

In Tennessee, Joint Resolution No. 42 provides for the appointment of a Committee to study the Workmen's Compensation Law and presumably to consider the recommendation for compensating occupational disease injuries. A Commission of three men has been appointed recently by the Governor.*

In Texas, House and Senate Resolution No. 256 provides for the appointment of a Committee of five members to investigate the operation of the Workmen's Compensation law. I am advised that this Committee has not been active and probably will not prepare or submit a report because of the lack of an appropriation to carry out its work.

In Utah, Senate Resolution No. 249 directed the State Board of Health, in cooperation with the United States Public Health Service and the Industrial Commission to make a survey of industry in Utah, and to determine the nature and extent of the occupational disease problem in that State. It is

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* The Governor's appointees are as follows:
Dr. Roy Garrison, President of Peabody College of Nashville.
Lee Loring, Labor Leader of Memphis.
W. T. Kennerley, a Knoxville attorney.

assumed that the State Board of Health will submit to the proper Legislative Committees a report dealing with this problem at the 1941 session.

3

Activities of State Bureaus of Industrial Hygiene

State Industrial Hygiene Units have been established and are now active in thirty-one States. To this report is appended a list of these units with the names and addresses of their directors.

During the past year there have been many instances where the services of these units have been made available to industry for the purpose of assisting in the control of specific occupational disease problems. The utilization of these services should be of material assistance to small industries without medical or engineering departments qualified to advise them in the methods of control of disease hazards.

The Divisions of Industrial Hygiene in several of the States, during 1940, have promulgated Codes and Rules for the protection of employees from occupational disease injuries. In New York State the State Department of Labor has submitted to the Board of Standards and Appeals for approval three proposed industrial codes relating to the following subjects:

1. Control of dust, fumes and gases in foundries.
2. Control of silica dust in the stone crushing industry.
3. Control of silica dust in the stone cutting and stone finishing industry.

These Codes were prepared pursuant to Section 29 of the New York Labor Law and include standards of dust concentrations permissible in the given industries. Public hearings upon the Codes were held during the past summer and closed on August 1, 1940. The Codes are now before the Board of Standards and Appeals for final action and, upon their approval, will have the force and effect of law. The New York State Department of Labor is now giving consideration to five additional Codes as follows:

1. A Code on temperature, humidity and ventilation. It is believed that this Code will be very narrow in its scope and will apply solely to underground operations.
2. Revision of Code No. 12 on dust, fumes and gases. This matter is being studied by the Department and additional data is being obtained to lay the groundwork for the new Code.
3. A Code to control dust in the Ceramics industry. The basic data is now being procured for the preparation of this Code.
4. Code on respiratory protection. The need for the approval of respiratory equipment has come to be recognized and the use of such equipment may be approved and recommended in certain types of industrial operations
5. Carbon Monoxide Code. The proposed study of this subject is being planned by the Department.

No Committees have as yet been appointed to draft these Codes, and the present departmental activity is confined to accumulating information relating to these subjects for the purpose of submitting such information to the Code Committees when appointed.

In Maryland the State Department of Health and the Department of

Health of Baltimore City have recently promulgated rules relating to permissible concentrations of vapors, fumes and gases hazardous to the health of employees. These rules have been advertised for public hearing on November 25th, after which they will be adopted or amended.* The proposed rules are patterned upon the Connecticut Code for similar control.

II. IMPORTANT COURT DECISIONS CONSTRUING COMPENSATION STATUTES

During the past year there have been many interesting Court decisions relating to the legal liability of an employer for health injuries sustained by employees in the course of employment. Some of these decisions have construed the Workmen's Compensation Acts to cover what appear to be occupational disease injuries under the theory that such injuries are accidents; others construe general coverage occupational disease compensation statutes as purporting to provide compensation for many of the human ills to which we may all be subject, unrelated to our employment or the industrial processes in which we may be engaged; while others relate to the establishment of the date from which the period of limitations runs for the filing of claims for occupational disease injuries under the compensation statutes.

In the time given to me it is impossible to discuss many of these decisions or to discuss even a few of them in detail; however, I believe they are so indicative of judicial trends in construing an employer's liability for such injuries, that I would like to call your attention to those that to me seem most important.

The case of Neill McRae v. Unemployment Compensation Commission of North Carolina, (1940), 9 S. E. (2d) 595, was a workmen's compensation

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* The following rules and regulations were approved at the hearing November 25 to apply both to the State and City:

Regulation 1. General. No person, firm, corporation or other employer shall use or permit to be used in the conduct of his business, manufacturing establishment or other place of employment, any process, material or method of working known to have an adverse effect on health, unless arrangements have been made to maintain the occupational environment in such a manner that injury to health shall not result.

Regulation 2. Threshold limits. Exposures to dusts, fumes, mists, vapors, gases, or any materials that may affect health shall be kept below the following concentration limits, as determined by accepted procedure:

Material	Concentrations
Benzene (Benzol)	75 parts per million
Carbon Tetrachloride	100 parts per million
Carbon Monoxide	100 parts per million
Chlorine	1 part per million
Chromic Acid	0.1 mg. per cubic meter
Formaldehyde	20 parts per million
Gasoline	1000 parts per million
Hydrogen Cyanide	20 parts per million
Hydrogen Chloride	10 parts per million
Hydrogen Fluoride	3 parts per million
Hydrogen Sulfide	20 parts per million
Lead	0.15 mgs. per cubic meter
Methanol	100 parts per million
Nitrogen Oxides	10 parts per million
Phosgene	1 part per million
Sulfur Dioxide	10 parts per million

This regulation, as it relates to Chromic Acid, shall be construed to apply only to that branch of industry concerned with Chromium plating.

* Exposure to other materials not included in the above shall be kept below injurious concentrations.

Dusts of mineralogical composition and organic dusts shall not exceed concentration limits which shall be stipulated, depending on the nature of the dust.

claim for tuberculosis alleged to have been contracted as the result of working with a fellow employee suffering from this disease. The claimant was employed by the State Unemployment Compensation Commission, and evidence was offered to show that its offices were crowded and that, during February, 1939, the claimant, with a fellow employee named Tyson, were placed across from each other at a very narrow table to do their work; that on or about February 15, 1939, Tyson, who was suffering from active pulmonary tuberculosis, coughed into the face of the claimant on several occasions; that, thereafter, the claimant, who had previously been strong and healthy, commenced to suffer bodily fatigue and general debility; and that, on June 5, 1939, upon physical examination, his case was diagnosed as being pulmonary tuberculosis. An award in favor of the claimant was made by the Commission on the theory that the injury so sustained was an accident within the meaning of the North Carolina Workmen's Compensation Act.

Upon appeal, the Court sustained the award, holding that the tuberculosis suffered by the claimant was directly attributable to his infection when his fellow employee involuntarily and unexpectedly coughed into the claimant's face, such coughing being "untoward, unfortunate and unusual in its proximity to and its effect upon the plaintiff," thus constituting an accident within the meaning of the compensation statute.

The case of Vogt v. Ford Motor Co., St. Louis Ct. of App., (April 2, 1940), 138 S. W. (2d) 634, was an appeal from a decision of the lower court, affirming an award of the Workmen's Compensation Commission in favor of the claimant, Vogt, who had applied for compensation for injuries, holding that his contraction of bronchial asthma, allegedly due to cold air, dust and paint fumes, to which he was exposed during the course of his employment, was an accident compensable under the Missouri Compensation Law.

The evidence showed that the plaintiff worked in the "trim room" where he assembled parts and put them upon cars as they passed along a conveyor line. He was stationed near an open door leading into a paint spraying room from which a fan blew paint spraying fumes through the door. Medical testimony was offered that the claimant had allergic bronchial asthma; that the working conditions to which he was subjected had caused this asthma.

The Court affirmed the opinion, finding that there was sufficient evidence to justify the Commission's finding that the claimant's condition was the result of his employment. It was further held that, since the claimant's condition was due to an allergy, it was not an occupational disease, since "asthma is not a disease, which from the common experience of humanity, is known to result to others engaged in the same work; in other words, the disease is not a general concomitant or the result of the work which he was doing" and was not a disease known to be incidental to that particular employment. However, the Court found that the injury sustained was an accident because it was "an unexpected and unforeseen event happening suddenly and violently with or without human fault and producing at the same time objective symptoms of an injury" found in the act. It was further found that the claimant could not have expected or foreseen that in his physical make-up there was a dormant allergy to the particular dust and fumes of his employment.

The case of Wolfe v. Brohman (Sept. 18, 1940), Supr. Ct. App. Div., 22 N. Y. S. (2d) 403, was an appeal from an award of disability compensation and death benefits in favor of the widow and minor child of the deceased

employee, made by the State Industrial Board under the Workmen's Compensation Law.

The deceased employee was engaged as a butcher and meat cutter, which occupations required him to go in and out of a refrigerator cooler, thereby subjecting himself to sharp variations in temperature. On March 20, 1937, he was compelled to remain in the cooler longer than usual and longer than he had ever remained on any previous occasion. While in the cooler he suffered a severe chill and was sent home and a doctor called. Pneumonia developed, from which he died. The State Industrial Board found that this pneumonia was the result of an accident and was also an occupational disease, which opinion was affirmed by the Court.

There are several cases wherein appellate courts construe occupational disease acts, which are of particular interest.

The case of Blassingame v. Southern Asbestos Co., et al., N. C. Supreme Court (March 6, 1940), 7 S. E. (2d) 478, was an appeal to the North Carolina Supreme Court from an opinion of the Superior Court, affirming an award by the Workmen's Compensation Commission in a claim for death from lobar pneumonia, allegedly due to asbestosis. The claimant's decedent had worked for the defendant for a period of seven years, and died on April 1, 1937, a few days after having driven to the State of Georgia for a vacation, and suffering with a heavy cold at the time of his trip. An autopsy was performed on May 10, 1937, and medical evidence was offered showing he was affected with the early stages of asbestosis. Notice and claim were made out July 19 and filed on July 20, 1937. The claim was referred to the members of the Advisory Medical Committee under the North Carolina Compensation statute, whose testimony established that asbestosis was not a factor in causing the decedent's death. The case was first heard before one member of the Commission, who denied compensation. The full Commission then reversed the decision of the individual Commissioner, finding, as a fact, that decedent's asbestosis lowered his resistance to the pneumonia. This decision was affirmed by the Superior Court and later affirmed by a four-three decision of the Supreme Court of North Carolina.

The two phases of this case that are of particular interest to this discussion are as follows:

1. The findings of the full Commission reversed the decision of the individual Commissioner to the effect that the decedent's asbestosis was a contributing cause of death, because of his lowered resistance to pneumonia, even though the Advisory Medical Committee had testified that the decedent's asbestosis had played no part in his death. Therefore, the majority members of the Compensation Commission, in fact, overruled the medical findings of the Advisory Medical Committee.

