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Dusts and Fumes in Industry

(Sources, Effects and Control)

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Introductory Statement

In dealing with occupational diseases and their effects, the attending physician should keep in mind that the etiology of these conditions is present in the industrial environment — if the conditions are to be considered truly industrial in origin.

It is likewise important to understand that rational diagnosis and treatment rests upon a fairly accurate knowledge of the etiology of the disturbances; this is especially true of occupational diseases and allied disorders.

Moreover, employers, industrial hygiene and medical departments are greatly interested in the prevention of various maladies which ordinarily affect employees; this clinical and economic interest on the part of industrial management further emphasizes the need for understanding the etiological factors in the working environment, so that they may be properly controlled.

This booklet on industrial dusts and fumes is the second of a series of which the purpose is to show the most important effects of occupations, show how these occur, give measures for treatment, diagnosis, and prevention, meanwhile considering the source or cause of such conditions. It is hoped that these simple and brief expositions will be of considerable help to industrial physicians, and to private practitioners who have among their patients employees of industrial plants subject to such exposure.

Definitions

Dusts may be defined as solid particles of matter which are ordinarily produced by grinding, crushing, drilling or blasting

processes, the composition of the particles being the same as that of the parent materials. These materials may be rock, metals, ores, or such products, for example, as coal, grains, or wood. The size of the particles varies from what is called ultra-microscopic to those which are visible. Authorities now agree that the hygienic significant particle size of dust ranges from a fraction of a micron to 10 microns. (A micron is 1/25,000 of an inch). The most frequently encountered dusts in industrial processes are mainly those from inorganic minerals, and a few organic dusts, such as wood and flour.

Fumes may be defined as solid particles which are formed by processes similar to combustion, condensation and sublimation. The most common types of fumes encountered in industry are usually caused by the oxidation of a metal, of which typical examples are the oxides of lead or zinc. Particle size is usually smaller than dust and ranges below one micron. In contrast to dusts, fumes tend to flocculate very easily. Many people erroneously speak of fumes when they mean vapors of liquid materials, such as acid solutions, organic solvents, and the like.

Incidence and Etiology

Dusts are the most common type of air contaminants encountered in industrial processes, and can occur whenever various materials, as mentioned before, are subjected to processes which break them up into fine particles; or where powdered or pulverized materials are handled or transported. Inorganic dusts are the prevailing type so far as frequency is concerned, but organic dusts are also found in different industrial operations.

Fumes are usually found wherever there are molten metals such as lead, zinc, and calmium being processed or used in industrial operations. Welding is an operation which has greatly increased in frequency of use and it may give rise to fumes of the metals which are welded or to fumes and gases from the variegated coatings of welding rods.

It is to be borne in mind that metals may produce both dusts and fumes, according to the nature of the operation and the state of the materials when processed. For example, among the heavy metals such as lead, dust may be of equal or greater etiologic importance with fumes — other mineral dusts and metallic dusts also are considered of etiological importance, as the following outline will show:

I. *Organic dusts:*
   1. Non-living organic dusts:
      (a) Toxic and/or irritant dusts; "(Various organic compounds such as trinitrotoluene, mercury fulminate, tetryl)."
      (b) allergy-producing dusts (woods, pollens, and the like).
   2. Living organic dusts:
      (a) bacteria
      (b) fungi

II. *Inorganic dusts:* (mineral dusts)
      (a) toxic and/or irritant dusts; (mainly the dusts from heavy metals and their salts, such as arsenic, lead, manganese.)
      (b) fibrosis-producing dusts, (examples are free silica and asbestos).
      (c) non-fibrosis-producing dusts; (the so-called inert dusts, such as alundum, corundum, emery, limestone, magnesite, marble, plaster-of-paris, and polisher's rouge).*

The most important of the non-metallic, inorganic mineral dusts is free silica, which produces silicosis, if breathed in sufficient quantities over a long period of time. Free silica dust and

* This classification is in accordance with the present state of knowledge. Future studies and experience will probably show some of the so-called inert or nuisance dusts to be harmful.

asbestos dust are the two kinds of mineral dusts which produce disease entities corresponding to the names of the dusts — silicosis and asbestosis. Mixed dusts of various kinds, such as various ores which contain different amounts of free silica, can produce different types of reactions, according to the kind of minerals which are in the ores. Where, for example, there are mixtures of iron, and various calcium-containing compounds such as gypsum, with free silica, a protective influence is exerted against the action of free silica on the lungs. Other modifications of free silica action occur according to the types of minerals present some of which tend to inhibit or restrain the action of free silica.

Another important conception with reference to the etiology of dust and fume diseases which may result from exposure to these classes of materials, is the use of the terms *exposure* and *hazard:* they are not synonymous — exposure merely means contact with some substance which is a *potential health hazard;* whether it is an *actual health hazard* can only be determined by an industrial hygiene engineering survey, in which the amount and kind of exposure is rated, according to accepted standards of concentration, size, and quality of dusts and fumes. Many persons erroneously therefore state that there is a lead hazard, for example, when it is only known that contact is being made with lead; and when there are no data to support the contention that there is an actual hazard present in the working environment.

Methods for the determination of dusts and fumes in the air, also with reference to the types of devices used for such determinations, will be mentioned later. The importance of air contamination will be realized when it is stated that the pathologic effects produced by dusts and fumes are brought about almost entirely by the inhalation of these compounds and materials. There is only one outstanding example, for instance, of the possibility of absorption of lead through the skin, namely tetraethyl lead, and this occurs in the form of a fluid. No dusts, so far as is known, produce any systemic pathological condi-

tions from skin absorption, and it is doubtful that any kind of true fume is absorbed by the skin.

From the etiologic point of view, particularly with reference to pathologic effects, the following outline shows how different types of substances (among the dusts and fumes) act on various systems in the body to produce pathological effects:

1. *General systemic poisoning:*
   Examples: Arsenic, lead, manganese, mercury.
2. *Substances acting on the circulatory system:*
   Examples: Arsenic, lead, mercury, vanadium.
3. *Substances acting on the respiratory system:*
   Examples: Asbestos, free silica.
4. *Substances acting on the gastro-intestinal system:*
   Examples: Antimony, cadmium, lead, mercury, zinc.
5. *Substances acting on the skeleton and joints:*
   Examples: Lead, mercury, mesothorium, phosphorus, radium.
6. *Substances acting on organs of special sense:*
   Examples: Arsenic, lead, manganese, mercury.
7. *Substances acting as fetal poisons:*
   Examples: Lead, mercury.
8. *Substances acting on the genito-urinary system:*
   Examples: Arsenic, lead, mercury, vanadium.
9. *Substances acting on the brain and nervous system:*
   Examples: Arsenic, lead, manganese.

General Diagnostic Methods

The accurate diagnosis of pathological conditions arising from the inhalation of industrial dusts and fumes has tremendous economic significance and also it is of medicolegal importance. Moreover, it is impossible to understand how proper and effica-

ceous treatment may be rendered unless a scientific diagnosis is made. If a claim results from the pathological condition alleged, it is evident that the disease sustained must be directly related to the employment and further, must produce disability to entitle the individual concerned to compensation.

Diagnostic methods with reference to dust and fume diseases need not differ widely, if at all, from those used as routine procedures in general scientific medicine, although there are specific methods as in any other application of general medical diagnostic principles to any special group of persons. New discoveries occur relative to diagnostic methods in occupational disease work and for the most part, reliance must be placed on information which is scattered throughout the current literature in addition to available textbook material which is better now than it was even five years ago.

Emphasis has already been placed upon the vital part played by etiology; it is wise to repeat that etiology forms the bulwark of the diagnostic and therapeutic background.

Among the methods which are used to make a fairly accurate diagnosis with reference to occupational diseases, is the taking of a thorough industrial history; consideration of symptomatology, the physical findings, the results of air examination; and finally, last but not least, a differential diagnosis, ruling out diseases of ordinary life which have no relation to occupation.

The industrial history may be of very great assistance in arriving at an opinion. Most industrial physicians of considerable experience believe that it is desirable to include a complete resume of the kinds of occupations which have been experienced by the individual being examined, dating from the first job to the last one. It is of course clear that the details of actual occupational exposure cannot be evaluated by this method, but important information bearing on the diagnosis may thus be obtained. It is the present practice, therefore, to list the occupations with the dates of occurrence, in the industrial history. It is

furthermore advisable to get more detailed information relative to the performance of the occupation, if that can be obtained. This will at least give information relative to the kind of exposure in industry, although it will give no idea of the severity of exposure.

The symptomatology in many kinds of occupational disease is likely to be confusing rather than useful, because in many instances it so closely simulates that of non-industrial disease that it is quite commonly not characteristic. Especial attention should be given, however, to the recording of the symptomatology and critical attempts made of its proper evaluation. Here again, distinction must be made between symptoms which may be due to non-occupational disease and those symptoms which are specific for occupational diseases — this is where the difficulty arises in adequately assessing symptomatology.

The physical findings, on the whole, are more reliable than the symptomatology, although in certain instances they too are not characteristic and are, therefore, liable to be somewhat puzzling. Where only one portion of the body is affected, as in silicosis, for example, it might be assumed that the physical findings would be more characteristic, but the opposite of this is usually true. In other instances, for example lead poisoning, where different organs and systems are pathologically affected, the picture is liable to be one which demands expert evaluation to differentiate from non-occupational conditions.

Clinical laboratory data, including X-ray findings, are frequently found to be decisive, although here again intelligent appraisal means proper interpretation of the data. When they are decisive, such tests are by the very specificity of their nature, of great value as diagnostic aids, although they should be definitely correlated with other findings.

Differential diagnosis presents perhaps the most difficult problem of all in the realm of diagnosis of occupational diseases. There is good reason to believe that differential diagnosis will

continue to be one of the most difficult phases of the medical ramifications of the medicolegal problems of occupational diseases. The reason which has already been indicated is that the symptomatology and frequently the physical findings imitate so closely the symptomatology and physical findings of non-industrial diseases, that one must make use of specific procedures which are designed especially as diagnostic guideposts. Frequently such specific procedures are not used to any great extent, except by those who are highly specialized in the field and whose work takes them into a review of the current literature containing these developments. For example, one may believe that an industrial employee has lead poisoning, largely because of second-hand knowledge that lead was used in his occupation, more easily than one could demonstrate adequate etiology and the existence of lead poisoning by scientific methods.

Because of all of these obvious and other difficulties, it seems wise to adapt specifically certain examination procedures to industrial problems arising out of occupational or suspected occupational diseases. Most authorities now agree that the examining physician can be of greater service and give more efficient advice regarding either acceptability for employment, continuance in it, opinion on the diagnosis of systemic pathologic conditions and the estimation of disability, if he is conversant with the following points:

1. The nature and extent of the industrial exposure into which the employee will go or where he has been. This can be determined only by scientific methods which will be referred to later.

2. The effect of this specific exposure upon normal and abnormal physical states of employees, as interpreted by the physical status of each individual on examination. (This refers specifically to etiology in the industrial environment and the effects which such etiology may or could produce.)

3. The nature of the protective devices supplied by the employer, if a hazardous exposure cannot be otherwise controlled, and as to whether these devices are functioning efficiently. (Reference will be made later to these devices, especially designed for protection against dust and fume exposures.)

It is realized that these procedures are somewhat removed from the usual program of physical examinations, particularly with reference to the work of part-time industrial physicians, but it is also expected that the physician will be called upon increasingly to know and appreciate such information so that he may function better as a diagnostic medium.

Because the most frequent and most significant exposures in industrial processes are free silica dust and lead and its compounds, it seems advisable to include in the next section some clinical examples, namely silicosis and lead poisoning; there will also be included a brief discussion of metal fume fever.

Clinical Examples

Simply stated, silicosis is a disease of the lungs, caused by the continuous inhalation over a long period of time of very finely divided, microscopic, particles of free silica dust. There have been various standards set up by different agencies, based upon studies of silicotic workmen and their exposure to free silica dust. The National Silicosis Conference Report on Medical Control of Silicosis says: "There is evidence that for prolonged exposure a concentration of more than five million particles per cubic foot of a highly siliceous dust is dangerous. Therefore, it is now considered good practice to hold concentrations of highly siliceous dust at 5 million particles per cubic foot, or less."

Because of the fact that industrial exposures, involving high free silica content in which the concentration of dust should be restricted to five million particles per cubic foot, are relatively infrequent in industry, and also because the opposite usually

occurs (an exposure to "mixed" dusts which contain appreciable amounts of free silica but also contain considerable amounts of other types of material) it is believed wise to refer to the formula* developed by the National Silicosis Conference with reference to a working criterion with respect to satisfactory or unsatisfactory conditions, when exposure to 'mixed' dusts occurs.

It is to be borne in mind, however, that any unprotected industrial operation, which involves the production of a lot of fine dust particles of material containing a relatively large percentage of free silica, offers a potential silicosis hazard.

The incidence of silicosis is not known with any degree of accuracy, although various estimates have been made. One of these made some time ago by the American Institute of Mining and Metallurgical Engineers indicated that in the United States the number of industrial workers exposed to the silica dust hazard is in excess of a half million. Figures quoted by the Committee Reports of the National Silicosis Conference disclosed estimates that approximately 2% of the working population of the United States or about one million workers are exposed in some way to the potential hazard of silicosis, and that perhaps half of this number, 500,000, or 1% of the total, are exposed to a serious hazard. It is furthermore believed that approximately 110,000, or 0.2 of 1% of the total, have silicosis to some degree, but likely only 4000 to 5000 (a small fraction of 1% of the total) suffer any degree of disability from the disease. (These figures are based upon prewar estimates.) It can readily be seen that silicosis, although not a rare disease, is not common and furthermore not as common as may ordinarily have been supposed by many persons.

Recent conceptions with regard to the contraction of silicosis have established the belief that approximately 25% of those exposed to a real silica hazard will eventually develop silicosis.

* FORMULA A: "Multiply the percentage of free silica by the total dust count. If the result is under 5 million, the condition may be considered permissible. If the result is over 5 million, the condition may be considered too high. For example, 10 per cent free silica with an average total dust concentration of 30 million particles per cubic foot would give 0.10 times 30 million, which equals 3 million (good practice); 30 per cent with an average total dust concentration of 90 million, would equal 0.3 times 50, or 15 million (unsatisfactory). This formula is not applicable to any dust containing less than 5 per cent free silica."

With respect to the occurrence of silicosis in the foundry industry, a prewar survey by the Division of Industrial Hygiene of the New York State Department of Labor, based on a study of 311 foundries, of which 80 plants included X-ray examinations involving 4,754 employees, provides some interesting figures: the X-ray summary showed that 3,259 or 80.2% were negative; 185 or 4.5% showed fibrosis; 110 or 2.7% showed silicosis with and without infection; 485 or 11.9% showed healed primary and re-infection types of tuberculosis; 27 or 0.07% showed clinically significant tuberculosis. It was concluded, as stated in the report, that it would appear that the silicosis hazard in the foundry industry in New York State, while existent, is of mild degree of severity and may be expected to yield satisfactorily to appropriate measures of control. The report further indicated that tuberculosis as manifested by X-ray films of the chest was found to be more prevalent among silicotics than among non-silicotics in foundrymen.

The occupational history, as previously indicated, is of considerable importance. Forms for the recording of occupational records have been devised by various organizations and groups and by physicians and insurance companies. One of the most complete and longer forms has been devised and used by the Saranac Laboratory, Saranac Lake, N. Y., in connection with silicosis studies and symposia on silicosis which have been held by this group. It is suggested that the physician, if interested, should contact the industrial hygiene bureau of his state health department through which detailed forms may be secured or information relative to them.

The symptoms and physical signs are not characteristic; they may be confused even by skilled diagnosticians in other diseases, with the symptoms and signs which commonly occur in both minor and the more serious types of the well-known respiratory infections. Characteristically, shortness of breath on exertion and decreased chest expansion frequently occur in advanced cases of silicosis, but these findings must also be differentiated as

to their casual relationships and their possible association with other diseases.

The clinical confirmation of the diagnosis of silicosis rests mainly on the characteristic X-ray appearance of the chest. This shows the typical lesions of silicosis, hyaline fibrous nodules which are scattered as opaque bodies throughout both lung fields, the density of these nodules being somewhat less than calcified bodies, but discrete in outline, all about the same size in the early stage (so-called millet-seed size) and evenly distributed throughout both lung fields. These nodules appear as the first indication of silicosis, later become somewhat confluent and in the latter stages, there are large masses of opaque material found near the periphery of the lung fields. In the later stages of silicosis, the disease is frequently complicated by infection, usually tuberculous in nature. As an aid to interpretation of the X-ray films of the chest where silicosis may be suspected, it is suggested that the physician refer to a tabulation of the X-ray changes in silicosis, with the underlying histological conditions which cause them, published in the transcript of the Second Silicosis Symposium, Saranac Lake, N. Y., 1935.

Diagnosis rests upon proof of occupational dust exposure sufficient to produce the disease, and the presence of characteristic nodular lesions in the X-ray film of the chest. The mere mention of occupation is not sufficient for diagnostic purposes — adequate knowledge concerning the quality and quantity of dust exposure, as well as the length of the employment period, is necessary to establish a distinct causal relationship.

Although considerable diagnostic reliance is usually and properly placed upon the history of adequate occupational dust exposure and the characteristic chest X-ray appearance, a thorough physical examination with appropriate clinical laboratory examinations should be done, especially with reference to the ruling out of other respiratory diseases. Diagnosis becomes essentially a differential diagnosis, as before explained, one which is likely to be difficult, necessitating special experience

and knowledge. Some of the conditions requiring differential consideration here are miliary tuberculosis, multiple calcifications of healed tuberculosis, cancer of the lungs, and fungous growths of the lungs.

Within recent years, superimposed infection has come to be the accepted cause for disability. The outlook in simple silicosis, therefore, (that is, without infection) is more favorable than was formerly believed. It is obvious that the chief problem for the silicotic is to avoid superimposed infection, which is usually tuberculous.

There is no one specific, efficacious treatment for silicosis, although in recent years, the inhalation of metallic aluminum powder has shown some promise. In cases where tuberculosis complicates the picture, it is obviously to be treated by the best known methods for that disease. There is some recent evidence that surgical therapy as used in ordinary forms of tuberculosis is also a help in the type of tuberculosis which follows silicosis. The evidence for this is not conclusive, but there are good results on record.

In general, recommendations for the treatment of cases of silicosis vary with experience and with the findings of each case. It is believed, for example, that no treatment is necessary in the case of an individual without symptoms, whose X-ray film of the chest (taken perhaps in a routine industrial survey) shows only widespread discrete nodulation without localized shadows indicating infection. However, the employee should have serial X-ray examinations with physical examinations of the chest at least once a year and should be removed from further silica exposure to some other job, since the disease is very slowly progressive and there is at this stage no disability which precludes continuation in employment. A person with localized conglomerate lesions may have symptoms and usually cannot do as heavy work as formerly; therefore, a lighter job should be found. In the presence of active infection where there are symptoms or incapacitation, the individual cannot obviously continue in his

former employment. If the case is one of open tuberculosis with positive sputum, it is evident that such an employee is a source of danger to his fellow workmen and should be placed in a sanitarium or hospital. In cases of inactive infection with no incapacitation, lighter work should be provided, without further dust exposure.

Dusts containing silica in combined form do not cause silicosis; however, one such dust, asbestos, a magnesium silicate, will produce a characteristic fibrosis which is called asbestosis. This disease is quite different in its pathology from silicosis, and has a characteristic 'ground-glass' appearance on the X-ray films of the chest. Animal experimentation indicates that the action of asbestos is probably mechanical, since it cannot be produced in other than the pulmonary tissue. Asbestosis may cause disability and a few fatal cases have been recorded, but in those cases showing marked disability, other organic disease is usually also present. The symptomatology and physical findings include cough, dyspnea, pallor, clubbing of fingers, and diminished chest expansion, with a characteristic X-ray appearance as previously mentioned.

Lead Poisoning

Lead poisoning offers a good example of an industrial poisoning which is contracted by the inhalation either of a toxic dust or fume, and has been stated to be one of the most frequent and important types of metallic poisoning in industry, on account of the widespread use of lead and its compounds.

Although there are no reliable statistics available with respect to the national incidence of lead poisoning, it is believed that lead poisoning is very prevalent and that most of it is mild. The incidence of severe types of cases, particularly lead encephalopathy and the more severe types of nervous system involvement are not seen with as great frequency as in former years. Many physicians who are used to handling industrial cases now rarely see a case of wrist drop due to lead poisoning, whereas 20 to 25 years ago it was a rather common phenomenon, in cases of lead intoxication.

The industrial history is of great importance, as previously indicated, and a thorough consideration of the nature and extent of the exposure to lead and its compounds is an essential point in the etiology. It is now generally accepted that the standard of the U. S. Public Health Service and the American Standards Association of 1.5 milligrams of lead per ten cubic meters of air, is the maximum allowable concentration. Because it is now conceded that practically all industrial lead poisoning is contracted by the breathing of lead dust and fumes, it becomes of considerable importance in working out the etiology to ascertain the amount and quality of lead dust and fumes in the air, during the occupation of the involved individual. This point will be discussed later with reference to prevention.

Formerly, the nature and variety of non-industrial sources of lead intake made it particularly difficult to establish industrial etiology on a sound and competent basis. More recently, however, non-industrial lead intake has been measured by scientific methods and with the exception of an occasional difficulty with various types of homemade beverages stored in utensils glazed with a lead compound, the non-industrial lead intake is not believed to be a considerable factor in the production of lead poisoning in adults. There are, however, some exceptions and these should be taken into account, as indicated.

The diagnosis of industrial lead poisoning is not a simple matter on account of the fact that clinical symptoms and signs are protean and are liable therefore to simulate other diseases. Great increase in our knowledge, however, has occurred within the past ten to fifteen years, and it is now known that there is a close relationship between the action of lead and its compounds within the body and the calcium metabolism, and further, normal and abnormal excretion of lead has been worked out on a scientific basis. It is now an accepted fact that persons without industrial exposure normally excrete a certain amount of lead in the urine. It has been stated that definite clinical findings of lead poisoning are practically always associated with abnormal urinary lead

excretion — the reverse, however, is not true — that abnormal urinary excretion of lead in the absence of characteristic clinical findings indicates lead poisoning.

Other laboratory findings are important, particularly the examination of the blood, some investigators and clinicians using the increase in basophilic stippled red cells as an indication of positive findings and others using what is known as the basophilic aggregation test. Both are found to be successful, the former more adaptable in cases of frank poisoning, the latter more useful in cases of lead absorption and suspected incipient poisoning.

Although as indicated, the clinical symptoms and signs of lead poisoning are protean, the principal effects are expressed in the blood, the gastrointestinal system, and the nervous system, named in order of their usual chronological occurrence. In brief, the most consistent findings are changes in the color and form of the red cells of the blood, colic and constipation, and weakness and atrophy of the extensors and flexors of the muscles of the wrists, with partial paralysis in the more advanced types of cases.

The differential diagnosis, as may be obvious, has to do mainly with differentiating industrial lead poisoning from non-industrial diseases which may involve the blood, the gastro-intestinal system, and the central and peripheral nervous systems, producing changes which are similar to those observed in frank lead poisoning. Such non-industrial diseases show a considerable variety of pathological processes affecting the above-named systems.

The treatment of lead poisoning has undergone considerable change within the past ten to fifteen years. In general, the treatment consists mainly of appropriate therapy for the toxic episodes and the mobilization of stored lead in the long bones. It is usually agreed that it is wise to facilitate the storage of lead by an excess calcium intake rather than to set lead free in the blood stream; after the passing of acute toxic episodes, the elimination of lead may be assisted by means of low calcium intake, the administra-

tion of weak organic acids and ammonium salts, sodium bicarbonate, and potassium iodide. Care should be exercised in using such forms of treatment on patients who have nephritis.

Two schools of thought with widely divergent views exist with reference to deleading the body: there are those who feel that in the majority of instances deleading will accomplish good results, while there are others who adopt a conservative attitude and because of the possibility of precipitating acute symptoms, feel that it is safer to produce artificial deleading by slow methods, or to resort to the natural process of letting the body delead itself over a period of time. The choice in the matter depends upon the individual case and the previous results which have been achieved at the hands of the physician in charge of the case.

Storage of lead may be facilitated in acute cases by a high calcium diet or by the slow intravenous injection of 10 cc of 20% calcium gluconate; variations with relationship to this form of treatment have been devised by various physicians. In the deleading process, low calcium diet together with oral administration of ammonium chloride has been found to be effective, whereas in certain cases parathyroid extract and sodium bicarbonate also have been useful. Within recent years it has been established that large doses of viosterol have been instrumental in increasing the excretion of lead. Other investigators have found that an adequate phosphorus intake is essential for the depositing of lead in the bones and for the removal from the bones of the excess cations, as the insoluble phosphate salts in the feces; it has therefore been recommended that deleading may be accomplished by giving a diet low in calcium and relatively high in phosphorus, using viosterol 20 minims daily.

There are also variations of treatment with reference to the amount of intoxication, and where there has been high, medium or low lead intake in industrial occupations. In all these types of treatment, deleading should be done in the hospital where the patient may be closely supervised. Some investigators advise

against deleading more often than once in four weeks and the course should not be prolonged for more than three or four days; some physicians believe that it may take from six to fifteen courses of deleading to free the individual of his most loosely-combined lead, which of course is the most dangerous type of lead. It is again warned that deleading should not be undertaken in the presence of acute symptoms; it is further agreed by all investigators that calcium therapy is considered to be the most specific at the present time.

The outlook in cases of lead poisoning is much better now than formerly because of the occurrence of moderate types of effects, the severer forms being a great deal less frequent than in former years.

Metal Fume Fever

This name is applied to the condition which results from breathing fairly heavy concentrations of freshly formed metallic oxide fumes, principally zinc and one of its alloys, brass; although the condition may result from the inhalation of other metallic oxide fumes. Metal fume fever is not a systemic poisoning, but it should be borne in mind that some of the metallic fumes that can cause it, such as lead, cadmium, mercury, and manganese, may also cause systemic poisoning.

The symptoms come on some hours after exposure, usually after leaving work, a dryness of the throat being first noticed, which may be followed by a dry cough and a feeling of heaviness in the chest. Rise of temperature, accompanied by sweating, and leucocytosis and elevation of the pulse rate and the blood pressure usually follow, sometimes with chills. The symptoms ordinarily pass off in 12 to 24 hours and the usual experience is that the employee returns to work the following day. A natural immunity is conferred in certain types of workers, which seems to be lost rapidly after removal from continuous exposure.

Diagnosis is made usually on a clear history of industrial exposure to metallic oxide fumes. It must be remembered that

the effects and symptoms of metal fume fever are very similar to those of influenza and that the malady is more often noticed in the fall and winter, when influenza usually occurs. The industrial history is, therefore, important in diagnosis, particularly with reference to a causative exposure to metallic oxide fumes.

The outlook is good for complete recovery in a short time and usually there is no time lost from work, although there may be temporary incapacity in terms of hours.

The treatment is symptomatic and especially directed toward comfort because the patient usually recovers within a relatively short time in all events. Rest in bed with adequate warmth and protection from drafts are recommended. However, all suspected cases of metal fume fever should be attended by a physician because the symptoms may closely simulate influenza which makes the differential diagnosis important.

Other Dusts and Fumes

It must not be concluded that although clinical examples were given of silicosis, lead poisoning, and metal fume fever, there are not other examples which are of importance. These subjects were selected because of their frequency of occurrence and because of the fact that they are commonly encountered as examples of dust alone, dust and fumes, and fumes.

Among the other metals which may be mentioned are arsenic, antimony, cadmium, manganese, mercury, radium, selenium, silver, tellurium, thallium, and vanadium. Not all of these are equally important with reference to toxic effects.

Probably the most important of these from a toxic point of view are arsenic, mercury, manganese, and cadmium. Arsenic produces chronic poisoning chiefly from breathing over a long period of thin air containing dusts or sprays of arsenic compounds. The compounds of arsenic such as lead arsenate, paris-green, arsenic oxide and arsenic sulphide are among the more dangerous forms.

Where dusts of mercury are present, they will be in the form of either mercurous or mercuric compounds, where metallic mercury is used in the manufacture of such items as thermometers, mercury switches, mercury vapor lamps, mercury boilers, etc., the hazard is from the vapor since mercury has a considerable vapor pressure even at normal temperatures. Mercurialism is characterized by three features: inflammation of the mouth, muscular tremors, and psychic irritability — sometimes all of these are encountered and sometimes only two or only one. In the more chronic or slow form of poisoning, a characteristic tremor is the commonest symptom, coming on late, and has been noted to occur particularly among mercury miners.

Manganese poisoning is an old industrial poisoning, having been described more than 100 years ago, but has receded in incidence in American industry. Characteristically it produces a peculiar slapping gait with phenomena of retropulsion and propulsion; masklike faces; monotonous voice with economical speech; muscular twitching varying from a fine tremor to gross rhythmical movements of the extremities; languor and sleepiness; cramps in the calves of the legs with stiffness; slight increase in the tendon reflexes without any disturbance of the deep or superficial sensation. Studies within the past five years indicate that dust is the most important type of exposure.

Cadmium is encountered in the smelting of ores, the handling of socalled 'blue powder' compounds, the spraying of pigments, the welding of alloys, and the melting of cadmium metal itself, as well as certain non-industrial exposures where utensils have been plated with this metal. In recent years there have been several outbreaks of cadmium poisoning among non-industrial users of such utensils. The use of the metal has increased tremendously and therefore, there is more industrial exposure. Two types of effects have been observed, those with reference to the lungs and also those with reference to the gastro-intestinal tract, the former being more important and more frequent. Pulmonary involvement is characterized by a feeling of constriction in the

chest, edema and congestion of the lungs, sometimes with hemorrhage, proliferative interstitial pneumonitis; while the latter is characterized by nausea and vomiting and a general irritative reaction of the gastro-intestinal tract, with diarrhea, hiccough, and oliguria.

If further details are desired on the clinical aspects of the metals mentioned, standard works on industrial hygiene and occupational diseases should be consulted.

Prevention

The general principles of prevention involve first the identification of industrial exposures and the evaluation of such exposures by proper scientific methods to determine whether or not they represent actual health hazards. This is done by sampling the air and determining the amount of air contaminants and the quality of them in each type of occupation or problem involved, and comparing the results with the accepted standards of maximum allowable concentration that have been adopted by official bodies. Second, after it has been determined that there is an actual health hazard represented by the occupation, control measures, both engineering and medical, are instituted to control the hazard, providing it cannot be entirely eliminated.

In discussing the methods of engineering control, we are concerned with three basic principles:

1. Investigation of occupational conditions.
2. The measurement of the occupational exposure and the rating of the hazard, if one exists.
3. Putting into effect the specific measures of protection which have been indicated by information secured under (1) and (2).

Among the methods of approach for engineering control measures may be mentioned:

1. Physical and hygienic survey of plant conditions.

2. Occupational history and analysis.
3. Adequate information concerning materials and processes used.
4. Atmospheric and gross sampling of materials, combined with quantitative physical and chemical determinations.
5. The rating of the hazard by comparing the findings in the given instance with accepted standards.
6. An appraisal of the efficiency of protective apparatus and devices.
7. An adequate and proper interpretation of the information so that it can be applied practically to the industrial problem involved.

When the foregoing methods of approach to engineering control have been carried out and information secured from them, the information can indicate the necessity for using specific methods of protection which have been found useful in different combinations:

1. The use of mechanical devices, such as exhaust ventilating systems, respirators, masks, and the like.
2. The changing of processes (segregation, enclosure, wetting, etc.).
3. Substitution of nontoxic materials for those of proved toxicity.
4. Educational programs for management, workers, engineers, and physicians.
5. Continuous maintenance and checking of the efficiency of protective equipment.

Atmospheric Sampling

The best method for collecting a sample of a dust or fume from the air will depend somewhat upon the type of contaminants to be sampled. The degree of hazard of fibrosis-producing dusts is measured in terms of particles per unit volume of air (example:

10 million particles of silica dust per cubic foot of air) while toxic dusts and fumes which may cause systemic poisoning are usually measured in terms of weight per unit volume of air. (Example: lead dust 1.5 milligrams per 10 cubic meters of air.)

Since results may vary with the type of air sampling device used, it is important to select a type accepted through general usage in the practice of industrial hygiene.

The larger Impinger which samples one cubic foot of air per minute by impinging the air containing the dust particles from a nozzle submerged in water or alcohol against a plate or bottom of the flask was used in all of the original studies in the United States on fibrosis-producing dusts. The large Impinger, however, has the disadvantage of being bulky and requiring electric current or compressed air for operation of the motor-driven air pump, which produces the suction stream for air sampling.