2. The North Carolina Occupational Disease Act, Sec. 50-½ (o), provides:

   "Unless written notice of the first distinct manifestation of an occupational disease shall be given to the employer . . . . or to the Industrial Commission within thirty (30) days after such manifestation, and, in case of death, unless also written notice of such death shall be given to the beneficiary hereunder to the employer or the Industrial Commission within ninety

(90) days after occurrence. . . ." the right to compensation for disability or death shall be barred.

In this case it will be noted that death occurred on April 1; that the autopsy was performed on May 10th, and that notice and claim were not made until July 19th. The Court found that the notice given was timely, on the theory that notice of death had been given within ninety days after the widow discovered her husband was suffering from asbestosis, even though the statute established that such notice should be given within ninety days after death. The majority opinion based its conclusion on the ground that the want of notice did not prejudice the employer.

The case of Moffett v. Harbison-Walker Refractories Co., 14 Atl. (2d) 111, decided June 14, 1940, by the Supreme Court of Pennsylvania, was an appeal from an order sustaining a statutory demurrer to plaintiff's statement of claim in a common law action against his employer for alleged partial disability from silicosis. The Pennsylvania Occupational Disease Compensation statute denies compensation for partial disability, and plaintiff's declaration alleged such partial disability sustained while in the defendant's employ. The plaintiff contended that, since the injuries sustained by him were not compensable under the occupational disease compensation act, he retained his right of action for such injuries at common law. In denying the right to the plaintiff to maintain his action the Court said:

> "It is common knowledge that in the early stages silicosis is difficult to detect. A Commission on Compensation for Occupational Diseases which reported to the Governor in 1933 recognized that silicosis and miner's asthma, because of their slow development, presented the 'greatest difficulties as administrative problems.' The Legislature recognized the difficulty of the subject and provided, in relief of the possible burdens on the employer, that the Commonwealth would contribute $100,000 for the purposes specified in Section 7. It also provided, in the circumstances stated in Section 10, for the appointment of a Medical Advisory Board to report to the compensation authorities on silicosis cases on trial, and in Section 11 for the 'entry of any physician, surgeon, or expert upon the premises of the defendant employer in order to ascertain the facts in any case arising under this act.' We think it is clear, therefore, that the legislature intended to bring all silicosis sufferers, whether partially or totally disabled, under the Act and that by accepting the provisions of the compensation acts, an employee agreed to look solely to the Act for compensation and to give up any remedy he might otherwise have had."

In the case of Peter Tomsic v. H. C. Frick Coke Co., decided by the Workmen's Compensation Board of Pennsylvania on September 6, 1940, the claimant filed a petition on January 17, 1939, alleging total disability resulting from silicosis or anthraco-silicosis, which he contracted during his employment with the defendant. The claimant offered medical testimony that he was suffering from chronic bronchitis, better known as "miner's asthma," and admitted that he was not suffering from silicosis or asbestosis.

The Act of July 2, 1937 (P. L. 2714, Sec. 2), which was the Occupational Disease Compensation Act effective during the time of the claimant's em-

ployment, did not provide compensation for miner's asthma. That act was amended by the Act of June 21, 1939, the amendment defining compensable occupational diseases to include "silicosis or anthraco-silicosis (commonly known as miner's asthma and hereinafter referred to as anthraco-silicosis)." The Board held that since this act did not take effect until October 1, 1939, it was not retroactive and that, therefore, the employer was not liable for the compensation sought in this case.

I would like also to call to your attention two decisions of the Missouri Appellate Courts, establishing the time from which limitations run for the filing of occupational disease claims under the Compensation Statute.

In the case of Cleveland v. Laclede Christy Clay Products Company, 129 S. W. (2d) 12, claim for silicosis injuries was made. The claimant operated a truing machine for the purpose of grinding and finishing tile. He was first employed in 1928 and in 1933 began to develop symptoms of chest pathology. He was attended by a physician on January 30, 1936, and advised to discontinue work. He laid off for a period of five weeks during which time roentgen examinations were made of his chest. At the end of five weeks he returned to work, but his condition became gradually worse and he discontinued work on June 22, 1936. He was again examined on January 7, 1937, and advised by his doctor on that date that he had silicosis, this being the first time that he was told the name of the disease from which he was suffering. Claim was filed on January 25, 1937.

The Compensation Commission awarded compensation, which award was reversed by the lower Court, the reversal being sustained by the Court on appeal, because the claim had not been filed within the six months period required by the statute. In passing upon this point, the Court said:

> "According to his own testimony, claimant knew more than six months prior to the filing of his claim, that he had this trouble in his chest, and that he had been treated for it. He knew that the chest trouble was the cause of his inability to do his full work and that the nature of the work aggravated this condition."

The Court further said:

> "This section (Section 3337) has been construed as a limitation on, and an extinguishment of the right itself to maintain the action when not exercised before the six-months period of limitation has run against it. It seems to be the well settled rule in respect to latent injuries that the six-months limitation for filing claim for compensation commences to run from the time it becomes reasonably apparent and discoverable that the employee has sustained a compensable injury."

The case of Renfro v. Pittsburgh Plate Glass Company, 130 S. W. (2d) 165, indicates the attitude of the courts with respect to a construction of the six-months limitation period in a manner as favorable to the claimant as possible in order to protect his claim. That case involved a claim for silicosis, the claim having been filed September 1, 1937, alleging that disability began January 4, 1937. The Commission found, as a matter of fact, that disability began January 4, 1937. On March 11, 1937, the employer and employee had entered into an agreement providing for voluntary payments to the employee, such payments, however, not to be construed as an admis-

sion of liability on the part of the employer for compensation. Payments were made to the employee under this agreement until December 12, 1937. In passing upon the question of limitations, the Court said:

> "If we were to adopt the employee's view in the case at bar, that the Commission's award as for an injury in December, 1935, is correct, although there was no disability or loss of earning power until January, 1937, then a proper regard for the law would require us to hold that the employee's claim herein is barred by the six-months statute of limitations, because his claim was not filed until September 1, 1937. It is extremely important that we should not adopt a rule which would result in claims of employees being barred by the Statute of Limitations under such circumstances as appear in this case. We are of the opinion that the statute of limitations is not involved at all under the evidence herein because we are holding that the Commission's award of compensation, based on an injury as of December, 1935, is incorrect. Such award as for permanent total disability contradicts the Commission's own findings of fact that there was no disability until January 4, 1937, and is not supported by the evidence. Furthermore, the evidence shows that the employee's claim was filed 'well within six months from the date of last payment' as provided by Section 3337 R. S. Mo. 1929."

CONCLUSION

It is apparent that the trend of judicial opinion is toward a liberal interpretation of our compensation statutes, in providing compensation for those injuries to health that are peculiar to the employment, and may be aggravated by conditions of the employment. It is also apparent that in those States having general coverage occupational disease compensation laws, compensation will be awarded for health impairment that is common to every-day life, provided there is medical testimony in the case expressing the opinion that the injuries so sustained may have been caused or aggravated by the conditions of employment. If this tendency continues substantial unanticipated expense will be imposed upon employers and their insurance carriers because of awards for tuberculosis, pneumonia, asthma, and other similar respiratory diseases.

During the State legislative sessions of 1941, we will find, undoubtedly, that many bills relating to this subject will be introduced and pressed for enactment, proposing to liberalize benefits and broaden the scope of compensation coverage.

These developments emphasize the fact that it has become and will continue to be good business for industry to concern itself with the protection of the health of industrial workers and, in giving consideration to the legal responsibility that arises from injuries to that health, we come to the full appreciation of the importance of comprehensive programs for industrial hygiene.

STATE INDUSTRIAL HYGIENE UNITS

STATE AND EXECUTIVE	REMARKS
ALABAMA Dr. John R. Cain, Director, Division of Industrial Hygiene, Department of Public Health, Montgomery, Alabama.	
CALIFORNIA Dr. J. P. Russell, Chief, Industrial Hygiene Service, Department of Public Health, 2002 Acton St., Berkeley, California. Dr. V. A. Nasatir, City Health Department, Los Angeles, California.	
COLORADO Robert J. Owens, Industrial Hygienist, Division of Industrial Hygiene, Colorado State Board of Health, State Office Building, Denver, Colorado.	
CONNECTICUT Dr. A. S. Gray, Director, Bureau of Occupational Diseases, Connecticut Department of Health, Hartford, Connecticut.	
IDAHO Dr. E. L. Berry, Director, Division of Public Health, Boise, Idaho.	
ILLINOIS Dr. M. H. Kronenberg, Chief, Division of Industrial Hygiene, Department of Public Health, 1800 Fillmore St., Chicago, Illinois.	
INDIANA Dr. Louis W. Spolyar, Director, Bureau Industrial Hygiene, Indiana State Board of Health, Indianapolis, Indiana.	
IOWA P. J. Houser, Industrial Hygiene Engineer, C. L. Campbell, Chemical Engineer, Department of Health, Division of Public Health Engineering and Industrial Hygiene, Des Moines, Iowa.	

STATE AND EXECUTIVE	REMARKS
KANSAS Dr. Ernest Boyce, Engineer & Director, Division of Sanitation, State Board of Health, Marvin Hall, University of Kansas, Lawrence, Kansas.	
MARYLAND Dr. John M. McDonald (Medical Department), Director, Bureau of Occupational Diseases. Dr. Wilmer H. Schulze, Director, Sanitary Section, Baltimore City Health Department, Baltimore, Maryland.	City of Baltimore only
MASSACHUSETTS Manfred Bowditch, Director, Bureau of Occupational Hygiene, Department of Labor and Industry, Boston, Massachusetts.	Conducted in a cooperative, joint approach to problem by labor and health departments
MICHIGAN Dr. Kenneth E. Markuson, Director, Bureau of Industrial Hygiene, Department of Health, 1151 Taylor Avenue, Detroit, Michigan.	
MINNESOTA Dr. A. J. Chesley, Executive Officer, Department of Health, 469 State Office Building, St. Paul, Minnesota	Service available after January 1, 1941
MISSISSIPPI Dr. J. W. Dugger, Director, Industrial Hygiene & Factory Inspection, State Board of Health, Jackson, Mississippi.	Data service will be available not yet announced
MISSOURI Dr. Harry F. Parker, State Health Commissioner, State Board of Health, Division of Engineering, Jefferson, Missouri.	
MONTANA Dr. Lloyd M. Farner, Director, Division of Industrial Hygiene, State Board of Health, Helena, Montana.	
NEW HAMPSHIRE Frederick J. Vintinner, Industrial Hygienist, Industrial Hygiene Unit, State Board of Health, Concord, New Hampshire.	