A portable Midget Impinger has been developed which has been proven to give results as reliable as the large Impinger. This apparatus samples at the rate of 1/10th of a cubic foot per minute and is operated by a hand cranked, positive action, four cylinder pump, in conjunction with a vacuum regulator which maintains an unfluctuating suction regardless of minor variations in rate or smoothness of cranking.

After the sample has been collected in the Impinger flask, a portion is placed in a special dust counting cell and counted under a microscope with an eyepiece having a special ruling similar to a blood count ruling.

Further analyses are necessary with reference to the amount of free silica in the dust and this is accomplished by the use of specific chemical, petrographic and X-ray diffraction methods.

The Impinger method of sampling is not entirely suitable for collecting fumes or dusts of toxic materials such as lead, cadmium, and others because the efficiency of the Impinger is not adequate when sampling the very fine fume particles and since the amount

or concentration of such materials in air which may be injurious is much smaller than the safe amounts of fibrosis-producing dusts, a correspondingly large air sample should be taken for reliable results.

The Electrostatic Precipitator is widely used in the collection of toxic dusts and fumes, particularly of metals and their compounds. A Portable Electrostatic Precipitator has been developed which operates from 110 volts, 60 cycle current, and by high voltage electrical precipitation collects the sample of the particulate matter dry on the inside of aluminum or glass cylinders. This device samples at the rate of 2 cubic feet per minute. After the sample is collected, a quantitative chemical analysis is made in the laboratory. This type of apparatus permits determinations of very low concentrations of many different kinds of atmospheric particulate matter.

Where equipment or personnel is not available for atmospheric sampling, state industrial hygiene bureaus or private testing laboratories may be engaged. Many insurance companies also have facilities for such work. The Industrial Hygiene Foundation in Pittsburgh, Pa., an association of industries for the advancement of healthful working conditions, also have an excellent staff of technicians for industrial hygiene surveys or consultations in this field.

Respiratory Protective Equipment

It frequently happens that because of the nature of the operation or process in an industry, it is not possible to thoroughly control a hazardous exposure resulting from the evolution of dusts and fumes. In such instances, it is considered wise to provide employees with personal respiratory protective devices. These vary with the kind of exposure and with the amount of materials in the air.

For ordinary conditions in which only dusts and fumes are present, simple filter type respirators are available which will

protect the worker against the hazards of breathing these dusts and fumes.

Such respirators normally consist of a facepiece covering the nose and mouth, made of some pliable material such as rubber which will conform to the face and provide a comfortable and dust-tight fit, and to which is attached a filter made of treated paper or felt through which the air is inhaled. If the filtering material is of the correct type and density, it will effectively filter particles down to less than a micron in size from the air.

Comfort is a most important consideration in a device of this type. Since a high breathing resistance will materially affect the comfort of the wearer, the filters should be of large area so that the inhalation resistance will be as low as possible. Valves should be provided which open on exhalation and further lessen the exhalation breathing resistance.

The particular type of filter employed will depend upon the kind of contaminant present in the air. Since the amounts of such toxic materials as lead which will affect the wearer are much smaller than the harmful amounts of fibrosis-producing dusts, a filter will have to be more effective when used against toxic dusts than against fibrosis-producing and nuisance dusts.

For special operations such as sandblasting, a mechanical filter type respirator is not practical because of the extremely high concentrations of dusts present. In such conditions, special air-supplied masks or respirators should be used in which air from a compressed air line is supplied through a hose to the facepiece for breathing. Such devices may also be employed if desired as protection in less severe and hazardous operations. The advantage of an air-supplied respirator is that there is no breathing resistance and the wearer is protected against any and all dusts and fumes in the surrounding atmosphere. However, the restriction by the air hose which must be attached to the wearer at all times between his facepiece and the air supply, prevents the use of this device in many operations where the worker must move freely

over a considerable area. Furthermore, caution must be taken to see that the air supplied to such devices is free from all injurious or objectionable odors and contaminants and that the humidity and temperature of the air is such as to provide reasonable comfort.

The United States Bureau of Mines (Department of the Interior) tests respiratory protective devices against various air contaminants under carefully controlled conditions. Devices meeting its tests receive that Bureau's official approval, and this is indicated by approval numbers on the device and a reproduction of the certificate on the package. Equipment approved by the U. S. Bureau of Mines for the particular contaminant or class of contaminants in the air should be used.

MEDICAL CONTROL

It is of course obvious that medical control measures are futile after a disease has been contracted because of the exposure to dusts or fumes except when the information is adapted to prevent additional cases. However, before the diseases are contracted, there are a number of things that can be done by physicians to prevent such occupational diseases, which may be summarized as follows:

1. The intelligent use of preemployment physical examinations, including chest X-ray films, and appropriate clinical laboratory tests as indicated.
2. Similar use of periodic physical examinations, chest X-ray films, and clinical laboratory tests on employees in occupations where typical hazards have been proved to exist, by industrial hygiene investigations.
3. Responsibility for seeing that hazardous exposures are properly taken care of, by various efficient methods of protection, including the supplying of personal respiratory protection, where industrial conditions make it impossible to control the hazard by other means.

4. Prompt transference to non-hazardous occupations of employees found to have symptoms of an occupational disease associated with their exposure.
5. Prompt removal from work of all persons suffering from active occupational diseases, when such persons are a menace to themselves, their fellow workmen or the employer.
6. Adequate provision for treatment, rehabilitation, and prompt return to work, of employees having disability resulting from occupational diseases.

Summary

In this brief discussion of dusts and fumes in industry, the sources, effects, and control have been considered under the following headings:

1. Definitions.
2. Incidence and etiology.
3. General diagnostic methods.
4. Clinical examples.
5. Prevention — general principles, including specific methods of air sampling and apparatus for this purpose; specific methods of protection, including mention of respiratory protective equipment available; and other general methods.

It should again be emphasized that industrial managers are interested in the prevention of trouble that may arise from exposure to dusts and fumes in industry, and that this principle is particularly important nowadays because of the need for every person in active production, and in addition, because so many physicians are being used in the armed services.

It is believed that if the suggestions made in the foregoing pages are followed, the problems arising from dusts and fumes in industry can be successfully managed.

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Throughout the many types of American Industry are the products of this Company — the largest commercial manufacturer of approved safety equipment in the world today. In 1914, Mine Safety Appliances Company was formed (as its name would indicate), to serve the mining industry. Today, the Company has far outgrown its original field, to include the design and manufacture of approved safety equipment for every major industry and the public protective services.

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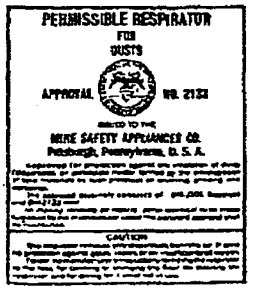

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M·S·A *Dustfoe* RESPIRATOR

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The Dustfoe Respirator

The Comfo Respirator

These widely-used respirators for protection against harmful dusts offer maximum breathing comfort and wearing ease. Both answer all respiratory protective requirements against pneumoconiosis-producing, nuisance or toxic dusts, and selection is a matter of personal preference.

The compact and light-weight Dustfoe Respirator is built of transparent plastic — durable, odorless, non-corrosive, non-conductive of electricity or heat. A molded sponge rubber face-cushion assures comfortable fit; the filter is easily replaceable; the entire unit can be sterilized without harm, and its transparent construction enables checking of interior condition at a glance without disassembly.

The original U.S. Bureau of Mines-approved Comfo Dust Respirator features twin side-placed filters of large area, offering minimum breathing resistance; the thin-edged filter containers of strong, molded plastic do not obstruct downward or sidewise vision, and there is no interference with wearing protective hats or goggles on the job. The flexible rubber face piece fits the face snugly; the respirator is easily cleaned and sterilized.

M·S·A *Comfo* METAL FUME RESPIRATOR

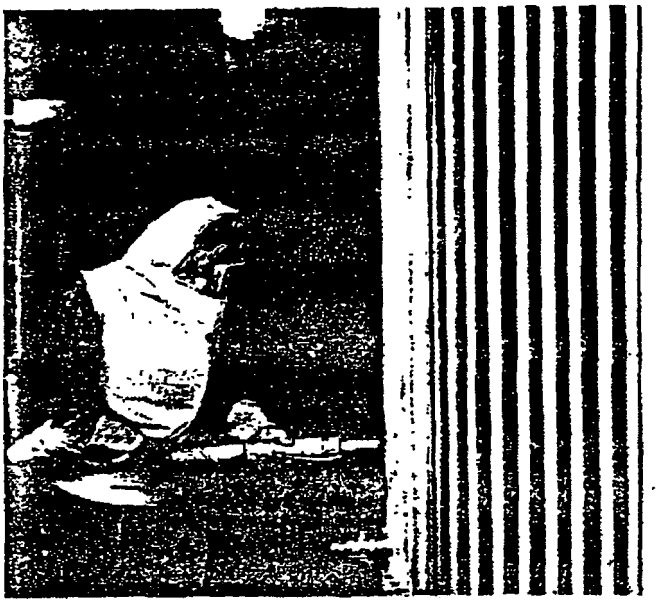
Of basic "Comfo" design, this respirator with a special filter affords respiratory protection to workers exposed to toxic fumes given off by metals in molten condition — such as alloys or materials containing lead, zinc, cadmium, manganese, chromium, arsenic, etc. —fumes from which may arise in burning, welding, melting, pouring, or cutting operations. The filters are quickly replaceable as required.

* * *

M·S·A *Air Line* RESPIRATOR

Gives complete respiratory protection against many hazardous classes of air contaminants by employing air from the shop compressed air line for breathing. The half-mask type rubber facepiece is equipped with twin exhalation valves and an air-deflector tube. The complete assembly includes a corrugated supply tube, quick release connection, "bump-proof" air control valve and an air hose with couplings. Where protection to the face and eyes is an additional requirement, an All-Vision gas mask type facepiece can be supplied.

M·S·A ABRASIVE MASK



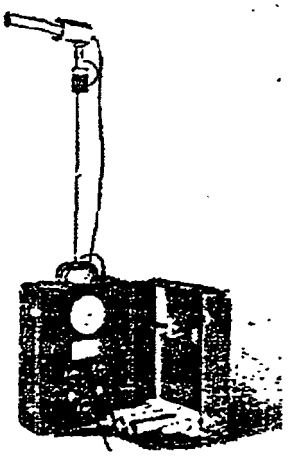
This sturdy assembly weighs less than 4 pounds, yet gives complete protection to the wearer from heavy concentrations of fine dust present in shot or sandblast rooms. Using supplied air, the M.S.A. Abrasive Mask comprises a facepiece and connecting tube, air flow control valve with "bump-proof" adjustment, quick release connection and optional air filter, web belt to carry valve and filter, and air hose with couplings. A lightweight, yet long-wearing hood covers the head and shoulders of the operator and fastens to the waist, cushioning the impact of abrasives. The facepiece has a wide-vision safety glass lens with a tilting screen cover of alloy steel. Both lens and screen are quickly removable and replaceable. The operator can immediately detach himself from the airline in the event of supply failure — breathing through the filter on the free end of the inhalation tube as he leaves the confined space.

M·S·A MIDGET IMPINGER

A portable instrument for quick and dependable dust sampling, the Midget Impinger is entirely self-contained and hand operated, yet weighs less than 10 lbs. The Impinger samples at the rate of 1/10 cu. ft. per minute, with pump maintaining an unfluctuating suction regardless of minor variations in cranking rate. The apparatus is contained in a convenient carrying case, complete with nine Impinger flasks and nozzles.

M·S·A *Electrostatic* DUST *and* FUME SAMPLER

This instrument provides highest precision in the quantitative sampling of dusts, fumes (including metal fumes) and mists — utilizing the principle of preionization with electrostatic precipitation of air-borne particles upon a collecting cylinder, for determination by weight, count or chemical analysis. Accurate determination is enabled of very low concentrations of atmospheric particulate matter.



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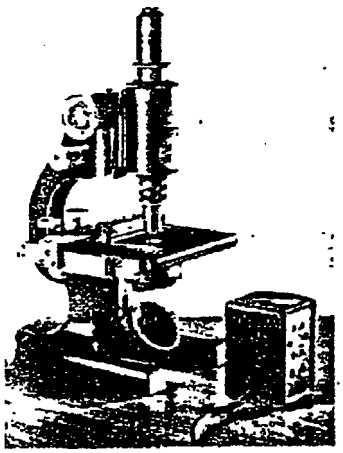
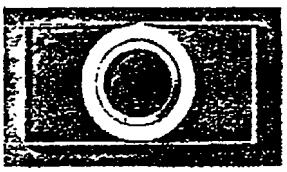
M-S-A *Dust Counting* MICROSCOPE



This is an instrument low in cost, yet of high grade workmanship and performance — specially equipped and adjusted for dust determination. The fine adjustment is by micrometer screw; coarse adjustment is by diagonal rack and pinion. Equipment includes bakelite stage with built-in mechanical stage; fork type substage divisible Abbe condenser, nosepiece adapter, 10-X eyepiece fitted with Whipple disc, 10-X objective, and other accessories. A special set of auxiliary laboratory dust counting equipment is available.

DUST COUNTING CELL



A new cell for counting impinger samples, with an accurate depth of 1 mm. Absence of corners makes cell easier to clean and prevents leaks and air bubbles.

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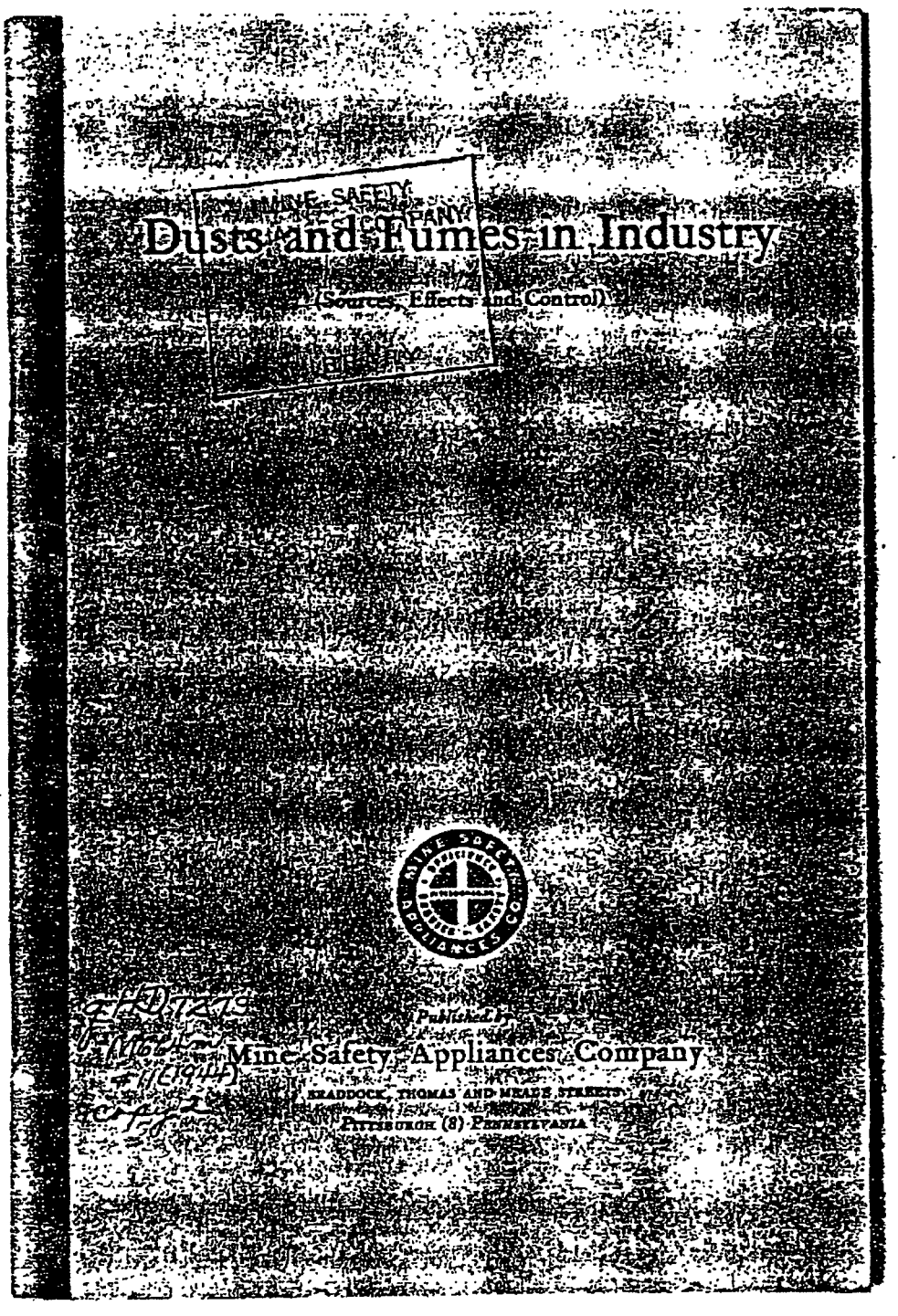
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(Sources, Effects and Control)
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Dusts and Fumes in Industry

(Sources, Effects and Control)

Introductory Statement

In dealing with occupational diseases and their effects, the attending physician should keep in mind that the etiology of these conditions is present in the industrial environment — if the conditions are to be considered truly industrial in origin.

It is likewise important to understand that rational diagnosis and treatment rests upon a fairly accurate knowledge of the etiology of the disturbances; this is especially true of occupational diseases and allied disorders.

Moreover, employers, industrial hygiene and medical departments are greatly interested in the prevention of various maladies which ordinarily affect employees; this clinical and economic interest on the part of industrial management further emphasizes the need for understanding the etiological factors in the working environment, so that they may be properly controlled.

This booklet on industrial dusts and fumes is the second of a series of which the purpose is to show the most important effects of occupations, show how these occur, give measures for treatment, diagnosis, and prevention, meanwhile considering the source or cause of such conditions. It is hoped that these simple and brief expositions will be of considerable help to industrial physicians, and to private practitioners who have among their patients employees of industrial plants subject to such exposure.

Definitions

Dusts may be defined as solid particles of matter which are ordinarily produced by grinding, crushing, drilling or blasting

processes, the composition of the particles being the same as that of the parent materials. These materials may be rock, metals, ores, or such products, for example, as coal, grains, or wood. The size of the particles varies from what is called ultra-microscopic to those which are visible. Authorities now agree that the hygienic significant particle size of dust ranges from a fraction of a micron to 10 microns. (A micron is 1/25,000 of an inch). The most frequently encountered dusts in industrial processes are mainly those from inorganic minerals, and a few organic dusts, such as wood and flour.

Fumes may be defined as solid particles which are formed by processes similar to combustion, condensation and sublimation. The most common types of fumes encountered in industry are usually caused by the oxidation of a metal, of which typical examples are the oxides of lead or zinc. Particle size is usually smaller than dust and ranges below one micron. In contrast to dusts, fumes tend to flocculate very easily. Many people erroneously speak of fumes when they mean vapors of liquid materials, such as acid solutions, organic solvents, and the like.

Incidence and Etiology

Dusts are the most common type of air contaminants encountered in industrial processes, and can occur whenever various materials, as mentioned before, are subjected to processes which break them up into fine particles; or where powdered or pulverized materials are handled or transported. Inorganic dusts are the prevailing type so far as frequency is concerned, but organic dusts are also found in different industrial operations.

Fumes are usually found wherever there are molten metals such as lead, zinc, and calmium being processed or used in industrial operations. Welding is an operation which has greatly increased in frequency of use and it may give rise to fumes of the metals which are welded or to fumes and gases from the variegated coatings of welding rods.

It is to be borne in mind that metals may produce both dusts and fumes, according to the nature of the operation and the state of the materials when processed. For example, among the heavy metals such as lead, dust may be of equal or greater etiologic importance with fumes — other mineral dusts and metallic dusts also are considered of etiological importance, as the following outline will show:

I. *Organic dusts:*
   1. Non-living organic dusts:
      (a) Toxic and/or irritant dusts; "(Various organic compounds such as trinitrotoluene, mercury fulminate, tetryl)."
      (b) allergy-producing dusts (woods, pollens, and the like).
   2. Living organic dusts:
      (a) bacteria
      (b) fungi

II. *Inorganic dusts:* (mineral dusts)
   (a) toxic and/or irritant dusts; (mainly the dusts from heavy metals and their salts, such as arsenic, lead, manganese.)
   (b) fibrosis-producing dusts, (examples are free silica and asbestos).
   (c) non-fibrosis-producing dusts; (the so-called inert dusts, such as alundum, corundum, emery, limestone, magnesite, marble, plaster-of-paris, and polisher's rouge).*

The most important of the non-metallic, inorganic mineral dusts is free silica, which produces silicosis, if breathed in sufficient quantities over a long period of time. Free silica dust and

* This classification is in accordance with the present state of knowledge. Future studies and experience will probably show some of the so-called inert or nuisance dusts to be harmful.

asbestos dust are the two kinds of mineral dusts which produce disease entities corresponding to the names of the dusts — silicosis and asbestosis. Mixed dusts of various kinds, such as various ores which contain different amounts of free silica, can produce different types of reactions, according to the kind of minerals which are in the ores. Where, for example, there are mixtures of iron, and various calcium-containing compounds such as gypsum, with free silica, a protective influence is exerted against the action of free silica on the lungs. Other modifications of free silica action occur according to the types of minerals present some of which tend to inhibit or restrain the action of free silica.

Another important conception with reference to the etiology of dust and fume diseases which may result from exposure to these classes of materials, is the use of the terms *exposure* and *hazard:* they are not synonymous — exposure merely means contact with some substance which is a *potential health hazard;* whether it is an *actual health hazard* can only be determined by an industrial hygiene engineering survey, in which the amount and kind of exposure is rated, according to accepted standards of concentration, size, and quality of dusts and fumes. Many persons erroneously therefore state that there is a lead hazard, for example, when it is only known that contact is being made with lead; and when there are no data to support the contention that there is an actual hazard present in the working environment.

Methods for the determination of dusts and fumes in the air, also with reference to the types of devices used for such determinations, will be mentioned later. The importance of air contamination will be realized when it is stated that the pathologic effects produced by dusts and fumes are brought about almost entirely by the inhalation of these compounds and materials. There is only one outstanding example, for instance, of the possibility of absorption of lead through the skin, namely tetraethyl lead, and this occurs in the form of a fluid. No dusts, so far as is known, produce any systemic pathological condi-

tions from skin absorption, and it is doubtful that any kind of true fume is absorbed by the skin.

From the etiologic point of view, particularly with reference to pathologic effects, the following outline shows how different types of substances (among the dusts and fumes) act on various systems in the body to produce pathological effects:

1. *General systemic poisoning:*
   Examples: Arsenic, lead, manganese, mercury.
2. *Substances acting on the circulatory system:*
   Examples: Arsenic, lead, mercury, vanadium.
3. *Substances acting on the respiratory system:*
   Examples: Asbestos, free silica.
4. *Substances acting on the gastro-intestinal system:*
   Examples: Antimony, cadmium, lead, mercury, zinc.
5. *Substances acting on the skeleton and joints:*
   Examples: Lead, mercury, mesothorium, phosphorus, radium.
6. *Substances acting on organs of special sense:*
   Examples: Arsenic, lead, manganese, mercury.
7. *Substances acting as fetal poisons:*
   Examples: Lead, mercury.
8. *Substances acting on the genito-urinary system:*
   Examples: Arsenic, lead, mercury, vanadium.
9. *Substances acting on the brain and nervous system:*
   Examples: Arsenic, lead, manganese.

General Diagnostic Methods

The accurate diagnosis of pathological conditions arising from the inhalation of industrial dusts and fumes has tremendous economic significance and also it is of medicolegal importance. Moreover, it is impossible to understand how proper and effica-

ceous treatment may be rendered unless a scientific diagnosis is made. If a claim results from the pathological condition alleged, it is evident that the disease sustained must be directly related to the employment and further, must produce disability to entitle the individual concerned to compensation.

Diagnostic methods with reference to dust and fume diseases need not differ widely, if at all, from those used as routine procedures in general scientific medicine, although there are specific methods as in any other application of general medical diagnostic principles to any special group of persons. New discoveries occur relative to diagnostic methods in occupational disease work and for the most part, reliance must be placed on information which is scattered throughout the current literature in addition to available textbook material which is better now than it was even five years ago.

Emphasis has already been placed upon the vital part played by etiology; it is wise to repeat that etiology forms the bulwark of the diagnostic and therapeutic background.

Among the methods which are used to make a fairly accurate diagnosis with reference to occupational diseases, is the taking of a thorough industrial history; consideration of symptomatology, the physical findings, the results of air examination; and finally, last but not least, a differential diagnosis, ruling out diseases of ordinary life which have no relation to occupation.

The industrial history may be of very great assistance in arriving at an opinion. Most industrial physicians of considerable experience believe that it is desirable to include a complete resume of the kinds of occupations which have been experienced by the individual being examined, dating from the first job to the last one. It is of course clear that the details of actual occupational exposure cannot be evaluated by this method, but important information bearing on the diagnosis may thus be obtained. It is the present practice, therefore, to list the occupations with the dates of occurrence, in the industrial history. It is

furthermore advisable to get more detailed information relative to the performance of the occupation, if that can be obtained. This will at least give information relative to the kind of exposure in industry, although it will give no idea of the severity of exposure.

The symptomatology in many kinds of occupational disease is likely to be confusing rather than useful, because in many instances it so closely simulates that of non-industrial disease that it is quite commonly not characteristic. Especial attention should be given, however, to the recording of the symptomatology and critical attempts made of its proper evaluation. Here again, distinction must be made between symptoms which may be due to non-occupational disease and those symptoms which are specific for occupational diseases — this is where the difficulty arises in adequately assessing symptomatology.

The physical findings, on the whole, are more reliable than the symptomatology, although in certain instances they too are not characteristic and are, therefore, liable to be somewhat puzzling. Where only one portion of the body is affected, as in silicosis, for example, it might be assumed that the physical findings would be more characteristic, but the opposite of this is usually true. In other instances, for example lead poisoning, where different organs and systems are pathologically affected, the picture is liable to be one which demands expert evaluation to differentiate from non-occupational conditions.

Clinical laboratory data, including X-ray findings, are frequently found to be decisive, although here again intelligent appraisal means proper interpretation of the data. When they are decisive, such tests are by the very specificity of their nature, of great value as diagnostic aids, although they should be definitely correlated with other findings.

Differential diagnosis presents perhaps the most difficult problem of all in the realm of diagnosis of occupational diseases. There is good reason to believe that differential diagnosis will

continue to be one of the most difficult phases of the medical ramifications of the medicolegal problems of occupational diseases. The reason which has already been indicated is that the symptomatology and frequently the physical findings imitate so closely the symptomatology and physical findings of non-industrial diseases, that one must make use of specific procedures which are designed especially as diagnostic guideposts. Frequently such specific procedures are not used to any great extent, except by those who are highly specialized in the field and whose work takes them into a review of the current literature containing these developments. For example, one may believe that an industrial employee has lead poisoning, largely because of second-hand knowledge that lead was used in his occupation, more easily than one could demonstrate adequate etiology and the existence of lead poisoning by scientific methods.

Because of all of these obvious and other difficulties, it seems wise to adapt specifically certain examination procedures to industrial problems arising out of occupational or suspected occupational diseases. Most authorities now agree that the examining physician can be of greater service and give more efficient advice regarding either acceptability for employment, continuance in it, opinion on the diagnosis of systemic pathologic conditions and the estimation of disability, if he is conversant with the following points:

1. The nature and extent of the industrial exposure into which the employee will go or where he has been. This can be determined only by scientific methods which will be referred to later.

2. The effect of this specific exposure upon normal and abnormal physical states of employees, as interpreted by the physical status of each individual on examination. (This refers specifically to etiology in the industrial environment and the effects which such etiology may or could produce.)

3. The nature of the protective devices supplied by the employer, if a hazardous exposure cannot be otherwise controlled, and as to whether these devices are functioning efficiently. (Reference will be made later to these devices, especially designed for protection against dust and fume exposures.)

It is realized that these procedures are somewhat removed from the usual program of physical examinations, particularly with reference to the work of part-time industrial physicians, but it is also expected that the physician will be called upon increasingly to know and appreciate such information so that he may function better as a diagnostic medium.

Because the most frequent and most significant exposures in industrial processes are free silica dust and lead and its compounds, it seems advisable to include in the next section some clinical examples, namely silicosis and lead poisoning; there will also be included a brief discussion of metal fume fever.

Clinical Examples

Simply stated, silicosis is a disease of the lungs, caused by the continuous inhalation over a long period of time of very finely divided, microscopic, particles of free silica dust. There have been various standards set up by different agencies, based upon studies of silicotic workmen and their exposure to free silica dust. The National Silicosis Conference Report on Medical Control of Silicosis says: "There is evidence that for prolonged exposure a concentration of more than five million particles per cubic foot of a highly siliceous dust is dangerous. Therefore, it is now considered good practice to hold concentrations of highly siliceous dust at 5 million particles per cubic foot, or less."

Because of the fact that industrial exposures, involving high free silica content in which the concentration of dust should be restricted to five million particles per cubic foot, are relatively infrequent in industry, and also because the opposite usually

occurs (an exposure to "mixed" dusts which contain appreciable amounts of free silica but also contain considerable amounts of other types of material) it is believed wise to refer to the formula* developed by the National Silicosis Conference with reference to a working criterion with respect to satisfactory or unsatisfactory conditions, when exposure to 'mixed' dusts occurs.

It is to be borne in mind, however, that any unprotected industrial operation, which involves the production of a lot of fine dust particles of material containing a relatively large percentage of free silica, offers a potential silicosis hazard.

The incidence of silicosis is not known with any degree of accuracy, although various estimates have been made. One of these made some time ago by the American Institute of Mining and Metallurgical Engineers indicated that in the United States the number of industrial workers exposed to the silica dust hazard is in excess of a half million. Figures quoted by the Committee Reports of the National Silicosis Conference disclosed estimates that approximately 2% of the working population of the United States or about one million workers are exposed in some way to the potential hazard of silicosis, and that perhaps half of this number, 500,000, or 1% of the total, are exposed to a serious hazard. It is furthermore believed that approximately 110,000, or 0.2 of 1% of the total, have silicosis to some degree, but likely only 4000 to 5000 (a small fraction of 1% of the total) suffer any degree of disability from the disease. (These figures are based upon prewar estimates.) It can readily be seen that silicosis, although not a rare disease, is not common and furthermore not as common as may ordinarily have been supposed by many persons.

Recent conceptions with regard to the contraction of silicosis have established the belief that approximately 25% of those exposed to a real silica hazard will eventually develop silicosis.

* FORMULA A: "Multiply the percentage of free silica by the total dust count. If the result is under 5 million, the condition may be considered permissible. If the result is over 5 million, the condition may be considered too high. For example, 10 per cent free silica with an average total dust concentration of 30 million particles per cubic foot would give 0.10 times 30 million, which equals 3 million (good practice); 30 per cent with an average total dust concentration of 50 million, would equal 0.3 times 50, or 15 million (unsatisfactory). This formula is not applicable to any dust containing less than 5 per cent free silica."

With respect to the occurrence of silicosis in the foundry industry, a prewar survey by the Division of Industrial Hygiene of the New York State Department of Labor, based on a study of 311 foundries, of which 80 plants included X-ray examinations involving 4,754 employees, provides some interesting figures: the X-ray summary showed that 3,259 or 80.2% were negative; 185 or 4.5% showed fibrosis; 110 or 2.7% showed silicosis with and without infection; 485 or 11.9% showed healed primary and reinfection types of tuberculosis; 27 or 0.07% showed clinically significant tuberculosis. It was concluded, as stated in the report, that it would appear that the silicosis hazard in the foundry industry in New York State, while existent, is of mild degree of severity and may be expected to yield satisfactorily to appropriate measures of control. The report further indicated that tuberculosis as manifested by X-ray films of the chest was found to be more prevalent among silicotics than among non-silicotics in foundrymen.

The occupational history, as previously indicated, is of considerable importance. Forms for the recording of occupational records have been devised by various organizations and groups and by physicians and insurance companies. One of the most complete and longer forms has been devised and used by the Saranac Laboratory, Saranac Lake, N. Y., in connection with silicosis studies and symposia on silicosis which have been held by this group. It is suggested that the physician, if interested, should contact the industrial hygiene bureau of his state health department through which detailed forms may be secured or information relative to them.

The symptoms and physical signs are not characteristic; they may be confused even by skilled diagnosticians in other diseases, with the symptoms and signs which commonly occur in both minor and the more serious types of the well-known respiratory infections. Characteristically, shortness of breath on exertion and decreased chest expansion frequently occur in advanced cases of silicosis, but these findings must also be differentiated as

to their casual relationships and their possible association with other diseases.