STATE AND EXECUTIVE	REMARKS
NEW YORK Dr. Leonard Greenburg, Executive Director, Division of Industrial Hygiene, Department of Labor, 80 Centre Street, New York, New York.	
NORTH CAROLINA Industrial Commissioner, North Carolina Industrial Commission, Raleigh, North Carolina.	Even though the Division of Industrial Hygiene is a department of state health department, Industrial Commission allocates certain funds to it.
OHIO Dr. J. B. Kistler, Chief, Adult Hygiene Division, Ohio Department of Public Health, Columbus, Ohio.	
OKLAHOMA Dr. G. F. Mathews, Commissioner of Health, H. J. Darcey, Chief Engineer, Director, Bureau of Sanitation, State Health Department, Oklahoma City, Oklahoma.	
PENNSYLVANIA Dr. John J. Shaw, Secretary of Health, Department of Health, Commonwealth of Pennsylvania, Harrisburg, Pennsylvania.	
RHODE ISLAND Charles L. Pool, Chief of Division, Division of Sanitary Engineering, Department of Health, State Office Building, Providence, Rhode Island.	
SOUTH CAROLINA Dr. Harry F. Wilson, Director, Division of Industrial Hygiene, State Board of Health, Columbia, South Carolina.	
TENNESSEE W. C. Williams, Commissioner Department of Public Health, State of Tennessee, Nashville, Tennessee.	
TEXAS Dr. C. A. Nau, Director, Division of Industrial Hygiene, State Department of Health, Austin, Texas.	

STATE AND EXECUTIVE	REMARKS
UTAH J. L. Jones, M. D., D. P. H., Director, Industrial Hygiene, State Board of Health, Salt Lake City, Utah.	
VERMONT Harold W. Slocum, Director, Tuberculosis Division of Industrial Hygiene, Department of Health, 348 College Street, Burlington, Vermont.	
VIRGINIA R. T. Homewood, Engineer, Bureau of Industrial Hygiene, Department of Health, Richmond, Virginia.	
WEST VIRGINIA Dr. Arthur E. McClure, State Health Commissioner, Department of Health, Charleston, West Virginia.	
WISCONSIN Dr. P. A. Brehm, Supervisor, Division of Industrial Hygiene, State Board of Health, Madison, Wisconsin.	

The following City Health Departments also have industrial hygiene divisions:

Flint, Grand Rapids, Newark, Saginaw and St. Louis County.

DR. MELLER: Thank you very much, Mr. Dorsett. Is there any discussion?

We go then to the Symposium on Industrial Health Defense. Professor Drinker will preside.

PROFESSOR DRINKER: Regardless of our jobs or our political affiliations, the general topic of this symposium is one of enormous interest to us. We have the advantage of having with us, as participants in this symposium, several men whom we have never had the pleasure of hearing at these meetings and some of whom have not attended the meetings at all before.

The first speaker, Dr. Neal, who is no longer acting chief but is chief of the division of industrial hygiene, is the successor of our very good friend and ardent supporter, Dr. Sayers. It is especially pleasant for us to have him here with us today. His subject and the activities of the Public Health Service in the national defense program are perhaps one of the chief guiding points for those of us whose professions lie in industrial hygiene. We look to them constantly for advice and for direction. It is especially pleasant for us to have the chief of the division with us today.

ACTIVITIES OF THE PUBLIC HEALTH SERVICE IN THE DEFENSE PROGRAM

PAUL A. NEAL, M. D.*

One of the outstanding developments of the present national defense program has been the recognition on the part of the leaders of our Nation that health is an essential element of preparedness. On September 19, 1940, the President approved an order establishing a Health and Medical Committee to advise the Council on National Defense, and to coordinate medical and health activities affecting national defense. This Committee consists of the following members: Dr. Irvin Abell, Chairman; the Surgeon General of the Army; the Surgeon General of the Navy; the Surgeon General of the Public Health Service; and the Chairman of the Division of Medical Sciences of the National Research Council. This Committee and its many sub-committees have the responsibility of advising the Council on National Defense regarding the health and medical aspects of national defense, and of coordinating health and medical activities affecting national defense. One of the important subcommittees set up by the Health and Medical Committee of the Council on National Defense is the one on industrial medicine.

Inasmuch as the program for national defense has already served to emphasize many problems pertaining to the public health, the Surgeon General of the Public Health Service, Dr. Thomas Parran, called a special conference of the State and Territorial Officers on September 16 and 17, for the purpose of reviewing plans and agreeing on a program of action. Dr. Parran enunciated the keynote of the conference in the introductory paragraph of his address. He said:

> "The most impelling problem which we face today is that of maintaining the safety of this country and its institutions. For their aggressive defense, we are gearing up governmental methods, mobilizing resources and manpower. For the first time in all history, world events have thrust upon us the concept of a total war. In preparing a total defense, all factors ultimately rest upon the one fundamental resource of the country, manpower. Medicine and public health, through the centuries, have been devoted to the conservation of manpower and its socially constructive use."

In any discussion of methods planned for effectively carrying out a program it is essential that the problem to be solved be definitely outlined. The various health problems confronting the Nation as a result of the present emergency were clearly defined at the Conference of State and Territorial Health Officers meeting with the Surgeon General last September in Washington. These may be briefly summarized as follows:

EMERGENCY HEALTH PROBLEMS

Rehabilitation

One of the pressing problems we are faced with is the physical rehabilitation of registrants disqualified for duty with the armed forces. Based on the experience of the last World War, we may expect approximately 25 per cent of those examined to be rejected, with about 35 per cent examined having one or more defects. Of the 17,000,000 registrants, probably 6,000,000

* Chief, Division of Industrial Hygiene, National Institute of Health.

in military and industrial mobilization areas." The Conference further urged the coordination of activities pertaining to industrial hygiene and stressed the necessity for supplementing the efforts of the States.

Industrial Hygiene

It is now a well established fact that military mobilization and expansion is impossible without industrial mobilization and expansion. The present industrial expansion has already brought in its wake many new problems in employee health. To insure maximum physical fitness, the industrial worker needs to develop a high sense of personal responsibility. Industry must continue and accelerate its efforts to protect and promote physical welfare among workers, and the various health agencies, both governmental and private, must give every possible assistance through scientific research, through the expansion of community sanitation measures, and through the application of our developments in both the prevention and treatment of disease. Despite continued progress in the field of industrial hygiene, we still have in this country 17,000 occupational deaths from accidents every year; 75,000 permanent disabilities; and 1,400,000 temporary disabilities. Recent surveys conducted in industry show that the working environment of about 1,000,000 persons requires investigation by trained industrial hygienists to determine whether or not the presence of silica dust in the air constitutes a health hazard. The conditions of work of 800,000 persons handling lead and its compounds also need study. Similar information is available on many other potentially hazardous exposures. For example, it is well known that many millions of workers are exposed to materials which, if improperly used, are capable of causing skin diseases. Furthermore, it is well known that industrial workers have higher rates of physical defects than non-industrial workers and that the death rate from all causes among unskilled workers is 100 per cent or more in excess of the death rate among agricultural employees. The many studies of illness among workers recently completed show that the average worker in this country loses 10 days a year on account of sickness. In fact, general illnesses have been found to account for 15 times as much time lost as that due to both accidents and occupational diseases. This problem of lost time from disability among workers is of vital importance now that our industrial machine is playing such a prominent role in the defense effort.

Our recent studies have also shown clearly that although industry is cognizant of the problem and is making progress, much still remains to be done. For example, only 25 per cent of our workers have full time safety direction services, only 15 per cent have full time medical services, and only 33 per cent have nursing services. Engineering studies have also brought out the fact that protection against exposures to hazardous materials and conditions is still far from adequate.

The present industrial activity on defense orders has already augmented these problems in industrial hygiene. Able-bodied men are being mobilized into our armed forces and these will have to be replaced in industry by older men, by young adults and, in some cases, by women. Many of these newly inducted industrial workers will not be as physically fit for arduous tasks, nor will they be as skilled. The problem of fatigue, so important in the first World War, will again reappear as the result of the rapid tempo of industrial production. Many hazardous chemicals, both old and new, will be introduced into industry. We may expect crowding in factories and we must fight the tendency to relax in the vigilance so necessary to prevent accidents

and diseases among workers. Dr. Parran has stated that our industrial machines are rated to be the most efficient in the world, and he rightfully insists that the men and women who operate these machines should have a comparable efficiency.

These, in brief, are some of the problems facing us today in our health preparedness program. The Committee on Health and Medical Preparedness recently established by the President, and its many subcommittees, are already coping with these various problems confronting us. The work of coordinating existing machinery both as to public health and medical facilities is under way. Insofar as industrial hygiene is concerned, the groundwork laid by research during the past quarter century and the machinery developed during the past several years for the application of this research by the States and by industry finds us better prepared to cope with industrial health problems than at any time in our industrial history. We know that every job can be done safely if we only apply our present knowledge.

THE PROGRAM

With reference to those activities on health and medical preparedness concerned with the civilian population and our armed forces while in training, the Public Health Service is working very closely with the Army, the Navy, and the State and local health departments. The Surgeon General of the Public Health Service has already appointed medical and engineering liaison officers to each of the Army corps areas, for the purpose of expediting the solution of the various problems discussed herein. Reconnaissance surveys are even now being made by the Public Health Service and State health department personnel in the various areas to determine the exact needs in each locality.

Fortunately, the industrial hygiene program could be launched immediately, in view of the fact that the various States in organizing their industrial hygiene services had already made detailed surveys of the problems in each locality. The States are therefore in a position to know where these services are needed. Furthermore, many of the research activities of the Division of Industrial Hygiene, which are carried out during peace time, furnish us with the key to the solution of problems of a military nature and some of our findings can also be applied to emergency problems in industry. For example, the study this Division recently completed on the fatigue status of truck drivers has furnished us with a great deal of information applicable to fatigue problems which may arise in industry as a result of increased activities.

The industrial medicine subcommittee established by the Health and Medical Committee of the Council on National Defense has already been assured the fullest cooperation of the Industrial Hygiene Division of the National Institute of Health. All of the facilities of this Division will be put at the disposal of the committee, and a greatly expanded employee health program is being developed in close collaboration with the 31 State industrial hygiene units, with industry, labor, and numerous official and unofficial agencies. In other words, a program has been developed for coordinating the work of all the Federal and local agencies, of industry, of labor, of the medical and engineering professions, and of all organizations vitally concerned with industrial health.

Insofar as the Division of Industrial Hygiene of the National Institute of Health is concerned, its functions may be considered as partly adminis-

trative, concerned with coordinating all activities both at the Federal and State level, and mostly concerned with the promotion of industrial hygiene services in State and local health departments, and with scientific investigations conducted both at its laboratories and in the field. The Division of Industrial Hygiene is already carrying out urgent research directly related with defense and it is working very closely with both the Army and the Navy. In addition to fundamental laboratory research on toxic materials of direct importance to national defense, some of the Division personnel are being employed to supplement the work of those States which have been confronted with emergency problems too great for successful handling by their own efforts. If the necessary funds for this phase of the work are obtained, it is planned to have at least 15 units in the field, each consisting of a trained physician, an engineer and technical support, for the purpose of assisting the State industrial hygiene units on emergency problems. Several such units are already doing work with the States.