The clinical confirmation of the diagnosis of silicosis rests mainly on the characteristic X-ray appearance of the chest. This shows the typical lesions of silicosis, hyaline fibrous nodules which are scattered as opaque bodies throughout both lung fields, the density of these nodules being somewhat less than calcified bodies, but discrete in outline, all about the same size in the early stage (so-called millet-seed size) and evenly distributed throughout both lung fields. These nodules appear as the first indication of silicosis, later become somewhat confluent and in the latter stages, there are large masses of opaque material found near the periphery of the lung fields. In the later stages of silicosis, the disease is frequently complicated by infection, usually tuberculous in nature. As an aid to interpretation of the X-ray films of the chest where silicosis may be suspected, it is suggested that the physician refer to a tabulation of the X-ray changes in silicosis, with the underlying histological conditions which cause them, published in the transcript of the Second Silicosis Symposium, Saranac Lake, N. Y., 1935.

Diagnosis rests upon proof of occupational dust exposure sufficient to produce the disease, and the presence of characteristic nodular lesions in the X-ray film of the chest. The mere mention of occupation is not sufficient for diagnostic purposes — adequate knowledge concerning the quality and quantity of dust exposure, as well as the length of the employment period, is necessary to establish a distinct causal relationship.

Although considerable diagnostic reliance is usually and properly placed upon the history of adequate occupational dust exposure and the characteristic chest X-ray appearance, a thorough physical examination with appropriate clinical laboratory examinations should be done, especially with reference to the ruling out of other respiratory diseases. Diagnosis becomes essentially a differential diagnosis, as before explained, one which is likely to be difficult, necessitating special experience

and knowledge. Some of the conditions requiring differential consideration here are miliary tuberculosis, multiple calcifications of healed tuberculosis, cancer of the lungs, and fungous growths of the lungs.

Within recent years, superimposed infection has come to be the accepted cause for disability. The outlook in simple silicosis, therefore, (that is, without infection) is more favorable than was formerly believed. It is obvious that the chief problem for the silicotic is to avoid superimposed infection, which is usually tuberculous.

There is no one specific, efficacious treatment for silicosis, although in recent years, the inhalation of metallic aluminum powder has shown some promise. In cases where tuberculosis complicates the picture, it is obviously to be treated by the best known methods for that disease. There is some recent evidence that surgical therapy as used in ordinary forms of tuberculosis is also a help in the type of tuberculosis which follows silicosis. The evidence for this is not conclusive, but there are good results on record.

In general, recommendations for the treatment of cases of silicosis vary with experience and with the findings of each case. It is believed, for example, that no treatment is necessary in the case of an individual without symptoms, whose X-ray film of the chest (taken perhaps in a routine industrial survey) shows only widespread discrete nodulation without localized shadows indicating infection. However, the employee should have serial X-ray examinations with physical examinations of the chest at least once a year and should be removed from further silica exposure to some other job, since the disease is very slowly progressive and there is at this stage no disability which precludes continuation in employment. A person with localized conglomerate lesions may have symptoms and usually cannot do as heavy work as formerly; therefore, a lighter job should be found. In the presence of active infection where there are symptoms or incapacitation, the individual cannot obviously continue in his

former employment. If the case is one of open tuberculosis with positive sputum, it is evident that such an employee is a source of danger to his fellow workmen and should be placed in a sanitarium or hospital. In cases of inactive infection with no incapacitation, lighter work should be provided, without further dust exposure.

Dusts containing silica in combined form do not cause silicosis; however, one such dust, asbestos, a magnesium silicate, will produce a characteristic fibrosis which is called asbestosis. This disease is quite different in its pathology from silicosis, and has a characteristic 'ground-glass' appearance on the X-ray films of the chest. Animal experimentation indicates that the action of asbestos is probably mechanical, since it cannot be produced in other than the pulmonary tissue. Asbestosis may cause disability and a few fatal cases have been recorded, but in those cases showing marked disability, other organic disease is usually also present. The symptomatology and physical findings include cough, dyspnea, pallor, clubbing of fingers, and diminished chest expansion, with a characteristic X-ray appearance as previously mentioned.

Lead Poisoning

Lead poisoning offers a good example of an industrial poisoning which is contracted by the inhalation either of a toxic dust or fume, and has been stated to be one of the most frequent and important types of metallic poisoning in industry, on account of the widespread use of lead and its compounds.

Although there are no reliable statistics available with respect to the national incidence of lead poisoning, it is believed that lead poisoning is very prevalent and that most of it is mild. The incidence of severe types of cases, particularly lead encephalopathy and the more severe types of nervous system involvement are not seen with as great frequency as in former years. Many physicians who are used to handling industrial cases now rarely see a case of wrist drop due to lead poisoning, whereas 20 to 25 years ago it was a rather common phenomenon, in cases of lead intoxication.

The industrial history is of great importance, as previously indicated, and a thorough consideration of the nature and extent of the exposure to lead and its compounds is an essential point in the etiology. It is now generally accepted that the standard of the U. S. Public Health Service and the American Standards Association of 1.5 milligrams of lead per ten cubic meters of air, is the maximum allowable concentration. Because it is now conceded that practically all industrial lead poisoning is contracted by the breathing of lead dust and fumes, it becomes of considerable importance in working out the etiology to ascertain the amount and quality of lead dust and fumes in the air, during the occupation of the involved individual. This point will be discussed later with reference to prevention.

Formerly, the nature and variety of non-industrial sources of lead intake made it particularly difficult to establish industrial etiology on a sound and competent basis. More recently, however, non-industrial lead intake has been measured by scientific methods and with the exception of an occasional difficulty with various types of homemade beverages stored in utensils glazed with a lead compound, the non-industrial lead intake is not believed to be a considerable factor in the production of lead poisoning in adults. There are, however, some exceptions and these should be taken into account, as indicated.

The diagnosis of industrial lead poisoning is not a simple matter on account of the fact that clinical symptoms and signs are protean and are liable therefore to simulate other diseases. Great increase in our knowledge, however, has occurred within the past ten to fifteen years, and it is now known that there is a close relationship between the action of lead and its compounds within the body and the calcium metabolism, and further, normal and abnormal excretion of lead has been worked out on a scientific basis. It is now an accepted fact that persons without industrial exposure normally excrete a certain amount of lead in the urine. It has been stated that definite clinical findings of lead poisoning are practically always associated with abnormal urinary lead

excretion — the reverse, however, is not true — that abnormal urinary excretion of lead in the absence of characteristic clinical findings indicates lead poisoning.

Other laboratory findings are important, particularly the examination of the blood, some investigators and clinicians using the increase in basophilic stippled red cells as an indication of positive findings and others using what is known as the basophilic aggregation test. Both are found to be successful, the former more adaptable in cases of frank poisoning, the latter more useful in cases of lead absorption and suspected incipient poisoning.

Although as indicated, the clinical symptoms and signs of lead poisoning are protean, the principal effects are expressed in the blood, the gastrointestinal system, and the nervous system, named in order of their usual chronological occurrence. In brief, the most consistent findings are changes in the color and form of the red cells of the blood, colic and constipation, and weakness and atrophy of the extensors and flexors of the muscles of the wrists, with partial paralysis in the more advanced types of cases.

The differential diagnosis, as may be obvious, has to do mainly with differentiating industrial lead poisoning from non-industrial diseases which may involve the blood, the gastro-intestinal system, and the central and peripheral nervous systems, producing changes which are similar to those observed in frank lead poisoning. Such non-industrial diseases show a considerable variety of pathological processes affecting the above-named systems.

The treatment of lead poisoning has undergone considerable change within the past ten to fifteen years. In general, the treatment consists mainly of appropriate therapy for the toxic episodes and the mobilization of stored lead in the long bones. It is usually agreed that it is wise to facilitate the storage of lead by an excess calcium intake rather than to set lead free in the blood stream; after the passing of acute toxic episodes, the elimination of lead may be assisted by means of low calcium intake, the administra-

tion of weak organic acids and ammonium salts, sodium bicarbonate, and potassium iodide. Care should be exercised in using such forms of treatment on patients who have nephritis.

Two schools of thought with widely divergent views exist with reference to deleading the body: there are those who feel that in the majority of instances deleading will accomplish good results, while there are others who adopt a conservative attitude and because of the possibility of precipitating acute symptoms, feel that it is safer to produce artificial deleading by slow methods, or to resort to the natural process of letting the body delead itself over a period of time. The choice in the matter depends upon the individual case and the previous results which have been achieved at the hands of the physician in charge of the case.

Storage of lead may be facilitated in acute cases by a high calcium diet or by the slow intravenous injection of 10 cc of 20% calcium gluconate; variations with relationship to this form of treatment have been devised by various physicians. In the deleading process, low calcium diet together with oral administration of ammonium chloride has been found to be effective, whereas in certain cases parathyroid extract and sodium bicarbonate also have been useful. Within recent years it has been established that large doses of viosterol have been instrumental in increasing the excretion of lead. Other investigators have found that an adequate phosphorus intake is essential for the depositing of lead in the bones and for the removal from the bones of the excess cations, as the insoluble phosphate salts in the feces; it has therefore been recommended that deleading may be accomplished by giving a diet low in calcium and relatively high in phosphorus, using viosterol 20 minims daily.

There are also variations of treatment with reference to the amount of intoxication, and where there has been high, medium or low lead intake in industrial occupations. In all these types of treatment, deleading should be done in the hospital where the patient may be closely supervised. Some investigators advise

against deleading more often than once in four weeks and the course should not be prolonged for more than three or four days; some physicians believe that it may take from six to fifteen courses of deleading to free the individual of his most loosely-combined lead, which of course is the most dangerous type of lead. It is again warned that deleading should not be undertaken in the presence of acute symptoms; it is further agreed by all investigators that calcium therapy is considered to be the most specific at the present time.

The outlook in cases of lead poisoning is much better now than formerly because of the occurrence of moderate types of effects, the severer forms being a great deal less frequent than in former years.

Metal Fume Fever

This name is applied to the condition which results from breathing fairly heavy concentrations of freshly formed metallic oxide fumes, principally zinc and one of its alloys, brass; although the condition may result from the inhalation of other metallic oxide fumes. Metal fume fever is not a systemic poisoning, but it should be borne in mind that some of the metallic fumes that can cause it, such as lead, cadmium, mercury, and manganese, may also cause systemic poisoning.

The symptoms come on some hours after exposure, usually after leaving work, a dryness of the throat being first noticed, which may be followed by a dry cough and a feeling of heaviness in the chest. Rise of temperature, accompanied by sweating, and leucocytosis and elevation of the pulse rate and the blood pressure usually follow, sometimes with chills. The symptoms ordinarily pass off in 12 to 24 hours and the usual experience is that the employee returns to work the following day. A natural immunity is conferred in certain types of workers, which seems to be lost rapidly after removal from continuous exposure.

Diagnosis is made usually on a clear history of industrial exposure to metallic oxide fumes. It must be remembered that

the effects and symptoms of metal fume fever are very similar to those of influenza and that the malady is more often noticed in the fall and winter, when influenza usually occurs. The industrial history is, therefore, important in diagnosis, particularly with reference to a causative exposure to metallic oxide fumes.

The outlook is good for complete recovery in a short time and usually there is no time lost from work, although there may be temporary incapacity in terms of hours.

The treatment is symptomatic and especially directed toward comfort because the patient usually recovers within a relatively short time in all events. Rest in bed with adequate warmth and protection from drafts are recommended. However, all suspected cases of metal fume fever should be attended by a physician because the symptoms may closely simulate influenza which makes the differential diagnosis important.

Other Dusts and Fumes

It must not be concluded that although clinical examples were given of silicosis, lead poisoning, and metal fume fever, there are not other examples which are of importance. These subjects were selected because of their frequency of occurrence and because of the fact that they are commonly encountered as examples of dust alone, dust and fumes, and fumes.

Among the other metals which may be mentioned are arsenic, antimony, cadmium, manganese, mercury, radium, selenium, silver, tellurium, thallium, and vanadium. Not all of these are equally important with reference to toxic effects.

Probably the most important of these from a toxic point of view are arsenic, mercury, manganese, and cadmium. Arsenic produces chronic poisoning chiefly from breathing over a long period of thin air containing dusts or sprays of arsenic compounds. The compounds of arsenic such as lead arsenate, paris-green, arsenic oxide and arsenic sulphide are among the more dangerous forms.

Where dusts of mercury are present, they will be in the form of either mercurous or mercuric compounds, where metallic mercury is used in the manufacture of such items as thermometers, mercury switches, mercury vapor lamps, mercury boilers, etc., the hazard is from the vapor since mercury has a considerable vapor pressure even at normal temperatures. Mercurialism is characterized by three features: inflammation of the mouth, muscular tremors, and psychic irritability — sometimes all of these are encountered and sometimes only two or only one. In the more chronic or slow form of poisoning, a characteristic tremor is the commonest symptom, coming on late, and has been noted to occur particularly among mercury miners.

Manganese poisoning is an old industrial poisoning, having been described more than 100 years ago, but has receded in incidence in American industry. Characteristically it produces a peculiar slapping gait with phenomena of retropulsion and propulsion; masklike faces; monotonous voice with economical speech; muscular twitching varying from a fine tremor to gross rhythmical movements of the extremities; languor and sleepiness; cramps in the calves of the legs with stiffness; slight increase in the tendon reflexes without any disturbance of the deep or superficial sensation. Studies within the past five years indicate that dust is the most important type of exposure.

Cadmium is encountered in the smelting of ores, the handling of socalled 'blue powder' compounds, the spraying of pigments, the welding of alloys, and the melting of cadmium metal itself, as well as certain non-industrial exposures where utensils have been plated with this metal. In recent years there have been several outbreaks of cadmium poisoning among non-industrial users of such utensils. The use of the metal has increased tremendously and therefore, there is more industrial exposure. Two types of effects have been observed, those with reference to the lungs and also those with reference to the gastro-intestinal tract, the former being more important and more frequent. Pulmonary involvement is characterized by a feeling of constriction in the

chest, edema and congestion of the lungs, sometimes with hemorrhage, proliferative interstitial pneumonitis; while the latter is characterized by nausea and vomiting and a general irritative reaction of the gastro-intestinal tract, with diarrhea, hiccough, and oliguria.

If further details are desired on the clinical aspects of the metals mentioned, standard works on industrial hygiene and occupational diseases should be consulted.

Prevention

The general principles of prevention involve first the identification of industrial exposures and the evaluation of such exposures by proper scientific methods to determine whether or not they represent actual health hazards. This is done by sampling the air and determining the amount of air contaminants and the quality of them in each type of occupation or problem involved, and comparing the results with the accepted standards of maximum allowable concentration that have been adopted by official bodies. Second, after it has been determined that there is an actual health hazard represented by the occupation, control measures, both engineering and medical, are instituted to control the hazard, providing it cannot be entirely eliminated.

In discussing the methods of engineering control, we are concerned with three basic principles:

1. Investigation of occupational conditions.
2. The measurement of the occupational exposure and the rating of the hazard, if one exists.
3. Putting into effect the specific measures of protection which have been indicated by information secured under (1) and (2).

Among the methods of approach for engineering control measures may be mentioned:

1. Physical and hygienic survey of plant conditions.

2. Occupational history and analysis.
3. Adequate information concerning materials and processes used.
4. Atmospheric and gross sampling of materials, combined with quantitative physical and chemical determinations.
5. The rating of the hazard by comparing the findings in the given instance with accepted standards.
6. An appraisal of the efficiency of protective apparatus and devices.
7. An adequate and proper interpretation of the information so that it can be applied practically to the industrial problem involved.

When the foregoing methods of approach to engineering control have been carried out and information secured from them, the information can indicate the necessity for using specific methods of protection which have been found useful in different combinations:

1. The use of mechanical devices, such as exhaust ventilating systems, respirators, masks, and the like.
2. The changing of processes (segregation, enclosure, wetting, etc.).
3. Substitution of nontoxic materials for those of proved toxicity.
4. Educational programs for management, workers, engineers, and physicians.
5. Continuous maintenance and checking of the efficiency of protective equipment.

Atmospheric Sampling

The best method for collecting a sample of a dust or fume from the air will depend somewhat upon the type of contaminants to be sampled. The degree of hazard of fibrosis-producing dusts is measured in terms of particles per unit volume of air (example:

10 million particles of silica dust per cubic foot of air) while toxic dusts and fumes which may cause systemic poisoning are usually measured in terms of weight per unit volume of air. (Example: lead dust 1.5 milligrams per 10 cubic meters of air.)

Since results may vary with the type of air sampling device used, it is important to select a type accepted through general usage in the practice of industrial hygiene.

The larger Impinger which samples one cubic foot of air per minute by impinging the air containing the dust particles from a nozzle submerged in water or alcohol against a plate or bottom of the flask was used in all of the original studies in the United States on fibrosis-producing dusts. The large Impinger, however, has the disadvantage of being bulky and requiring electric current or compressed air for operation of the motor-driven air pump, which produces the suction stream for air sampling.

A portable Midget Impinger has been developed which has been proven to give results as reliable as the large Impinger. This apparatus samples at the rate of 1/10th of a cubic foot per minute and is operated by a hand cranked, positive action, four cylinder pump, in conjunction with a vacuum regulator which maintains an unfluctuating suction regardless of minor variations in rate or smoothness of cranking.

After the sample has been collected in the Impinger flask, a portion is placed in a special dust counting cell and counted under a microscope with an eyepiece having a special ruling similar to a blood count ruling.

Further analyses are necessary with reference to the amount of free silica in the dust and this is accomplished by the use of specific chemical, petrographic and X-ray diffraction methods.

The Impinger method of sampling is not entirely suitable for collecting fumes or dusts of toxic materials such as lead, cadmium, and others because the efficiency of the Impinger is not adequate when sampling the very fine fume particles and since the amount

or concentration of such materials in air which may be injurious is much smaller than the safe amounts of fibrosis-producing dusts, a correspondingly large air sample should be taken for reliable results.

The Electrostatic Precipitator is widely used in the collection of toxic dusts and fumes, particularly of metals and their compounds. A Portable Electrostatic Precipitator has been developed which operates from 110 volts, 60 cycle current, and by high voltage electrical precipitation collects the sample of the particulate matter dry on the inside of aluminum or glass cylinders. This device samples at the rate of 2 cubic feet per minute. After the sample is collected, a quantitative chemical analysis is made in the laboratory. This type of apparatus permits determinations of very low concentrations of many different kinds of atmospheric particulate matter.

Where equipment or personnel is not available for atmospheric sampling, state industrial hygiene bureaus or private testing laboratories may be engaged. Many insurance companies also have facilities for such work. The Industrial Hygiene Foundation in Pittsburgh, Pa., an association of industries for the advancement of healthful working conditions, also have an excellent staff of technicians for industrial hygiene surveys or consultations in this field.

Respiratory Protective Equipment

It frequently happens that because of the nature of the operation or process in an industry, it is not possible to thoroughly control a hazardous exposure resulting from the evolution of dusts and fumes. In such instances, it is considered wise to provide employees with personal respiratory protective devices. These vary with the kind of exposure and with the amount of materials in the air.

For ordinary conditions in which only dusts and fumes are present, simple filter type respirators are available which will

protect the worker against the hazards of breathing these dusts and fumes.

Such respirators normally consist of a facepiece covering the nose and mouth, made of some pliable material such as rubber which will conform to the face and provide a comfortable and dust-tight fit, and to which is attached a filter made of treated paper or felt through which the air is inhaled. If the filtering material is of the correct type and density, it will effectively filter particles down to less than a micron in size from the air.

Comfort is a most important consideration in a device of this type. Since a high breathing resistance will materially affect the comfort of the wearer, the filters should be of large area so that the inhalation resistance will be as low as possible. Valves should be provided which open on exhalation and further lessen the exhalation breathing resistance.

The particular type of filter employed will depend upon the kind of contaminant present in the air. Since the amounts of such toxic materials as lead which will affect the wearer are much smaller than the harmful amounts of fibrosis-producing dusts, a filter will have to be more effective when used against toxic dusts than against fibrosis-producing and nuisance dusts.

For special operations such as sandblasting, a mechanical filter type respirator is not practical because of the extremely high concentrations of dusts present. In such conditions, special air-supplied masks or respirators should be used in which air from a compressed air line is supplied through a hose to the facepiece for breathing. Such devices may also be employed if desired as protection in less severe and hazardous operations. The advantage of an air-supplied respirator is that there is no breathing resistance and the wearer is protected against any and all dusts and fumes in the surrounding atmosphere. However, the restriction by the air hose which must be attached to the wearer at all times between his facepiece and the air supply, prevents the use of this device in many operations where the worker must move freely

over a considerable area. Furthermore, caution must be taken to see that the air supplied to such devices is free from all injurious or objectionable odors and contaminants and that the humidity and temperature of the air is such as to provide reasonable comfort.

The United States Bureau of Mines (Department of the Interior) tests respiratory protective devices against various air contaminants under carefully controlled conditions. Devices meeting its tests receive that Bureau's official approval, and this is indicated by approval numbers on the device and a reproduction of the certificate on the package. Equipment approved by the U. S. Bureau of Mines for the particular contaminant or class of contaminants in the air should be used.

MEDICAL CONTROL

It is of course obvious that medical control measures are futile after a disease has been contracted because of the exposure to dusts or fumes except when the information is adapted to prevent additional cases. However, before the diseases are contracted, there are a number of things that can be done by physicians to prevent such occupational diseases, which may be summarized as follows:

1. The intelligent use of preemployment physical examinations, including chest X-ray films, and appropriate clinical laboratory tests as indicated.
2. Similar use of periodic physical examinations, chest X-ray films, and clinical laboratory tests on employees in occupations where typical hazards have been proved to exist, by industrial hygiene investigations.
3. Responsibility for seeing that hazardous exposures are properly taken care of, by various efficient methods of protection, including the supplying of personal respiratory protection, where industrial conditions make it impossible to control the hazard by other means.

4. Prompt transference to non-hazardous occupations of employees found to have symptoms of an occupational disease associated with their exposure.
5. Prompt removal from work of all persons suffering from active occupational diseases, when such persons are a menace to themselves, their fellow workmen or the employer.
6. Adequate provision for treatment, rehabilitation, and prompt return to work, of employees having disability resulting from occupational diseases.

Summary

In this brief discussion of dusts and fumes in industry, the sources, effects, and control have been considered under the following headings:

1. Definitions.
2. Incidence and etiology.
3. General diagnostic methods.
4. Clinical examples.
5. Prevention — general principles, including specific methods of air sampling and apparatus for this purpose; specific methods of protection, including mention of respiratory protective equipment available; and other general methods.

It should again be emphasized that industrial managers are interested in the prevention of trouble that may arise from exposure to dusts and fumes in industry, and that this principle is particularly important nowadays because of the need for every person in active production, and in addition, because so many physicians are being used in the armed services.

It is believed that if the suggestions made in the foregoing pages are followed, the problems arising from dusts and fumes in industry can be successfully managed.

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Dusts and Fumes in Industry

(Sources, Effects and Control)

Introductory Statement

In dealing with occupational diseases and their effects, the attending physician should keep in mind that the etiology of these conditions is present in the industrial environment — if the conditions are to be considered truly industrial in origin.

It is likewise important to understand that rational diagnosis and treatment rests upon a fairly accurate knowledge of the etiology of the disturbances; this is especially true of occupational diseases and allied disorders.

Moreover, employers, industrial hygiene and medical departments are greatly interested in the prevention of various maladies which ordinarily affect employees; this clinical and economic interest on the part of industrial management further emphasizes the need for understanding the etiological factors in the working environment, so that they may be properly controlled.

This booklet on industrial dusts and fumes is the second of a series of which the purpose is to show the most important effects of occupations, show how these occur, give measures for treatment, diagnosis, and prevention, meanwhile considering the source or cause of such conditions. It is hoped that these simple and brief expositions will be of considerable help to industrial physicians, and to private practitioners who have among their patients employees of industrial plants subject to such exposure.

Definitions

Dusts may be defined as solid particles of matter which are ordinarily produced by grinding, crushing, drilling or blasting

processes, the composition of the particles being the same as that of the parent materials. These materials may be rock, metals, ores, or such products, for example, as coal, grains, or wood. The size of the particles varies from what is called ultra-microscopic to those which are visible. Authorities now agree that the hygienic significant particle size of dust ranges from a fraction of a micron to 10 microns. (A micron is 1/25,000 of an inch). The most frequently encountered dusts in industrial processes are mainly those from inorganic minerals, and a few organic dusts, such as wood and flour.

Fumes may be defined as solid particles which are formed by processes similar to combustion, condensation and sublimation. The most common types of fumes encountered in industry are usually caused by the oxidation of a metal, of which typical examples are the oxides of lead or zinc. Particle size is usually smaller than dust and ranges below one micron. In contrast to dusts, fumes tend to flocculate very easily. Many people erroneously speak of fumes when they mean vapors of liquid materials, such as acid solutions, organic solvents, and the like.

Incidence and Etiology

Dusts are the most common type of air contaminants encountered in industrial processes, and can occur whenever various materials, as mentioned before, are subjected to processes which break them up into fine particles; or where powdered or pulverized materials are handled or transported. Inorganic dusts are the prevailing type so far as frequency is concerned, but organic dusts are also found in different industrial operations.

Fumes are usually found wherever there are molten metals such as lead, zinc, and calmium being processed or used in industrial operations. Welding is an operation which has greatly increased in frequency of use and it may give rise to fumes of the metals which are welded or to fumes and gases from the variegated coatings of welding rods.

It is to be borne in mind that metals may produce both dusts and fumes, according to the nature of the operation and the state of the materials when processed. For example, among the heavy metals such as lead, dust may be of equal or greater etiologic importance with fumes — other mineral dusts and metallic dusts also are considered of etiological importance, as the following outline will show:

I. *Organic dusts:*
   1. Non-living organic dusts:
      (a) Toxic and/or irritant dusts; "(Various organic compounds such as trinitrotoluene, mercury fulminate, tetryl)."
      (b) allergy-producing dusts (woods, pollens, and the like).
   2. Living organic dusts:
      (a) bacteria
      (b) fungi

II. *Inorganic dusts:* (mineral dusts)
      (a) toxic and/or irritant dusts; (mainly the dusts from heavy metals and their salts, such as arsenic, lead, manganese.)
      (b) fibrosis-producing dusts, (examples are free silica and asbestos).
      (c) non-fibrosis-producing dusts; (the so-called inert dusts, such as alundum, corundum, emery, limestone, magnesite, marble, plaster-of-paris, and polisher's rouge).*

The most important of the non-metallic, inorganic mineral dusts is free silica, which produces silicosis, if breathed in sufficient quantities over a long period of time. Free silica dust and

* This classification is in accordance with the present state of knowledge. Future studies and experience will probably show some of the so-called inert or nuisance dusts to be harmful.

asbestos dust are the two kinds of mineral dusts which produce disease entities corresponding to the names of the dusts — silicosis and asbestosis. Mixed dusts of various kinds, such as various ores which contain different amounts of free silica, can produce different types of reactions, according to the kind of minerals which are in the ores. Where, for example, there are mixtures of iron, and various calcium-containing compounds such as gypsum, with free silica, a protective influence is exerted against the action of free silica on the lungs. Other modifications of free silica action occur according to the types of minerals present some of which tend to inhibit or restrain the action of free silica.

Another important conception with reference to the etiology of dust and fume diseases which may result from exposure to these classes of materials, is the use of the terms *exposure* and *hazard:* they are not synonymous — exposure merely means contact with some substance which is a *potential health hazard;* whether it is an *actual health hazard* can only be determined by an industrial hygiene engineering survey, in which the amount and kind of exposure is rated, according to accepted standards of concentration, size, and quality of dusts and fumes. Many persons erroneously therefore state that there is a lead hazard, for example, when it is only known that contact is being made with lead; and when there are no data to support the contention that there is an actual hazard present in the working environment.

Methods for the determination of dusts and fumes in the air, also with reference to the types of devices used for such determinations, will be mentioned later. The importance of air contamination will be realized when it is stated that the pathologic effects produced by dusts and fumes are brought about almost entirely by the inhalation of these compounds and materials. There is only one outstanding example, for instance, of the possibility of absorption of lead through the skin, namely tetraethyl lead, and this occurs in the form of a fluid. No dusts, so far as is known, produce any systemic pathological condi-

tions from skin absorption, and it is doubtful that any kind of true fume is absorbed by the skin.

From the etiologic point of view, particularly with reference to pathologic effects, the following outline shows how different types of substances (among the dusts and fumes) act on various systems in the body to produce pathological effects:

1. *General systemic poisoning:*
   Examples: Arsenic, lead, manganese, mercury.
2. *Substances acting on the circulatory system:*
   Examples: Arsenic, lead, mercury, vanadium.
3. *Substances acting on the respiratory system:*
   Examples: Asbestos, free silica.
4. *Substances acting on the gastro-intestinal system:*
   Examples: Antimony, cadmium, lead, mercury, zinc.
5. *Substances acting on the skeleton and joints:*
   Examples: Lead, mercury, mesothorium, phosphorus, radium.
6. *Substances acting on organs of special sense:*
   Examples: Arsenic, lead, manganese, mercury.
7. *Substances acting as fetal poisons:*
   Examples: Lead, mercury.
8. *Substances acting on the genito-urinary system:*
   Examples: Arsenic, lead, mercury, vanadium.
9. *Substances acting on the brain and nervous system:*
   Examples: Arsenic, lead, manganese.

General Diagnostic Methods

The accurate diagnosis of pathological conditions arising from the inhalation of industrial dusts and fumes has tremendous economic significance and also it is of medicolegal importance. Moreover, it is impossible to understand how proper and effica-

ceous treatment may be rendered unless a scientific diagnosis is made. If a claim results from the pathological condition alleged, it is evident that the disease sustained must be directly related to the employment and further, must produce disability to entitle the individual concerned to compensation.

Diagnostic methods with reference to dust and fume diseases need not differ widely, if at all, from those used as routine procedures in general scientific medicine, although there are specific methods as in any other application of general medical diagnostic principles to any special group of persons. New discoveries occur relative to diagnostic methods in occupational disease work and for the most part, reliance must be placed on information which is scattered throughout the current literature in addition to available textbook material which is better now than it was even five years ago.

Emphasis has already been placed upon the vital part played by etiology; it is wise to repeat that etiology forms the bulwark of the diagnostic and therapeutic background.

Among the methods which are used to make a fairly accurate diagnosis with reference to occupational diseases, is the taking of a thorough industrial history; consideration of symptomatology, the physical findings, the results of air examination; and finally, last but not least, a differential diagnosis, ruling out diseases of ordinary life which have no relation to occupation.

The industrial history may be of very great assistance in arriving at an opinion. Most industrial physicians of considerable experience believe that it is desirable to include a complete resume of the kinds of occupations which have been experienced by the individual being examined, dating from the first job to the last one. It is of course clear that the details of actual occupational exposure cannot be evaluated by this method, but important information bearing on the diagnosis may thus be obtained. It is the present practice, therefore, to list the occupations with the dates of occurrence, in the industrial history. It is

furthermore advisable to get more detailed information relative to the performance of the occupation, if that can be obtained. This will at least give information relative to the kind of exposure in industry, although it will give no idea of the severity of exposure.

The symptomatology in many kinds of occupational disease is likely to be confusing rather than useful, because in many instances it so closely simulates that of non-industrial disease that it is quite commonly not characteristic. Especial attention should be given, however, to the recording of the symptomatology and critical attempts made of its proper evaluation. Here again, distinction must be made between symptoms which may be due to non-occupational disease and those symptoms which are specific for occupational diseases — this is where the difficulty arises in adequately assessing symptomatology.

The physical findings, on the whole, are more reliable than the symptomatology, although in certain instances they too are not characteristic and are, therefore, liable to be somewhat puzzling. Where only one portion of the body is affected, as in silicosis, for example, it might be assumed that the physical findings would be more characteristic, but the opposite of this is usually true. In other instances, for example lead poisoning, where different organs and systems are pathologically affected, the picture is liable to be one which demands expert evaluation to differentiate from non-occupational conditions.

Clinical laboratory data, including X-ray findings, are frequently found to be decisive, although here again intelligent appraisal means proper interpretation of the data. When they are decisive, such tests are by the very specificity of their nature, of great value as diagnostic aids, although they should be definitely correlated with other findings.

Differential diagnosis presents perhaps the most difficult problem of all in the realm of diagnosis of occupational diseases. There is good reason to believe that differential diagnosis will

continue to be one of the most difficult phases of the medical ramifications of the medicolegal problems of occupational diseases. The reason which has already been indicated is that the symptomatology and frequently the physical findings imitate so closely the symptomatology and physical findings of non-industrial diseases, that one must make use of specific procedures which are designed especially as diagnostic guideposts. Frequently such specific procedures are not used to any great extent, except by those who are highly specialized in the field and whose work takes them into a review of the current literature containing these developments. For example, one may believe that an industrial employee has lead poisoning, largely because of second-hand knowledge that lead was used in his occupation, more easily than one could demonstrate adequate etiology and the existence of lead poisoning by scientific methods.