In brief, the work which the Industrial Hygiene Division is already performing, and which it hopes to expand, may be summarized as follows:

1. Industrial hygiene services to Government arsenals and other Federal projects.
2. Assistance to the States on industrial hygiene problems in defense industries.
3. Toxicological research on defense materials.
4. An appraisal of the fatigue status in relationship to the national defense program.
5. The determination of methods for the absorption of handicapped persons into vital industries for national defense.
6. The preparation and dissemination of information on various toxic materials and processes, including approved designs of exhaust systems for the control and elimination of atmospheric contaminants.

Practically all of the States having industrial hygiene divisions have already submitted programs to the Public Health Service for assisting industry in maintaining healthful working conditions and healthy workers. These programs differ in no way from those already practiced by the States in the past, except that emphasis will be given to directing their efforts to industries engaged on defense contracts. In other words, the work in the States will embrace the following activities:

1. The evaluation and control of the various health hazards arising from exposure to dusts, fumes, gases and vapors.
2. Advice to industry in the construction of new plants and in the renovation of old plants in the interest of safety and health.
3. The promotion of physical examinations and medical services to workers by industry.
4. The promotion of measures for the control of communicable disease, such as syphilis, pneumonia, tuberculosis, and others.
5. The preparation and dissemination of information on various toxic materials and processes, including approved designs of exhaust systems for the control and elimination of atmospheric contaminants.

Industry is thoroughly convinced of the fact that it pays to control accidents. In reducing the frequency, severity and monetary losses involved in

industrial accidents industry attacked the problem from two viewpoints: First, by the provision of well guarded equipment, and second, by the education of workers in safety measures and practices. We are thoroughly convinced that the same tactics will materially aid in reducing lost time from occupational diseases and other illnesses. In other words, first, we must provide a safe and healthful environment, and second, we must educate the worker in safe practices and healthful living.

It is quite evident that insofar as public health and medical preparedness are concerned, plans have been created which are not emergency improvisations, but are designed to be an integral part of our national life in the future. This is especially true of our industrial hygiene program, and it is hoped that we will not forfeit the gains we have made so far for the sake of expediency. All of us, be we public health workers, private medical practitioners, engineers, chemists, industrial managers, or factory workers, must assume leadership and responsibility and coordinate all of our efforts, so that the men and women in our industries will attain a high level of efficiency and health.

PROFESSOR DRINKER: I am sure that Dr. Neal will be glad to answer questions as to the matters which he has brought out.

MR. CONNOR: I should like to ask the speaker whether or not he can tell me this. I have about sixty young men of draft age who are receiving regular routine treatments for syphilis. They are continually asking me if when they are called the government will continue their treatments in active service or whether they will be rejected. I can't tell them. I'd like to have some help.

DR. NEAL: I can't speak for the Army, naturally, on that. My opinion would be, and I think it is the present thought, that each individual case is decided on its own merits in connection with this communicable disease, as others. If the men are not disabled, and we assume these are not—in other words, they don't have cardiovascular disease—they will receive the same treatment they are getting now after they are inducted into the service. Whether they will be inducted into the service or not I don't know. I think it will be decided on each individual case.

PROFESSOR DRINKER: Dr. Sayers, I think we should all be glad to hear a word from you.

DR. SAYERS: I am very much interested, as you know, in the program that has been outlined by Dr. Neal for you and to you. It is one that has been developing for a matter of years. There are many of you sitting around here who have been associated with us in the development of that program. Most of you in the room have had something to do with it.

The program is not new and it is not a program that is just for defense alone. As in all industry, as in all the work that we are going to do, it must be speeded up if it is going to accomplish the purpose we are to have.

In answer to the question, if I may expand a little on Dr. Neal's statement, I think that whether any individual who comes up is inducted or not will be determined by your local draft board to a great extent. I don't think we need to worry very much about those people with syphilis. They will receive treatment. They will no longer be communicable from one to another in the army. I don't think we need worry too much about it.

I do think that we all have a real problem in regard to industrial

hygiene. I think we all must get behind it and push it. We cannot succeed in this war unless we have our workers in good health. Whenever a man is away, it doesn't make any difference whether it is occupational disease, accident, or just illness that causes him to be away, it interferes with production, and production is important to us today. It is a very serious thing.

Again, we are not here to scare anybody. We just want to work along efficiently, and scared people are not efficient. We have a job in front of us, and it is a big job. We must all work. I hope we can support all the group.

There are several here on this medical defense who belong to the Advisory Committee, I believe, of the Medical Defense Commission—or is it the Defense Council? You know the Defense Council is made up of secretaries in our cabinet. There is an Advisory Commission to the Defense Council. Then they have special committees in the Research Council that advise them, and the Medical Committee is one of that organization, as I understand the present group. Then there is a sub-committee on industrial hygiene. The program is being worked out, I believe, expeditiously.

We cannot work too fast. The groundwork has been laid in the period of years that has gone by and part of that groundwork is within this organization here. Dr. Neal has emphasized to you the importance of cooperation and coordination. That must be had if it is to be efficient.

It is a great pleasure for me to be here with you today. I thank you all.

PROFESSOR DRINKER: If there are no further questions we will proceed with the next paper. Thank you very much, Dr. Neal.

Our role as a nation in this present emergency, whether we are helping the Allies or whether we are simply helping ourselves, is distinctly up to the present time one of production—production of armaments. It has been said by those in position to know that there is probably no single operation that is more important to the production program than welding, both electric welding and gas welding. Dr. McCord, with his colleagues, for some time has been engaged in the study of the health aspects of arc welding. The first paper that they have published on this subject appeared recently in the Journal of Industrial Hygiene. Perhaps some of you have had the opportunity to read the paper.

It is particularly pleasant for us, therefore, in the Foundation, to have had Dr. McCord accede to our request that he come and talk to us today on his own work. I have much pleasure in introducing to you Dr. Carey P. McCord, Director of the Industrial Health Conservancy Laboratories.

THE HEALTH OF ELECTRIC ARC WELDERS IN THE NATIONAL DEFENSE PROGRAM

GORDON C. HARROLD, Ph. D.,* STUART F. MEEK, M. D.,* and CAREY P. McCORD, M. D.†

Introduction

Arc welding in itself cannot defend a country or win a war, but neither of these national enterprises well may be prosecuted without reliance upon arc welding for many essential fabrications. The extent of arc welding in this country may be gauged by the fact that in 1939 152,363,400 pounds of welding electrodes were utilized, with a corresponding figure for the first six months of 1940 of 86,644,800 pounds. From another source has come the statement that the total production of welding rods, including automatic welding rods, was, for the year 1939, 137,430,206 pounds, of which 129,050,211 represented heavily coated carbon steel rods, with only 10,154,186 pounds of bare washed or dust coated rods. These figures indicate that less than 8% of the country's arc welding is carried out with bare rods. However, this marked shifting to heavily coated rods is not true for all industries. In one automobile plant, where the greater portion of welding is accomplished by spot or pressure welding, thus calling for no electrodes, more than a million pounds of bare welding rods were applied in a recent 12 months' period, but of this total poundage of welding rods only 0.8% was represented by coated rods of all varieties. Every modern standard automobile possesses more than 5,100 welded points with, however, only some 140 arc welds, but several of these arc welds are many inches long.

This year of 1940 saw the first all-welded ocean-going steamer, the 17,000 ton Exchequer, launched in Mississippi in June. Commercial naval construction technology of this war period in some measure may become symbolized by this first all-welded freighter. The extent of welding, without at this time specific reference to arc welding, again is reflected by the fact that about 200,000 welders are now employed in the United States. To express this in military terms, these welders represent an army of approximately 23 stream-lined divisions or seven army corps.

As welding applications increase, more and more the need is for a better understanding of the hygienic exposures that may arise. The world of industrial hygiene is not in a happy position in its knowledge of the nature and extent of exposures for arc welders. Apparently many of us are hampered by too much reliance upon traditional standards, particularly with regard to nitrous gases. Examination of the original publications upon which some of these much advocated standards are based reveals a scantiness of evidence far in disproportion to the significance long attached to them.

In facing the actuality of industrial hygienic shortcomings with regard to the exposures connected with arc welding, several things contribute to the obvious complexity of the situation. First, the *quantitative determination* of some welding gases, such as the several nitrogen oxides and ozone is far more difficult than for many other substances. Second, the coating materials applied to welding rods are so diversified and so rapidly changed as to baffle almost any attempt to keep abreast of developments. Third, alloyed steels,

* Chrysler Industrial Hygiene Laboratories.
† Industrial Health Conservancy Laboratories.

such as nickel-chrome steel, or metals that have been coated, such as in galvanizing, are introducing new difficulties in connection with welding operations. As a result, the attending industrial hygienic situations are far from simple.

In general, the potential sources of injury from arc welding other than from trauma and thermal effects, seemingly may be grouped in the following categories:

Oxygen deficiency.
Carbon monoxide.
Nitrous gases.
Ozone.
Metal fume from alloyed steel.
Metal fume from the metal electrodes.
Silica from welding rod coatings.
Harmful substances other than silica in rod coatings.
Injurious substances coated on to the metal being welded.
Radiations.
Possibly other gases such as cyanides theoretically produced in the electric arc.

From fairly extensive first hand experimental work and medical department experience with several hundred welders, we have reached a number of conclusions soon to be presented, but unfortunately space does not permit adequate presentation of the evidences upon which these conclusions are based. Before entering upon any further discussion of some arc welding exposures, it is here emphasized that arc welding does not preempt the field of possible injuries related to welding and that gas welding in particular may provide dangers under fortuitous circumstances.

Brief Review of Our Recent Experimental Work with Bare Iron Electrodes[1]

In our welding experiments, welding was carried out for six hours a day, five days a week, in a metal gassing chamber 10x10x10'. In these experiments, the welder was located on the outside of the chamber performing welding with arms inserted through portholes. Animals in cages protected against ultraviolet light from welding were exposed for six and one-half hours daily. The exhaust system of this exposure chamber was dampered to less than two changes per hour. The rate of welding was represented by the maximum number of rods that might be burned in a unit of time. In one experiment with a voltage of 44 and an amperage of 300-350, 48 to 50 bare rods were burned in 24 minutes of every hour, which aggregated approximately 19 pounds per welding day. In four major experiments, limited to uncoated welding rods, 250 animals (rabbits and albino rats), including 87 controls, were observed over a period of 20 months with the longest experiment continuing 9 weeks (45 exposure days).

These experiments established that nitrous gases (reported as nitrogen dioxide [NO_2]), increased with every increase in voltage across the arc as utilized. At 27 volts (180-200 amp.) the average ppm. were 29; at 31 volts (200-225 amp.), 33 ppm.; at 32 volts (250-275 amp.), 40 ppm.; and 44 volts (300-350 amp.), 70 ppm. While the average concentrations found were as just indicated, concentrations at times ranged much higher, averaging 97

ppm. under some conditions with an extreme maximum of a single sample of 135 ppm. with a duplicate of 127.

While suitable tests were made for carbon monoxide, excess carbon dioxide, oxygen deficiency, excessive temperatures, chlorine, etc., chief determinations made were the quantities of nitrogen dioxide, ozone and metallic fume. Nitrogen dioxide was determined through the nitrate method and samples collected throughout the day, immediately at the cessation of every welding cycle, 4", 10" and 16" thereafter, that is, in every hour there were two welding cycles, each with welding continuing for 12-14" and sample taking from 16-18". Thus, in a single hour, as many as 8 samples for nitrogen dioxide alone at times were collected. In the aggregate, approximately 2,500 determinations were made for welding gases and fumes.

The quantity of particulate iron present as a fume and arising from the arc ranged from 33-395 mgms. per cubic meter of air. The quantity of iron fume constituted no measure of the quantity of nitrous gases present. Likewise, the quantity of fume stands in no fixed relation to the voltage across the arc.

Ozone (O_3) both has been affirmed and denied as existing in the atmosphere about the electric arc. In our experience, from 10-32 ppm. were found within 1" of the arc, from 0.5 to 9 ppm. 4" from the arc and from 0.1 to 1 ppm. in the center of the chamber, all samples being taken during the flaming of the arc. Unlike nitrous gases, increase in voltage apparently did not create increased quantities of ozone.

No product of this experimental welding, regardless of voltage or of the prolonged duration of exposure produced any obvious changes characteristic of the action of nitrous gases. With a voltage of 44, the experimental animals were exposed for 125 hours to a concentration of 70 ppm. of nitrogen dioxide, including 33 hours in the aggregate at 84 ppm. of nitrogen dioxide and with the exception of a portion of the first hour of every welding day, no exposure to nitrous gases during the 40 days of elapsed time was lower than 46 ppm.

In all experiments, the survivors exceeded 90% of the animals at the beginning of the exposures, not including those deliberately sacrificed at various intervals. Death rates did not exceed those for control groups.

In general, we sought to produce as high quantities of nitrous gases as were possible under any conditions resembling practical arc welding. We utilized voltages well above those commonly utilized in practical welding. We burned rods at a much higher rate than any welder is likely to do. We carried out such experimental welding in a confined area as represented by a thousand cubic feet with only about one change of air per hour. Notwithstanding, we were unable to produce any gases or fume from bare welding rods that brought about any readily observable injurious action on exposed animals except for the formation of methemoglobin, the significance of which is discussed in a later section.

In view of the significant absence of pathology after several weeks of exposure to nitrous gases in excess of those quantities regarded as harmful for even brief exposures, we are disposed to question the standards that are now widely accepted and promulgated. While a few hours have been spent by humans of our own group in the gassing chamber with concentrations of nitrous gases at 84 ppm. for 3 hours, 93 ppm. for 40 minutes and 103 ppm. for 3 minutes, and while many scores of casual observations have been made upon practical welders, it is to be recognized that our outstanding evidence

as to the action of nitrous gases originating in arc welding is limited to animals. Within these limitations, we are disposed to regard arc welding in open areas carried out with bare mild steel rods on uncoated common types of steel as little associated with practical dangers from nitrous gases.

Coated Welding Rods

Thus far we have had opportunity to expose animals to the gases and fumes of but one coated welding rod. This rod presented the following approximate analysis:

Moisture and volatile matter	8.44
SiO_2	20.20
TiO_2	43.12
MnO_2	11.00
$CaCO_3$	4.37
$Mg_3(PO_4)_2$ (theoretical)	13.30
	100.43

The last item is open to considerable question. It is known that phosphorus is present, but in what quantity and in what form has proved to be difficult to establish. The mild steel on which this coating has been applied has not been analyzed, but it believed to conform to the common type of steel rod with approximately 98% of iron and the remainder being made up of the following:

Carbon	0.13-0.18%	
Manganese	0.40-0.60%	
Silicon	0.06	% maximum
Sulfur	0.04	% maximum
Phosphorus	0.04	% maximum

This coated rod was burned in simulated welding at a voltage of 45 at the rate of 46 rods per welding hour or 23 pounds per day (ignoring rod stubs). Forty animals (rabbits and albino rats) were exposed for 6.5 hours daily, 5 days per week, for 45 days. The chief atmospheric determinations were for nitrous gases and ozone. At this voltage the bare rods burned at the same rate yielded an average concentration of 70 parts of nitrogen dioxide per million. The arc shielded by the gases from this particular rod led to the formation of considerably less nitrogen dioxide, the average quantity being 33 ppm. It appears to be generally true that the shielded arc is associated with the formation of smaller quantities of nitrous gases. Insofar as nitrous gases are the offending agents in arc welding, a preference may be expressed for the coated welding rod.

At this time, unusual interest exists in the nature of the coating materials for welding rods. Many partial analyses have been published, but some of these are highly confusing since the findings on addition may total from 40 to 140%. The chemical nature of many rods is still a well guarded secret, but it is known that an almost unlimited variety of substances have been tried out as welding rod coatings. The number includes silica, asbestos, other inorganic silicates, organic silicates, titanium, calcium, magnesium, manganese, fluorine, aluminum, columbium, nickel, iron, barium, chromium, phosphorus, borax, wood meal, cellulose, gum arabic, sage, tea leaves, asphalt, cotton, jute, paper, cereals, string, starches, sugars, etc. Whatever

constituents enter into the rod, it is generally conceived that they serve some of the following purposes.

1. To provide a slag coating of the metal weld during the period of freezing.
2. To furnish alloy material not present in the metal core in the rod in order that the weld itself may conform to the original metal.
3. To confine or stabilize the arc itself so that the size of the arc is within reason constant and the flow of electric power essentially uniform.
4. It is usually stated that the shielded arc excludes atmospheric nitrogen and oxygen. This possibly may be doubted and instead possibly the shielded arc alters the action of ultraviolet rays upon the atmospheric gases.

To meet these various purposes served by the rod coating, highly different substances enter into the coating mixture. Thus, to produce visible cloud about the arc, there may be present such ingredients as cotton lint, paper, powdered coke, etc. For the protecting slag layer immediately over the welding point, inorganic substances chiefly are used including borates, barium, calcium, aluminum and manganese compounds along with many others. In order to make up the deficiencies of the welding rod itself in relation to the metal to be welded, manganese, chromium, lead may enter into the coating itself. Lastly, it is necessary to bind these ingredients on to the rod, which calls for another class of materials, some of which in addition to being binders are gas generators. The number includes asphalt, resin, casein, ethyl silicate, sodium silicate, etc.

By way of practical objections to the use of heavily coated rods, welders have indicated that it is difficult to (1) observe the weld being made because of the overlying slag; (2) the cleaning of welds made with heavy coated rods is more difficult than the bare rod; (3) the increased visible clouds from the coated rods are disturbing and unwanted.

A somewhat representative analysis of the coating for rustless steel rods is the following:

Organic materials	19.2 %
Silica (SiO_2)	22.02%
Fluorine	14.4 %
Calcium oxide and carbonate	30.1 %
Iron oxide	1.7 %
Manganese oxide	2.46%
Aluminum oxide	11.1 %
	100.98%

An analysis of a similar coating material for high chromium nickel rods as furnished by Captain Brown, the next speaker, is as follows:

Lime	30-35%
Sodium fluoride	30-35%
Ferromanganese	6- 7%
Silica	6- 7%
Sodium silicate	11%
Cellulose	4%
	± 100%

From the same source were obtained analyses of coatings for carbon steel rods:

Cellulose	30%
Asbestos	15%
Ferromanganese	12%
Titanium dioxide	12%
Sodium silicate	30%
	99%

Lime	5– 6%
Iron oxide	20–25%
Ferromanganese	15–20%
Asbestos	3%
Feldspar (silicate)	30%
Sodium silicate	11%
Pyrolusite (MnO_2)	8%
	= 100%

While many of the varied substances that enter into the coating of welding rods well may be looked upon with suspicion as sources of injury to welders, our personal observations and conclusions are restricted to four items related to only one type of coated welding rod, namely:

1. The shielded arc apparently diminishes the quantity of nitrous gases.
2. Significant weight and growth deviations occur.
3. The quantity of free silica present in the dust from arc welding with coated rods containing free silica appears to be insufficient to stimulate any proliferative reactions on intraperitoneal injections.
4. Methemoglobin is produced in the blood of animals exposed to the products of welding with coated rods. (This statement does not imply that methemoglobin is not formed from the products of welding with bare rods.)

The last two items above have been made the object of further comment in two sections now following.

Methemoglobin

Methemoglobin and methemoglobinemia long have been associated with nitrous gases and at least casually with welding.

In our early experiments, no opportunity arose for any blood counts or methemoglobin determinations. In the sixth of the series of experiments already mentioned, methemoglobin determinations were made with the Pulfrich-Zeiss photometer, standardized respectively for male and female rats and rabbits, at the end of 45 days of exposure when the concentration of nitrous gases averaged 25 ppm. and when the voltage utilized was 45 (300-330 amp.) Table I presents the quantities of methemoglobin found in 10 male rats, together with results one week after the cessation of exposure. However, on other occasions, subsequent to exposure, the percentages of methemoglobin were in some instances higher, but always within the limits of experimental error. At the time the experimental animals were

TABLE I

METHEMOGLOBIN IN MALE RATS

Rat Identification	Percentage Methemoglobin	
	8-13-40 After 45 Exposure Days (63 Elapsed Days) to 25 ppm. NO_2 from Coated Rods	8-20-40 Seven Days After Last Exposure Date
1955	21.7	2.7
1953	17.5	1.5
1980	10.4	2.5
1978	14.7	4.2
1957	11.5	3.8
1969	16.0	4.2
1936	16.3	0.0
1992	10.4	0.7
1988	13.0	3.1
1963	18.7	2.3

Controls

Rat Identification	Percentage Methemoglobin	
1945	0.0 (8-13-40)	4.2 (8-27-40)
1985	0.0 (8-13-40)	3.5 (8-20-40)

tested (8-13-40) two control male rats presented no measurable trace of methemoglobin, but on other occasions methemoglobin has been detected in control rats up to a maximum of 4.2. However, this figure embraces the probable error circa ±2%.

The assumption was that the occurrence of methemoglobin was in nowise peculiarly associated with coated welding rods, which caused us to repeat for a few days an earlier experiment with bare rods using a voltage of 45. Table II presents methemoglobin percentages just prior to exposure, at the end of one day of exposure, and three days after the cessation of a three day welding period. Although the results shown are sometimes within the limits of experimental error, the uniformity of results suggests that one day of exposure may increase the blood content of methemoglobin, which quickly disappears by the end of the third day after exposure. During this period the average quantity of nitrous gases was 70 ppm.