Because of all of these obvious and other difficulties, it seems wise to adapt specifically certain examination procedures to industrial problems arising out of occupational or suspected occupational diseases. Most authorities now agree that the examining physician can be of greater service and give more efficient advice regarding either acceptability for employment, continuance in it, opinion on the diagnosis of systemic pathologic conditions and the estimation of disability, if he is conversant with the following points:

1. The nature and extent of the industrial exposure into which the employee will go or where he has been. This can be determined only by scientific methods which will be referred to later.

2. The effect of this specific exposure upon normal and abnormal physical states of employees, as interpreted by the physical status of each individual on examination. (This refers specifically to etiology in the industrial environment and the effects which such etiology may or could produce.)

3. The nature of the protective devices supplied by the employer, if a hazardous exposure cannot be otherwise controlled, and as to whether these devices are functioning efficiently. (Reference will be made later to these devices, especially designed for protection against dust and fume exposures.)

It is realized that these procedures are somewhat removed from the usual program of physical examinations, particularly with reference to the work of part-time industrial physicians, but it is also expected that the physician will be called upon increasingly to know and appreciate such information so that he may function better as a diagnostic medium.

Because the most frequent and most significant exposures in industrial processes are free silica dust and lead and its compounds, it seems advisable to include in the next section some clinical examples, namely silicosis and lead poisoning; there will also be included a brief discussion of metal fume fever.

Clinical Examples

Simply stated, silicosis is a disease of the lungs, caused by the continuous inhalation over a long period of time of very finely divided, microscopic, particles of free silica dust. There have been various standards set up by different agencies, based upon studies of silicotic workmen and their exposure to free silica dust. The National Silicosis Conference Report on Medical Control of Silicosis says: "There is evidence that for prolonged exposure a concentration of more than five million particles per cubic foot of a highly siliceous dust is dangerous. Therefore, it is now considered good practice to hold concentrations of highly siliceous dust at 5 million particles per cubic foot, or less."

Because of the fact that industrial exposures, involving high free silica content in which the concentration of dust should be restricted to five million particles per cubic foot, are relatively infrequent in industry, and also because the opposite usually

occurs (an exposure to "mixed" dusts which contain appreciable amounts of free silica but also contain considerable amounts of other types of material) it is believed wise to refer to the formula* developed by the National Silicosis Conference with reference to a working criterion with respect to satisfactory or unsatisfactory conditions, when exposure to 'mixed' dusts occurs.

It is to be borne in mind, however, that any unprotected industrial operation, which involves the production of a lot of fine dust particles of material containing a relatively large percentage of free silica, offers a potential silicosis hazard.

The incidence of silicosis is not known with any degree of accuracy, although various estimates have been made. One of these made some time ago by the American Institute of Mining and Metallurgical Engineers indicated that in the United States the number of industrial workers exposed to the silica dust hazard is in excess of a half million. Figures quoted by the Committee Reports of the National Silicosis Conference disclosed estimates that approximately 2% of the working population of the United States or about one million workers are exposed in some way to the potential hazard of silicosis, and that perhaps half of this number, 500,000, or 1% of the total, are exposed to a serious hazard. It is furthermore believed that approximately 110,000, or 0.2 of 1% of the total, have silicosis to some degree, but likely only 4000 to 5000 (a small fraction of 1% of the total) suffer any degree of disability from the disease. (These figures are based upon prewar estimates.) It can readily be seen that silicosis, although not a rare disease, is not common and furthermore not as common as may ordinarily have been supposed by many persons.

Recent conceptions with regard to the contraction of silicosis have established the belief that approximately 25% of those exposed to a real silica hazard will eventually develop silicosis.

* FORMULA A: "Multiply the percentage of free silica by the total dust count. If the result is under 5 million, the condition may be considered permissible. If the result is over 5 million, the condition may be considered too high. For example, 10 per cent free silica with an average total dust concentration of 50 million particles per cubic foot would give 0.10 times 50 million, which equals 5 million (good practice). 10 per cent with an average total dust concentration of 50 million, would equal 0.3 times 50, or 15 million (unsatisfactory). This formula is not applicable to any dust containing less than 5 per cent free silica."

Clinical Examples

With respect to the occurrence of silicosis in the foundry industry, a prewar survey by the Division of Industrial Hygiene of the New York State Department of Labor, based on a study of 311 foundries, of which 80 plants included X-ray examinations involving 4,754 employees, provides some interesting figures: the X-ray summary showed that 3,259 or 80.2% were negative; 185 or 4.5% showed fibrosis; 110 or 2.7% showed silicosis with and without infection; 485 or 11.9% showed healed primary and re-infection types of tuberculosis; 27 or 0.07% showed clinically significant tuberculosis. It was concluded, as stated in the report, that it would appear that the silicosis hazard in the foundry industry in New York State, while existent, is of mild degree of severity and may be expected to yield satisfactorily to appropriate measures of control. The report further indicated that tuberculosis as manifested by X-ray films of the chest was found to be more prevalent among silicotics than among non-silicotics in foundrymen.

The occupational history, as previously indicated, is of considerable importance. Forms for the recording of occupational records have been devised by various organizations and groups and by physicians and insurance companies. One of the most complete and longer forms has been devised and used by the Saranac Laboratory, Saranac Lake, N. Y., in connection with silicosis studies and symposia on silicosis which have been held by this group. It is suggested that the physician, if interested, should contact the industrial hygiene bureau of his state health department through which detailed forms may be secured or information relative to them.

The symptoms and physical signs are not characteristic; they may be confused even by skilled diagnosticians in other diseases, with the symptoms and signs which commonly occur in both minor and the more serious types of the well-known respiratory infections. Characteristically, shortness of breath on exertion and decreased chest expansion frequently occur in advanced cases of silicosis, but these findings must also be differentiated as

to their casual relationships and their possible association with other diseases.

The clinical confirmation of the diagnosis of silicosis rests mainly on the characteristic X-ray appearance of the chest. This shows the typical lesions of silicosis, hyaline fibrous nodules which are scattered as opaque bodies throughout both lung fields, the density of these nodules being somewhat less than calcified bodies, but discrete in outline, all about the same size in the early stage (so-called millet-seed size) and evenly distributed throughout both lung fields. These nodules appear as the first indication of silicosis, later become somewhat confluent and in the latter stages, there are large masses of opaque material found near the periphery of the lung fields. In the later stages of silicosis, the disease is frequently complicated by infection, usually tuberculous in nature. As an aid to interpretation of the X-ray films of the chest where silicosis may be suspected, it is suggested that the physician refer to a tabulation of the X-ray changes in silicosis, with the underlying histological conditions which cause them, published in the transcript of the Second Silicosis Symposium, Saranac Lake, N. Y., 1935.

Diagnosis rests upon proof of occupational dust exposure sufficient to produce the disease, and the presence of characteristic nodular lesions in the X-ray film of the chest. The mere mention of occupation is not sufficient for diagnostic purposes — adequate knowledge concerning the quality and quantity of dust exposure, as well as the length of the employment period, is necessary to establish a distinct causal relationship.

Although considerable diagnostic reliance is usually and properly placed upon the history of adequate occupational dust exposure and the characteristic chest X-ray appearance, a thorough physical examination with appropriate clinical laboratory examinations should be done, especially with reference to the ruling out of other respiratory diseases. Diagnosis becomes essentially a differential diagnosis, as before explained, one which is likely to be difficult, necessitating special experience

and knowledge. Some of the conditions requiring differential consideration here are miliary tuberculosis, multiple calcifications of healed tuberculosis, cancer of the lungs, and fungous growths of the lungs.

Within recent years, superimposed infection has come to be the accepted cause for disability. The outlook in simple silicosis, therefore, (that is, without infection) is more favorable than was formerly believed. It is obvious that the chief problem for the silicotic is to avoid superimposed infection, which is usually tuberculous.

There is no one specific, efficacious treatment for silicosis, although in recent years, the inhalation of metallic aluminum powder has shown some promise. In cases where tuberculosis complicates the picture, it is obviously to be treated by the best known methods for that disease. There is some recent evidence that surgical therapy as used in ordinary forms of tuberculosis is also a help in the type of tuberculosis which follows silicosis. The evidence for this is not conclusive, but there are good results on record.

In general, recommendations for the treatment of cases of silicosis vary with experience and with the findings of each case. It is believed, for example, that no treatment is necessary in the case of an individual without symptoms, whose X-ray film of the chest (taken perhaps in a routine industrial survey) shows only widespread discrete nodulation without localized shadows indicating infection. However, the employee should have serial X-ray examinations with physical examinations of the chest at least once a year and should be removed from further silica exposure to some other job, since the disease is very slowly progressive and there is at this stage no disability which precludes continuation in employment. A person with localized conglomerate lesions may have symptoms and usually cannot do as heavy work as formerly; therefore, a lighter job should be found. In the presence of active infection where there are symptoms or incapacitation, the individual cannot obviously continue in his

former employment. If the case is one of open tuberculosis with positive sputum, it is evident that such an employee is a source of danger to his fellow workmen and should be placed in a sanitarium or hospital. In cases of inactive infection with no incapacitation, lighter work should be provided, without further dust exposure.

Dusts containing silica in combined form do not cause silicosis; however, one such dust, asbestos, a magnesium silicate, will produce a characteristic fibrosis which is called asbestosis. This disease is quite different in its pathology from silicosis, and has a characteristic 'ground-glass' appearance on the X-ray films of the chest. Animal experimentation indicates that the action of asbestos is probably mechanical, since it cannot be produced in other than the pulmonary tissue. Asbestosis may cause disability and a few fatal cases have been recorded, but in those cases showing marked disability, other organic disease is usually also present. The symptomatology and physical findings include cough, dyspnea, pallor, clubbing of fingers, and diminished chest expansion, with a characteristic X-ray appearance as previously mentioned.

Lead Poisoning

Lead poisoning offers a good example of an industrial poisoning which is contracted by the inhalation either of a toxic dust or fume, and has been stated to be one of the most frequent and important types of metallic poisoning in industry, on account of the widespread use of lead and its compounds.

Although there are no reliable statistics available with respect to the national incidence of lead poisoning, it is believed that lead poisoning is very prevalent and that most of it is mild. The incidence of severe types of cases, particularly lead encephalopathy and the more severe types of nervous system involvement are not seen with as great frequency as in former years. Many physicians who are used to handling industrial cases now rarely see a case of wrist drop due to lead poisoning, whereas 20 to 25 years ago it was a rather common phenomenon, in cases of lead intoxication.

Clinical Examples

The industrial history is of great importance, as previously indicated, and a thorough consideration of the nature and extent of the exposure to lead and its compounds is an essential point in the etiology. It is now generally accepted that the standard of the U. S. Public Health Service and the American Standards Association of 1.5 milligrams of lead per ten cubic meters of air, is the maximum allowable concentration. Because it is now conceded that practically all industrial lead poisoning is contracted by the breathing of lead dust and fumes, it becomes of considerable importance in working out the etiology to ascertain the amount and quality of lead dust and fumes in the air, during the occupation of the involved individual. This point will be discussed later with reference to prevention.

Formerly, the nature and variety of non-industrial sources of lead intake made it particularly difficult to establish industrial etiology on a sound and competent basis. More recently, however, non-industrial lead intake has been measured by scientific methods and with the exception of an occasional difficulty with various types of homemade beverages stored in utensils glazed with a lead compound, the non-industrial lead intake is not believed to be a considerable factor in the production of lead poisoning in adults. There are, however, some exceptions and these should be taken into account, as indicated.

The diagnosis of industrial lead poisoning is not a simple matter on account of the fact that clinical symptoms and signs are protean and are liable therefore to simulate other diseases. Great increase in our knowledge, however, has occurred within the past ten to fifteen years, and it is now known that there is a close relationship between the action of lead and its compounds within the body and the calcium metabolism, and further, normal and abnormal excretion of lead has been worked out on a scientific basis. It is now an accepted fact that persons without industrial exposure normally excrete a certain amount of lead in the urine. It has been stated that definite clinical findings of lead poisoning are practically always associated with abnormal urinary lead

excretion — the reverse, however, is not true — that abnormal urinary excretion of lead in the absence of characteristic clinical findings indicates lead poisoning.

Other laboratory findings are important, particularly the examination of the blood, some investigators and clinicians using the increase in basophilic stippled red cells as an indication of positive findings and others using what is known as the basophilic aggregation test. Both are found to be successful, the former more adaptable in cases of frank poisoning, the latter more useful in cases of lead absorption and suspected incipient poisoning.

Although as indicated, the clinical symptoms and signs of lead poisoning are protean, the principal effects are expressed in the blood, the gastrointestinal system, and the nervous system, named in order of their usual chronological occurrence. In brief, the most consistent findings are changes in the color and form of the red cells of the blood, colic and constipation, and weakness and atrophy of the extensors and flexors of the muscles of the wrists, with partial paralysis in the more advanced types of cases.

The differential diagnosis, as may be obvious, has to do mainly with differentiating industrial lead poisoning from non-industrial diseases which may involve the blood, the gastro-intestinal system, and the central and peripheral nervous systems, producing changes which are similar to those observed in frank lead poisoning. Such non-industrial diseases show a considerable variety of pathological processes affecting the above-named systems.

The treatment of lead poisoning has undergone considerable change within the past ten to fifteen years. In general, the treatment consists mainly of appropriate therapy for the toxic episodes and the mobilization of stored lead in the long bones. It is usually agreed that it is wise to facilitate the storage of lead by an excess calcium intake rather than to set lead free in the blood stream; after the passing of acute toxic episodes, the elimination of lead may be assisted by means of low calcium intake, the administra-

tion of weak organic acids and ammonium salts, sodium bicarbonate, and potassium iodide. Care should be exercised in using such forms of treatment on patients who have nephritis.

Two schools of thought with widely divergent views exist with reference to deleading the body: there are those who feel that in the majority of instances deleading will accomplish good results, while there are others who adopt a conservative attitude and because of the possibility of precipitating acute symptoms, feel that it is safer to produce artificial deleading by slow methods, or to resort to the natural process of letting the body delead itself over a period of time. The choice in the matter depends upon the individual case and the previous results which have been achieved at the hands of the physician in charge of the case.

Storage of lead may be facilitated in acute cases by a high calcium diet or by the slow intravenous injection of 10 cc of 20% calcium gluconate; variations with relationship to this form of treatment have been devised by various physicians. In the deleading process, low calcium diet together with oral administration of ammonium chloride has been found to be effective, whereas in certain cases parathyroid extract and sodium bicarbonate also have been useful. Within recent years it has been established that large doses of viosterol have been instrumental in increasing the excretion of lead. Other investigators have found that an adequate phosphorus intake is essential for the depositing of lead in the bones and for the removal from the bones of the excess cations, as the insoluble phosphate salts in the feces; it has therefore been recommended that deleading may be accomplished by giving a diet low in calcium and relatively high in phosphorus, using viosterol 20 minims daily.

There are also variations of treatment with reference to the amount of intoxication, and where there has been high, medium or low lead intake in industrial occupations. In all these types of treatment, deleading should be done in the hospital where the patient may be closely supervised. Some investigators advise

against deleading more often than once in four weeks and the course should not be prolonged for more than three or four days; some physicians believe that it may take from six to fifteen courses of deleading to free the individual of his most loosely-combined lead, which of course is the most dangerous type of lead. It is again warned that deleading should not be undertaken in the presence of acute symptoms; it is further agreed by all investigators that calcium therapy is considered to be the most specific at the present time.

The outlook in cases of lead poisoning is much better now than formerly because of the occurrence of moderate types of effects, the severer forms being a great deal less frequent than in former years.

Metal Fume Fever

This name is applied to the condition which results from breathing fairly heavy concentrations of freshly formed metallic oxide fumes, principally zinc and one of its alloys, brass; although the condition may result from the inhalation of other metallic oxide fumes. Metal fume fever is not a systemic poisoning, but it should be borne in mind that some of the metallic fumes that can cause it, such as lead, cadmium, mercury, and manganese, may also cause systemic poisoning.

The symptoms come on some hours after exposure, usually after leaving work, a dryness of the throat being first noticed, which may be followed by a dry cough and a feeling of heaviness in the chest. Rise of temperature, accompanied by sweating, and leucocytosis and elevation of the pulse rate and the blood pressure usually follow, sometimes with chills. The symptoms ordinarily pass off in 12 to 24 hours and the usual experience is that the employee returns to work the following day. A natural immunity is conferred in certain types of workers, which seems to be lost rapidly after removal from continuous exposure.