In the case of four welders, samples of blood collected during the period of welding work revealed respectively 2.5, 2.3, 2.3 and 2.6 per cent of methemoglobin. One human control yielded no methemoglobin at this time. Of 13 determinations for nitrous gases made in connection with the possible exposure of these welders, the highest was 13.3 ppm. of nitrogen dioxide in a sample collected from an automobile body in the course of construction.

The inference is that some methemoglobin formation is a common result from mild exposures to welding gases. This statement scarcely applies to experimental rabbits, which animal appears to be more resistant to methemoglobin formation.[2] Should the need arise, it may prove possible to utilize the occurrence of methemoglobinemia as qualitative proof of exposure to welding gases. In the absence of other sources of methemoglobin causation, the presence of any considerable quantity of this substance in an arc or gas welder suggests exposure.

The nature and physiologic significance of methemoglobin in the blood is far from clear. In congenital or familial methemoglobinemia, as much as 40%[3,4] has been reported as continuous and little associated with any evidences of ill effects. However, in many publications dealing with clinical methemoglobinemia, the method of demonstrating the quantity present may be open to question. It has been stated that methemoglobinemia up to 60%[5,6] of saturation is compatible with life. Other publications indicate that the behavior of the body in the presence of methemoglobinemia conforms in general to the pattern that governs carbon monoxide hemoglobin.[7-8,9,10,11,12,13,14,15]

It is frequently asserted that methemoglobin is a precise antagonist to cyanide action and that its presence in the blood in cases of cyanide poisoning is a fortunate occurrence. It has been claimed that methylene blue as a treatment for cyanide poisoning is efficacious from its causation of methemoglobinemia, which substance in turn combines with the cyanides with resulting body protection through the prevention of internal asphyxiation. Thus if it be true that some cyanides may be produced in connection with arc welding, the presence of some methemoglobin may serve a beneficent rather than detrimental purpose. Granting some possibility of an anti-toxic action of methemoglobin under some circumstances, it may not be inferred that the oxygen requirements of the tissues of the body may be met in the presence of any high percentage of methemoglobin, since the combination of methemoglobin and oxygen provides little or no available oxygen for tissue use. It is here emphasized that the present state of evidence is far from conclusive.[16,17,18]

TABLE II

METHEMOGLOBIN IN MALE RATS

Rat Identification	Percentage Methemoglobin			
	9-4-40 Prior to Exposure	9-4-40 (3 o'clock) After 1 Day of Exposure to 70 ppm. NO_2 from Bare Rods	9-6-40 (3 o'clock) After 3 Days Exposure to 70 ppm. NO_2 from Bare Rods	3 Days After Cessation of Exposure
2078	0.5	2.4	2.3	0.0
2069	2.0	3.8	2.4	0.0
2082	0.6	3.0	3.5	0.0
2065	0.0	2.1	3.1	0.0
2084	0.0	1.0	3.6	0.1
2071	0.4	0.0	3.1	0.2

Controls

Rat Identification	Percentage Methemoglobin
1964	0.5
2069	0.0
2055	1.5
1981	1.8

Intraperitoneal Injection with Welding Dust after Welding with Silica-Containing Coated Rods.

Large numbers of coated rods contain silica or silicates. The prime use of silicates is as a binder for other coating materials. Organic silicates are used for this same purpose, but this use is limited. The high temperatures (circa 6,500° F.) of the electric arc may convert the greater portion of combined silicon to silica. In the analysis of rod coating materials used in one of our series of experiments, the SiO_2 ranged between 17 and 20%. It has been computed that from the burning of a single 14", 5/32" coated rod more than 150 mgms. of fine particulate matter is introduced into the atmosphere. While this figure in itself is of little significance, it leads up to the statement that arc welding is or may be a dusty operation.

Because of a maximum content of 20% of free silica of the total weight of the electrode of the one rod utilized by us, we collected with the electrical precipitator sufficient quantities of this dust for intraperitoneal injections. On analysis, this dust proved to have a content of only 8% silica, a larger percentage consisting of iron dust from the mild steel rod on which the coating was implanted. This material in 0.2 gram amounts suspended in saline solution was introduced intraperitoneally into six rats, using the technique described by Miller and Sayers,[18] and extensively used by us in other experiments.[20,21] For comparative purposes, injections were made of similar quantities of free silica, iron oxide and saline solution. Between the 60th and 70th day, these animals were sacrificed and examined. The silica used for control purposes created the expectable typical proliferative nodulation, the iron oxide induced only an inert reaction with deposits on the anterior peritoneal surface in the omentum, on the genitalia, etc.; the collected welding dust proved to exert only an inert reaction closely resembling that from the iron oxide. There was no suggestion of proliferation. It was concluded that dusts from coated rods containing silica or silicates, of the type used in our experiments, are unlikely to constitute any practical threat of silicosis.

Welding on Steel Alloys and Welding Rods of Other than Mild Steel

In arc welding, it is frequently but not necessarily desirable that the constituents of the welding rod conform to that of the materials being welded, that is, both the metal being welded and the rods themselves may give rise to greater opportunity for injury to welders than in the case of essentially pure steel welded with mild steel rods. In addition to alloyed steel, the same type of problem may arise if the metal to be welded previously has been coated, such as in galvanizing. The outstanding example of the adjustment of welding rods to an alloyed steel is to be found in the relatively high percentage of chromium and nickel in welding rods for use on rust-proof steel. Such a rod may present the following approximate analysis:

Carbon	0.05 %
Manganese	1.1 %
Phosphorus	0.031%
Sulfur	0.09 %
Silicon	0.43 %
Nickel	9.4 %
Chromium	19.7 %
Copper	0.06 %
Vanadium	0.03 %
Remainder Iron	

A type of welding rod widely used in welds on cast iron is represented by the following:

Carbon	0.11 %
Manganese	1.70 %
Phosphorus	0.03 %
Sulfur	0.013%
Nickel	53.52 %
Cobalt	38.5 %
Iron	1.2 %
	100.3 %

An analysis of another rod designed for use on chrome-nickel steel has been borrowed from the work of Captain Brown:

Carbon	0.09 %
Manganese	1.64 %
Sulfur	0.005%
Silicon	0.32 %
Nickel	21.14 %
Chromium	26.04 %
Remainder Iron	

In view of the high temperature of the welding arc, almost any constituent of the metal being welded or the welding rod, to say nothing of its coating, may be introduced into the atmosphere to some degree. Among other possibilities are zinc, cadmium, manganese, chromium, nickel, copper, molybdenum, iron, arsenic, aluminum, phosphorus, selenium, silicon, titanium, vanadium and tungsten. Without intending to culpate any of these metals or in the case of coatings, non-metal material, it is pointed out as obvious that many of these substances are dangerous and that some of them are present in the atmosphere about welding operations in quantities sufficient to induce harm.[2] Thus when welding is carried out upon galvanized steel more than 75% of the fume may consist of zinc oxide. In general it is believed that in this domain may be found the truly disturbing situations connected with arc welding. Already some complaints are arising over the possibilities of chromium from chrome-nickel steel; lead may be measured in the atmosphere near to welding on leaded steels.[3] However, as yet there is no reliable case record clearly associating injury with either of these metals in welding. A few deaths have been connected with welding on galvanized steel, but to associate these deaths directly with the galvanizing or any other individual aspect of welding certainly is not warranted at this time.

Practical Applications and Implications

1. In our experience and from our appraisal of the literature, it does not appear that the health of arc welders as a class may be set apart as especially different to metal workers in general. Although occasional records of deaths appear associated with some aspect of welding as the cause, we have no evidence of any prevailing diseases characteristic of the work of the arc welder (radiation effects excepted).

2. In our experimental studies, with the rate of rod burning much higher than in practical welding, and with welding confined to a comparatively small chamber of 1,000 cubic feet, with at times only one air change

per hour, we have not been able to find any suggestion of oxygen deficiency.

3. The much discussed point of ozone formation in the welding arc has in our experience resolved itself as follows:

Much ozone (10-32 ppm.) is produced immediately in the arc, but quickly is diminished (1-9 ppm.) a few inches (4") away from the arc and in the center of the chamber the residual is not more than 1 ppm. of ozone. Although this exceeds the somewhat accepted threshold of injury of 0.4 ppm., no injury from this gas has been determined by us.

4. Numerous tests for carbon monoxide have been made under various conditions—always without any significant results.

5. Other things being equal, low voltage and amperage are desirable in arc welding, since it appears to be true that low voltage is conducive to the low formation of nitrous gases.

6. Our best efforts to produce from arc welding sufficiently high concentrations of nitrous gases to induce injury to animals after long exposure were not successful. Practical arc welding in open areas seldom leads to higher concentrations of nitrous gases than 20 ppm.

7. It is our observation that concentrations of nitrous gases near the widely accepted threshold of 39 ppm. are quite harmless to laboratory animals after prolonged exposure and further that double this quantity for prolonged exposures and treble this quantity for limited exposures likewise are without discernible ill effects on animals. Human beings exposed to such quantities as 84 to 103 ppm. of nitrous oxides for a few hours (three) suffered no ill consequences.

8. We possess some evidence that methomoglobin is formed both in laboratory animals and human beings exposed to welding gases, but the quantity of this methemoglobin appears to be without injurious propensities and further there is theoretical evidence that comparatively low percentages of methemoglobin in the blood may under some circumstances serve beneficently.

9. Limited experimental observation is unfavorable to any belief that silicosis may be brought about from silica dust exposures incident to arc welding.

10. The heavy iron content of the air incident to animal exposure in welding has not been shown, from clinical, x-ray and autopsy observations, to produce any injury beyond pigmentation.

11. Apparently coated rods are to be favored over bare rods insofar as the production of nitrous gases is concerned.

12. Finally, in appraising arc welding as an industrial procedure essential to national defense and with particular reference to hygienic problems we at least are convinced that some over-emphasis in the past may have been placed on the significance of nitrous gases, carbon monoxide, ozone and iron fume. To the contrary, if there be practical hygienic problems related to arc welding (in addition to the obvious ultra-violet emanations) these may be expected in the little explored field of the constituents of coatings on rods, coatings on metals, and special constituents of the metal welding rod or the metals being welded.

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PROFESSOR DRINKER: We welcome any discussion on this paper or questions to Dr. McCord.

DR. SANDER: I should like to ask Dr. McCord what he meant by the possible protective action of methemoglobin formation.

DR. McCORD: I am very glad to go into that, but I do want to confess that nobody can be sure of that situation and instead of relying upon my own offhand comment here I wish to refer to my notes in that connection.

In the first place, methemoglobin may not be highly dangerous in itself. There are several cases on record of familial methemoglobin anemia which, according to methods that were used for testing, and some of those may be doubted, yielded up to forty per cent of methemoglobin in the blood without any apparent disturbance to the owner of that particular congenital defect. In a much larger number of cases the quantity of methemoglobin continually present has been on the order of twenty or thirty per cent.

From the work on methylene blue in the treatment of cyanide poisoning and that type of poisoning, it apparently has been established or may be established that the only good or the chief good that the methylene blue does is to form methemoglobin, and the methemoglobin in turn acts as a direct antagonist to the cyanides.

We have no proof yet that cyanides or nitriles, which are about the same, are produced in connection with welding, but we have some vague belief that such is the case. On the assumption that these cyanides or nitriles may arise we believe that they might be overcome or destroyed because of their combination with or because of an antagonistic action of methemoglobin, which apparently readily combines with cyanides and that type of chemical in the body.

PROFESSOR DRINKER: One of the largest, if not the largest, employers in the country today, in the factory sense, is the United States Navy. The Army will shortly be, if it is not already, in just about the same category. In the production program and the accomplishment of our desire to have a two ocean navy, our navy yards have necessarily become enormous producers and enormous employers of labor. Their industrial hygiene problems are therefore just those that would be created by such a huge expansion necessarily made without as much time and preparation as we would like.

I personally have had the pleasure of knowing the next speaker, Captain Brown, for a number of years. In industrial hygiene matters I first met him in connection with the dismantling of ships and the industrial hygiene problem that that created. Now he is in charge of the medical aspects of one of the largest yards in the world.

It is a great pleasure to us to have here today Captain Ernest W. Brown, of the Medical Corps of the United States Navy.

INDUSTRIAL HYGIENE AND THE NAVY IN NATIONAL DEFENSE

CAPTAIN ERNEST W. BROWN (MC), U. S. N.

1. Introduction

One of the most important concerns of the Medical Department of the Navy today is industrial hygiene and especially in Navy Yard practice. This is a situation of ever increasing moment in view of the present era of enormous expansion in naval construction, unparalleled in the history of this country. This is bringing about a vast increase in the industrial force of our Navy Yards and, in all probability, new problems in industrial hygiene will emerge, incident to new materials and processes.

It should be remembered in this connection that the policy of the Navy Department is to allot new naval construction on an equal basis to government and commercial yards. It follows that the commercial establishments are also undergoing rapid development with an enormous rise in industrial personnel. They will therefore be confronted with the problems of industrial hygiene similar to many of those arising in Navy Yards.

Industrial hygiene is a field which is now undergoing rapid development. This appears to be due to certain significant trends, the most important of which has been the recent setting up of many Industrial Hygiene Units in state or city Departments of Health through funds released by the passage of the Social Security Act. These trends, in fact, particularly that just mentioned, reflect a definite renaissance of industrial hygiene as a phase of public health in the United States.

This movement is receiving increasing recognition in naval industrial circles, and industrial hygiene is now listed as a specialty of the naval medical officer along with other specialties outside of the purely clinical fields, such as aviation medicine, submarine medicine, chemical warfare medicine, etc.

Those just mentioned, however, are concerned primarily with naval personnel. Industrial hygiene, on the other hand, is largely occupied with federal industrial personnel. It therefore follows that the status of the Senior Medical Officer of a major Navy Yard in relation to the Industrial Department is analogous in many respects to that of the Medical Director of a large commercial industrial plant. The object of this paper is to present an outline of the administration of industrial hygiene in Navy Yards which are the chief industrial units of the Navy.

Mention should be made in this connection of the Sub-committee on Industrial Hygiene of the Health and Medical Committee of the National Defense Council which has recently been established. The Surgeon General of the Navy is represented by liaison officers in conjunction with this Sub-committee. Important recommendations pertinent to the Navy and industrial health will result and many of them will undoubtedly be put in effect.

The term, industrial hygiene, as applied in the present discussion, is used in the specific sense of the prevention and control of occupational disease. The fact may be of interest that the first compensation law for occupational diseases in this country was one passed in 1908 by Congress for U. S. Civil Service employees. Compensation laws for industrial diseases have lagged far behind legislation covering accidents. Only 16 states of the Union pro-

vided compensation for one or more occupational diseases up to the year 1937, although all but two provided legislation for accidental injuries.

2. The Industrial Organization of Navy Yards

The mission of a Navy Yard is primarily the construction, maintenance and repair of naval vessels. The central administration of Navy Yards, and in fact, of all industrial shore stations of the Navy, is vested in the Assistant Secretary of the Navy, in whose office is the Shore Establishments Division of the Navy Department.

There are eleven Navy Yards located as follows: Portsmouth, N. H.; Boston; New York City; Philadelphia; Washington, D. C.; Norfolk, Va.; Charleston, S. C.; Mare Island, Calif.; Puget Sound, Wash.; Pearl Harbor, Hawaii; and Cavite in the Philippines.

In addition, mention should also be made of the following specialized industrial plants: for the building of submarines at Portsmouth, N. H. and Mare Island, Calif.; the Naval Gun Factory at the Navy Yard, Washington, D. C., for the production of high caliber naval guns, torpedo tubes and accessories; the torpedo factories at Newport, R. I., and Alexandria, Va., for the manufacture of torpedoes; the powder plant at Indian Head, Md., for the production of Navy smokeless powder; the aircraft factory at Philadelphia for experimental air craft construction and repair; the naval armor plant at Charleston, W. Va.; and the Naval Clothing Factory at Brooklyn, N. Y.

Organization of the New York Navy Yard: The organization of the New York Navy Yard may be taken as typical of a major yard. It falls under two departments, i. e., the Industrial Department headed by a naval captain of the engineering branch and an Operations or Military Department under the direction of a naval line captain. As a conservative estimate it may be stated that 90% of the activities of a Navy Yard are industrial.

Under the Industrial Manager there are at present twenty-three shops of different types with a force per shop varying from 30 to 3,200 men. The total of civil employees of this yard is now 17,000. This is rapidly rising and, it is estimated, will exceed 20,000 in 1941.

3. Extent of the Civilian Industrial Force of the Navy

The combined industrial force of all Navy Yards is now approximately 100,000. In view of the pending program of naval construction it is estimated that this will reach 130,000 in 1941. If inclusive of all shore stations this would probably be close to 160,000.

In addition to the industrial force of Navy Yards we should also consider the employee volume in the commercial naval ship building plants such as the Newport News and Dry Dock Co., the New York Shipbuilding Corporation at Camden, N. J., and the Bethlehem concern at Quincy, Mass., which now employ from 12,000 to 15,000 men each. It is a conservative estimate that the combined industrial personnel of all such plants on both east and west coasts will reach a peak of over 100,000.

4. Organization of the Medical Department of a Navy Yard

The medical staff of the New York Yard consists of 10 medical officers, 5 dental officers, 1 nurse, 45 enlisted men and 2 civilian clerks. The chief activities with reference to industrial personnel may be summarized as fol-

lows: (a) Pre-employment physical examinations: all applicants for federal jobs are examined physically, although the standards for acceptance vary to some extent for different occupations. (b) Periodic physical examinations: these, of course, are conducted with the object of medical supervision of certain groups of employees exposed to definite potential health hazards, as foundrymen and spray painters. (c) Physical examination of federal employees for retirement: this is for evaluation of degree of disability and opinion as to disposition when total disability is alleged. (d) Diagnosis, treatment and disposition of industrial injuries and occupational diseases. (e) Administration of compensation cases. (f) Industrial hygiene and plant sanitation.

5. The Industrial Medical Officer

An officer of the medical staff of the Navy Yard is specifically detailed for industrial hygiene administration subject to the direction of the Senior Medical Officer. The accompanying slide outlines the scope of his activities.

(a) Advisor to the Safety Engineer: The adequate practice of industrial hygiene in Navy Yards, as in civil industry, is dependent upon the close and efficient correlation of the safety engineer and the industrial medical officer. It is essential to obtain a grasp of the functions of both of these officers in order to properly visualize industrial business administration.

(b) Inspections: The industrial medical officer is responsible for the general supervision of plant sanitation, i. e., ventilation, illumination, water supply, general cleanliness, adequacy and condition of sanitary facilities, etc. He also conducts shop inspections for occupational health hazards and their measures of control.

He cannot expect to evaluate working conditions and thereby detect occupational health hazards early unless he makes periodic inspections through the plant. In this way he can observe the adequacy of existing measures against specific hazards and whether such methods are being properly utilized. These inspections also have a psychological value in that they create greater respect for the medical service in the minds of the employees.

(c) Supervision of Special Physical Examinations: pre-employment cases when the applicant reports previous exposure to potential industrial health hazards such as lead fumes or foundry dust; periodic examinations of groups exposed to such potential occupational hazards, as spray painters and sandblasters; examination of cases referred for transfer to other shops where there is a question of occupational disability; clinical studies for a decision as to industrial origin in obscure cases.

(d) Medical surveys of occupational groups; this will be discussed later.

(e) Administration of the medical aspects of compensation claimed for occupational disease pending before the U. S. Employees Compensation Commission.

(f) Supervision of preparation of accident and occupational disease reports for the Navy Department.

6. The Safety Engineer

A civilian Safety Engineer is stationed at the Navy Department as the advisor to the head of the Shore Establishments Division. A naval officer is assigned to each Navy Yard as the safety engineer.

(1) Accident and unsafe practice control: Safety engineering is one of

the divisions of the Navy Yard organizations. The Safety Engineer conducts an investigation of all lost-time accidents with a view to fixing the cause and advising as to measures to prevent their recurrence. The basic features of approach to the safety problem in Navy Yards are provision of safety devices, such as mechanical guarding, and the safety education of workmen and their supervisors.

Another important aspect is the competitive approach which has proved very effective in stimulating interest in accident prevention. The Navy Department publishes the comparative safety scores of all Navy Yards monthly.

The accompanying slide emphasizes the advance made by the Navy Department in accident reduction beginning with an intensive safety campaign in Navy Yards in 1926. We find the frequency accident rate lowered from 20 to practically 10 per year in a 12 year period; the severity rate reduced from 2.2 per year to 0.5. On the other hand it will be noted that the total man hours worked during the period increased to 115 million from 65 million per year, the average number of employees rising from approximately 30,000 to 66,000.

(2) Occupational Health Control: The control of occupational disease in Navy Yards naturally lies within the sphere of both the safety engineer and the industrial medical officer. Although the safety engineer is administratively charged with this task, the medical officer is actually coordinate with him in this phase.

As a matter of fact, the medical officer is the key man in the prevention of industrial disease in Navy Yards, in that he usually discovers its existence. The diagnosis having been made, the occupational history and the pre-employment examination record are carefully reviewed in order to reach a decision, if possible, whether the hazard can be traced to present or past employment. If ascribed to, or aggravated by environmental conditions, the medical officer confers with the Safety Engineer and an industrial hygiene survey is usually recommended to the Commandant.

7. The Industrial Hygiene Survey

Such a survey is of course a combined medical and engineering task.

(a) The Medical Survey.

This consists of a complete clinical study with detailed occupational histories of all exposed personnel as a case finding procedure under the supervision of the industrial medical officer. Although not directly responsible beyond the matter of the medical survey, it is important that the medical officer have a reasonable grasp of the entire problem so that he will be in a position to utilize all data that have any bearing on the interpretation of his medical findings. In addition, he may act in an advisory capacity to the safety engineer with respect to certain technical phases in the planning and conduct of the engineering aspects of the survey.

(b) The Engineering Survey.

The engineering phases of an industrial hygiene survey in a Navy Yard fall, naturally, under the direction of the safety engineer. As in a commercial industry, this embraces essentially a complete story of the occupational duties, physical conditions of work, and the materials, processes, and equipment of the individual shop, in other words, the environmental conditions.

Laboratory facilities: There are no facilities provided for technical

studies in the Navy Yard organization and there is no central laboratory unit in Washington which could supply industrial hygienists for field studies. This is an urgent need and recommendations have recently been made to the end of setting up such an agency.

The Department has been most fortunate in the past in securing the services of the Division of Industrial Hygiene of the Public Health Service to conduct such technical studies much in the same way that industries in the states utilize the facilities of State Bureaus of Industrial Hygiene.

The safety engineer formulates the control program of the health hazard on the basis of the data of the survey. After all, once the etiology is disclosed prevention of occupational disease is largely an engineering problem.

8. The Reporting of Industrial Accidents and Illnesses

An important advance in accident prevention by the Navy was initiated on July 1st of this year when the Secretary directed that a report of each accident and illness, both "lost-time" and "no lost-time," occurring among civil personnel of Navy Shore Establishments be made to the Bureau of Medicine and Surgery. The report of each accident is submitted on a form, known as F-C, shown in the accompanying slide. This presents the diagnosis of the injury and the essential details as to the cause. Punch cards are made up from these records for mechanical tabulating and indexing through a sorting machine. This provides facilities for the statistical analysis of these accidents and diseases, and the data obtained promise to be a far-reaching contribution to the subject of accident control. Prior to this system a crude form of accident reporting to the Department was in effect but it was not adapted to statistical analysis.

9. Occupational Health Hazards in Navy Yards

The chief potential occupational health hazards in Navy Yards will now be considered, the data being based chiefly on reports to the U. S. Employees Compensation Commission over a series of years. It hardly requires emphasis that industrial medical officers must be constantly on the alert for new hazards.

(1) Dust Diseases.

(a) Silicosis: Important units of all Navy Yards are iron, steel and brass foundries.

The dust control problem is a major concern in these plants as in civil industry.

One of the difficulties met with in combating the foundry dust problem in navy yards is the fact that silicosis dust is not particularly irritating or obnoxious in concentrations which may ultimately lead to pulmonary damage. As a result we find a tendency to an indifference on the part of the workers and even supervisors and executives, which must be overcome in order to accomplish effective and permanent dust control. Another reason for this attitude is the long period of time necessary for those foundry workers exposed to only moderate concentrations of dust, such as molders, to develop silicosis.

A medical survey of the foundries of the Navy Yard, Washington, D. C., was conducted by the speaker in 1939: of 525 men x-rayed, approximately 60% had a record of 10 years or over; and 36% a record of 20 years or over

of total foundry employment. Twelve cases of silicosis were found or 2.4% of the total—all in Stage I or II; these data leading to recommendations for improved dust control methods.

Industrial hygiene surveys in foundries other than the Washington Navy Yard have not as yet been carried out but would, in all probability, disclose a certain incidence of silicosis, even if not of disabling type.

The medical control of silicosis in the naval establishment consists of an x-ray of the chest before employment and an annual radiograph of all men exposed to the higher silica dust concentrations such as sandblasters and shake-out men. It is believed that medical surveys of foundries in all Navy Yards will soon be required.

(b) Asbestosis.

This is a potential occupational disease hazard from the inhalation of asbestos dust among workers engaged in the manufacture of asbestos insulating covers for flanges, valves and high temperature steam turbines.

The speaker recently conducted a medical survey of the workers of the Pipe Insulating Shop of the New York Yard inclusive of x-ray studies, the maximum working period of exposure being 17 years. No asbestosis cases were found. Similar findings have been reported from two other yards but the study should be extended to all men in this trade.

Medical control consists of an x-ray of the lungs annually. The asbestos is moistened and localized exhaust ventilation is installed over the work area. A respirator is worn during the dustiest aspect of the process.

(2) Lead and Lead Compounds.

Lead poisoning has become comparatively infrequent in recent years both among industrial and service naval personnel; apparently due in part to changes in materials and methods, and partly to improved measures of control. Zinc and titanium paints have largely replaced leaded material for ship interiors. Red lead paint is still in use as the priming coat on hull exteriors but the finishing coats contain either no lead or a greatly reduced proportion. All painters regularly handling lead-containing paint are examined semi-annually for evidences of lead absorption.

(3) Volatile Organic Solvents.

Lacquer spray painting occurs on an extensive scale in Navy Yards and therefore demands rigid medical supervision. Another important application is in degreasing measures. All spray lacquer personnel are subject to semi-annual physical examination.

(4) X-ray and Radium Hazards.

The x-ray and radium are continually utilized for the detection of flaws in castings and pipe welded joints for high pressure steam installations. Radium has an advantage in the small size of the equipment in that it is adapted to tests in the confined machinery spaces of ships.

The question of protection from irradiation of the operating and other personnel working in the vicinity of the apparatus has received thorough study, the practice of the Bureau of Standards being generally followed. Complete blood counts of all technicians are conducted quarterly and special pre-employment examinations are prescribed.

Another potential hazard of radium is that of ingestion incident to radium painting of luminous dials, more especially for fire control instru-

ments and aircraft. The control measures advised by the Public Health Service are generally in force, plus certain local regulations.

(5) Welding.

The hygienic supervision of welders is another outstanding feature of medical responsibility. Approximately 2,500 welders were on the rolls of the combined shore establishments as of January 1, 1940, including 653 at New York. This number has progressively increased and will continue to rise.

The immense volume of work in confined spaces is characteristic of naval welding. A battleship of 35,000 tons displacement under construction contains approximately 500 compartments in which electric welding is mandatory, certain of these spaces being extremely small and forcing the welder to work in very cramped positions. These conditions complicate the question of effective preventive control of the hazards.

The chief hazards which have to be considered at present are "nitrous fume" poisoning, zinc fume fever, as it is popularly termed, and actinic ophthalmia from ultraviolet radiation of the welding arc. "Nitrous fume" poisoning, while comparatively rare, is a serious condition.

No accent need be placed on the fact that these injuries would be still further reduced in number if the control measures provided were properly utilized. It may be of interest to note that of 317 cases of actinic ophthalmia reported at the New York Yard in 9 months of the current year, only 20% occurred among welders; the remaining 80% among men exposed in spaces adjacent to the welding arc but not utilizing available protective goggles.

Chronic poisoning among naval welders from manganese, fluorides or silicon, which might be ascribed to inhalation of these metallic or mineral oxides in the welding fumes originating in the rod coatings, has not been reported. Such a possibility, of course, cannot be denied. The limitation of time prevents mention of additional hazards.

10. Annual Examination of Crane Operators, Enginemen (Hoisting and Portable) and Locomotive Engineers

These classes of workers are physically examined annually with special emphasis on blood pressure, hearing and vision. In view of the nature of their duties, physical failure, such as sudden collapse, might involve critical injury to themselves and others; and in addition, damage to material. If corrective measures are impracticable the worker is retired or transferred to some other type of employment for which he may be suitable. A crane operator, for instance, presenting a marked hypertension would be referred to his private physician and transferred to other duties.

11. Time Loss from Industrial versus Non-Industrial Disabilities

In a limited study of 116 industrial companies in various parts of the United States conducted by the American College of Surgeons a few years ago, it was found that the time loss from non-industrial types of illness was approximately 15 times that industrially connected. The speaker in his capacity as Senior Medical Officer of the Washington Navy Yard made the following observations for the calendar year 1933: Total industrial force, 7,000; number of days lost from industrial accidents, 0.14; number of days lost from non-industrial illness or accident, 5.20. The number of days loss from non-industrial disability was therefore 37 times that from industrial

causes. A similar study by the speaker at the New York Yard for 1939 revealed that the non-industrial loss was roughly four times the industrial figure. The possible factors in the difference between the two yards have not been analyzed.

Disparities of the same general order have been reported in recent statistical studies by certain large commercial industries and reflect an enormous economic loss. Much of this non-industrial illness is preventable. It is believed that in the naval establishments this wastage of preventable illness can be greatly reduced, if the problem is attacked by an annual physical examination of all employees, referring cases requiring corrective treatment to their private physicians or other agencies. This would require a heavy increase in medical staffs but it would be a profitable investment by naval industry in the saving of man power for national defense. It is a question worthy of being explored.

PROFESSOR DRINKER: We were informed the other day in Washington that the Navy is now in the mining business and is confronted with the conventional mining hazards as well. Therefore we can say that their hazards are about all those we meet in all of our industries.

I am sure that Captain Brown will be glad to answer questions.

MR. WEIL: I should like to ask Captain Brown what his eye test consists of for his men in so-called key positions. We are using a keystone and we are a little at a loss as to how to interpret some of the findings.

CAPTAIN BROWN: I didn't get the drift of that.

MR. WEIL: I want to know what the eye examination consists of and what is your experience in interpreting the keystone test, if you use it.

CAPTAIN BROWN: I don't think we have had any contact with that particular test. We simply use the ordinary southern test, with definite standards.

PROFESSOR DRINKER: A teacher always judges the value of a book, first, by his ability to keep it in his own library or in his own laboratory. They have a way of disappearing, particularly if your students are eager for it. One also judges the value of a book by the general appearance of the cover, whether it begins to fall apart after a month or two.

Haggard's "Noxious Gases" is the standard book throughout the English-speaking world on the subject. I am on my fourth copy. I don't know how the rest of you are doing. Two of mine have been stolen, one is worn out, and the one I have has been rebound once. The University also has a number of them at the Medical School and at the Engineering School Libraries. I state this to show you how eager we are to use it and how much we do use it.

It is pleasant to welcome to our group—at least it is for me as a professor at Harvard—a professor in a sister university and one with whom we have friendly rivalry of all sorts. In this case I have to admit, with a good deal of professional jealousy and very sincere admiration, that there isn't any rivalry. The lead is entirely with Yale.

It is to me, therefore, a great personal privilege to be able to introduce to you Dr. Howard W. Haggard, of Yale University, who will speak on the "Absorption and Excretion of Volatile Solvents."