Diagnosis is made usually on a clear history of industrial exposure to metallic oxide fumes. It must be remembered that

the effects and symptoms of metal fume fever are very similar to those of influenza and that the malady is more often noticed in the fall and winter, when influenza usually occurs. The industrial history is, therefore, important in diagnosis, particularly with reference to a causative exposure to metallic oxide fumes.

The outlook is good for complete recovery in a short time and usually there is no time lost from work, although there may be temporary incapacity in terms of hours.

The treatment is symptomatic and especially directed toward comfort because the patient usually recovers within a relatively short time in all events. Rest in bed with adequate warmth and protection from drafts are recommended. However, all suspected cases of metal fume fever should be attended by a physician because the symptoms may closely simulate influenza which makes the differential diagnosis important.

Other Dusts and Fumes

It must not be concluded that although clinical examples were given of silicosis, lead poisoning, and metal fume fever, there are not other examples which are of importance. These subjects were selected because of their frequency of occurrence and because of the fact that they are commonly encountered as examples of dust alone, dust and fumes, and fumes.

Among the other metals which may be mentioned are arsenic, antimony, cadmium, manganese, mercury, radium, selenium, silver, tellurium, thallium, and vanadium. Not all of these are equally important with reference to toxic effects.

Probably the most important of these from a toxic point of view are arsenic, mercury, manganese, and cadmium. Arsenic produces chronic poisoning chiefly from breathing over a long period of thin air containing dusts or sprays of arsenic compounds. The compounds of arsenic such as lead arsenate, paris-green, arsenic oxide and arsenic sulphide are among the more dangerous forms.

Where dusts of mercury are present, they will be in the form of either mercurous or mercuric compounds, where metallic mercury is used in the manufacture of such items as thermometers, mercury switches, mercury vapor lamps, mercury boilers, etc., the hazard is from the vapor since mercury has a considerable vapor pressure even at normal temperatures. Mercurialism is characterized by three features: inflammation of the mouth, muscular tremors, and psychic irritability — sometimes all of these are encountered and sometimes only two or only one. In the more chronic or slow form of poisoning, a characteristic tremor is the commonest symptom, coming on late, and has been noted to occur particularly among mercury miners.

Manganese poisoning is an old industrial poisoning, having been described more than 100 years ago, but has receded in incidence in American industry. Characteristically it produces a peculiar slapping gait with phenomena of retropulsion and propulsion; masklike faces; monotonous voice with economical speech; muscular twitching varying from a fine tremor to gross rhythmical movements of the extremities; languor and sleepiness; cramps in the calves of the legs with stiffness; slight increase in the tendon reflexes without any disturbance of the deep or superficial sensation. Studies within the past five years indicate that dust is the most important type of exposure.

Cadmium is encountered in the smelting of ores, the handling of socalled 'blue powder' compounds, the spraying of pigments, the welding of alloys, and the melting of cadmium metal itself, as well as certain non-industrial exposures where utensils have been plated with this metal. In recent years there have been several outbreaks of cadmium poisoning among non-industrial users of such utensils. The use of the metal has increased tremendously and therefore, there is more industrial exposure. Two types of effects have been observed, those with reference to the lungs and also those with reference to the gastro-intestinal tract, the former being more important and more frequent. Pulmonary involvement is characterized by a feeling of constriction in the

chest, edema and congestion of the lungs, sometimes with hemorrhage, proliferative interstitial pneumonitis; while the latter is characterized by nausea and vomiting and a general irritative reaction of the gastro-intestinal tract, with diarrhea, hiccough, and oliguria.

If further details are desired on the clinical aspects of the metals mentioned, standard works on industrial hygiene and occupational diseases should be consulted.

Prevention

The general principles of prevention involve first the identification of industrial exposures and the evaluation of such exposures by proper scientific methods to determine whether or not they represent actual health hazards. This is done by sampling the air and determining the amount of air contaminants and the quality of them in each type of occupation or problem involved, and comparing the results with the accepted standards of maximum allowable concentration that have been adopted by official bodies. Second, after it has been determined that there is an actual health hazard represented by the occupation, control measures, both engineering and medical, are instituted to control the hazard, providing it cannot be entirely eliminated.

In discussing the methods of engineering control, we are concerned with three basic principles:

1. Investigation of occupational conditions.
2. The measurement of the occupational exposure and the rating of the hazard, if one exists.
3. Putting into effect the specific measures of protection which have been indicated by information secured under (1) and (2).

Among the methods of approach for engineering control measures may be mentioned:

1. Physical and hygienic survey of plant conditions.

2. Occupational history and analysis.
3. Adequate information concerning materials and processes used.
4. Atmospheric and gross sampling of materials, combined with quantitative physical and chemical determinations.
5. The rating of the hazard by comparing the findings in the given instance with accepted standards.
6. An appraisal of the efficiency of protective apparatus and devices.
7. An adequate and proper interpretation of the information so that it can be applied practically to the industrial problem involved.

When the foregoing methods of approach to engineering control have been carried out and information secured from them, the information can indicate the necessity for using specific methods of protection which have been found useful in different combinations:

1. The use of mechanical devices, such as exhaust ventilating systems, respirators, masks, and the like.
2. The changing of processes (segregation, enclosure, wetting, etc.).
3. Substitution of nontoxic materials for those of proved toxicity.
4. Educational programs for management, workers, engineers, and physicians.
5. Continuous maintenance and checking of the efficiency of protective equipment.

Atmospheric Sampling

The best method for collecting a sample of a dust or fume from the air will depend somewhat upon the type of contaminants to be sampled. The degree of hazard of fibrosis-producing dusts is measured in terms of particles per unit volume of air (example:

10 million particles of silica dust per cubic foot of air) while toxic dusts and fumes which may cause systemic poisoning are usually measured in terms of weight per unit volume of air. (Example: lead dust 1.5 milligrams per 10 cubic meters of air.)

Since results may vary with the type of air sampling device used, it is important to select a type accepted through general usage in the practice of industrial hygiene.

The larger Impinger which samples one cubic foot of air per minute by impinging the air containing the dust particles from a nozzle submerged in water or alcohol against a plate or bottom of the flask was used in all of the original studies in the United States on fibrosis-producing dusts. The large Impinger, however, has the disadvantage of being bulky and requiring electric current or compressed air for operation of the motor-driven air pump, which produces the suction stream for air sampling.

A portable Midget Impinger has been developed which has been proven to give results as reliable as the large Impinger. This apparatus samples at the rate of 1/10th of a cubic foot per minute and is operated by a hand cranked, positive action, four cylinder pump, in conjunction with a vacuum regulator which maintains an unfluctuating suction regardless of minor variations in rate or smoothness of cranking.

After the sample has been collected in the Impinger flask, a portion is placed in a special dust counting cell and counted under a microscope with an eyepiece having a special ruling similar to a blood count ruling.

Further analyses are necessary with reference to the amount of free silica in the dust and this is accomplished by the use of specific chemical, petrographic and X-ray diffraction methods.

The Impinger method of sampling is not entirely suitable for collecting fumes or dusts of toxic materials such as lead, cadmium, and others because the efficiency of the Impinger is not adequate when sampling the very fine fume particles and since the amount

or concentration of such materials in air which may be injurious is much smaller than the safe amounts of fibrosis-producing dusts, a correspondingly large air sample should be taken for reliable results.

The Electrostatic Precipitator is widely used in the collection of toxic dusts and fumes, particularly of metals and their compounds. A Portable Electrostatic Precipitator has been developed which operates from 110 volts, 60 cycle current, and by high voltage electrical precipitation collects the sample of the particulate matter dry on the inside of aluminum or glass cylinders. This device samples at the rate of 2 cubic feet per minute. After the sample is collected, a quantitative chemical analysis is made in the laboratory. This type of apparatus permits determinations of very low concentrations of many different kinds of atmospheric particulate matter.

Where equipment or personnel is not available for atmospheric sampling, state industrial hygiene bureaus or private testing laboratories may be engaged. Many insurance companies also have facilities for such work. The Industrial Hygiene Foundation in Pittsburgh, Pa., an association of industries for the advancement of healthful working conditions, also have an excellent staff of technicians for industrial hygiene surveys or consultations in this field.

Respiratory Protective Equipment

It frequently happens that because of the nature of the operation or process in an industry, it is not possible to thoroughly control a hazardous exposure resulting from the evolution of dusts and fumes. In such instances, it is considered wise to provide employees with personal respiratory protective devices. These vary with the kind of exposure and with the amount of materials in the air.

For ordinary conditions in which only dusts and fumes are present, simple filter type respirators are available which will

protect the worker against the hazards of breathing these dusts and fumes.

Such respirators normally consist of a facepiece covering the nose and mouth, made of some pliable material such as rubber which will conform to the face and provide a comfortable and dust-tight fit, and to which is attached a filter made of treated paper or felt through which the air is inhaled. If the filtering material is of the correct type and density, it will effectively filter particles down to less than a micron in size from the air.

Comfort is a most important consideration in a device of this type. Since a high breathing resistance will materially affect the comfort of the wearer, the filters should be of large area so that the inhalation resistance will be as low as possible. Valves should be provided which open on exhalation and further lessen the exhalation breathing resistance.

The particular type of filter employed will depend upon the kind of contaminant present in the air. Since the amounts of such toxic materials as lead which will affect the wearer are much smaller than the harmful amounts of fibrosis-producing dusts, a filter will have to be more effective when used against toxic dusts than against fibrosis-producing and nuisance dusts.

For special operations such as sandblasting, a mechanical filter type respirator is not practical because of the extremely high concentrations of dusts present. In such conditions, special air-supplied masks or respirators should be used in which air from a compressed air line is supplied through a hose to the facepiece for breathing. Such devices may also be employed if desired as protection in less severe and hazardous operations. The advantage of an air-supplied respirator is that there is no breathing resistance and the wearer is protected against any and all dusts and fumes in the surrounding atmosphere. However, the restriction by the air hose which must be attached to the wearer at all times between his facepiece and the air supply, prevents the use of this device in many operations where the worker must move freely

over a considerable area. Furthermore, caution must be taken to see that the air supplied to such devices is free from all injurious or objectionable odors and contaminants and that the humidity and temperature of the air is such as to provide reasonable comfort.

The United States Bureau of Mines (Department of the Interior) tests respiratory protective devices against various air contaminants under carefully controlled conditions. Devices meeting its tests receive that Bureau's official approval, and this is indicated by approval numbers on the device and a reproduction of the certificate on the package. Equipment approved by the U. S. Bureau of Mines for the particular contaminant or class of contaminants in the air should be used.

MEDICAL CONTROL

It is of course obvious that medical control measures are futile after a disease has been contracted because of the exposure to dusts or fumes except when the information is adapted to prevent additional cases. However, before the diseases are contracted, there are a number of things that can be done by physicians to prevent such occupational diseases, which may be summarized as follows:

1. The intelligent use of preemployment physical examinations, including chest X-ray films, and appropriate clinical laboratory tests as indicated.
2. Similar use of periodic physical examinations, chest X-ray films, and clinical laboratory tests on employees in occupations where typical hazards have been proved to exist, by industrial hygiene investigations.
3. Responsibility for seeing that hazardous exposures are properly taken care of, by various efficient methods of protection, including the supplying of personal respiratory protection, where industrial conditions make it impossible to control the hazard by other means.

4. Prompt transference to non-hazardous occupations of employees found to have symptoms of an occupational disease associated with their exposure.
5. Prompt removal from work of all persons suffering from active occupational diseases, when such persons are a menace to themselves, their fellow workmen or the employer.
6. Adequate provision for treatment, rehabilitation, and prompt return to work, of employees having disability resulting from occupational diseases.

Summary

In this brief discussion of dusts and fumes in industry, the sources, effects, and control have been considered under the following headings:

1. Definitions.
2. Incidence and etiology.
3. General diagnostic methods.
4. Clinical examples.
5. Prevention — general principles, including specific methods of air sampling and apparatus for this purpose; specific methods of protection, including mention of respiratory protective equipment available; and other general methods.

It should again be emphasized that industrial managers are interested in the prevention of trouble that may arise from exposure to dusts and fumes in industry, and that this principle is particularly important nowadays because of the need for every person in active production, and in addition, because so many physicians are being used in the armed services.

It is believed that if the suggestions made in the foregoing pages are followed, the problems arising from dusts and fumes in industry can be successfully managed.

APPROVED

M-S-A EQUIPMENT

FOR

Dust and Fume Protection

MSA Protection for men and women workers against harmful dusts is the result of many years of research into all phases of every industry's respiratory protective problems. In a well-equipped Dust Research Laboratory, which is a special division of our Research Department, continual study of dust hazards is maintained, and from this research group have come many of the most important improvements in dust protection—dating from the first U. S. Bureau of Mines-approved dust respirator, the famous M-S-A "Comfo." The M-S-A trademark on the products you recommend is a solid assurance of excellence in design, manufacture and performance.

All applicable M-S-A personal protective equipment against harmful dusts or fumes is approved by the highest authority—the United States Bureau of Mines, official testing agency of the United States Government for safety equipment. The approval certificate of the Bureau signifies that each product has passed the most stringent impartial tests—fit for the most exacting service in industry.

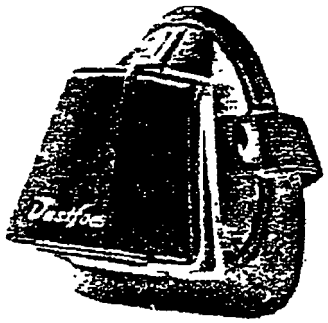
We will be glad to send you, upon request, descriptive bulletins on any or all of the products mentioned in this booklet—without obligation.

M-S-A® Filter Respirators

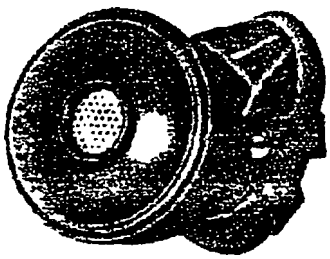
Designed for protection against harmful dusts and fumes by the use of interchangeable filters. Each of these Respirators answers all respiratory protective requirements against pneumoconiosis-producing, nuisance or toxic dusts.

M-S-A® Dustfoe 66

Bureau of Mines Approved. An improved compact and lightweight Respirator with larger exhalation valves reducing resistance to a minimum. These spring-retained valves eliminate disturbing variation in breathing resistance, and prevent accidental removal of valves. Facepiece cushion of neoprene sponge is contoured to provide automatic fit, great wearing comfort, and air-tight seal without adjustment. Electrostatically charged resin-treated filters supplement mechanical holding action of filter's fibers. Simple construction assures quick and easy filter changing and cleaning of Respirator. Weight, including headband: 3 ounces.



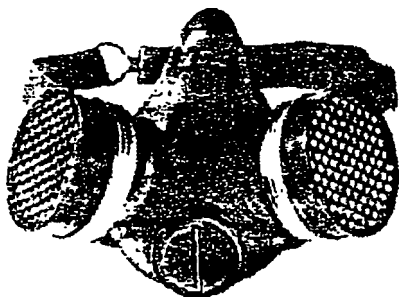
M-S-A Dustfoe *Ultra-Filter* Respirator



This compact and lightweight Respirator of aluminum is designed for unusually toxic dusts and extremely fine particles. Its filter holder allows for good field of vision and freedom of head movement. Facepiece forms to contour of wearer's face. Neoprene sponge cushion assures air-tight seal with minimum adjustment and maximum comfort. Neoprene stays soft and pliable; will not harden with age. M-S-A Type H Ultra-Filter protects against toxic aerosols, radioactive dusts and fine particle smoke; also effective against airborne bacteria and certain viruses. Construction is simple for easy filter changing and cleaning.

M-S-A Comfo Filter Respirator

Most versatile of the M-S-A Respirators. Facepiece is made of a special new soft compound for improved wearing comfort and greater durability. Provides dependable air-tight seal with wearer's face, and will not harden or crack with age. Positive-action exhalation valve offer little resistance to air. The Comfo features interchangeability of three filters: Type F (Bureau of Mines Approved) for dusts not significantly more toxic than lead; Type S (Bureau of Mines Approved) for dusts, mists and metal fumes; and Type H Ultra-Filters for toxic aerosols, *extremely* fine particles, radioactive dusts, bacteria and certain viruses.



M-S-A® Air Line Respirators

Designed for complete respiratory protection in contaminated atmospheres that are not immediately harmful to life. Each M-S-A Air Line Respirator features lightness in weight, comfortable fit, and freedom of head movement.

M-S-A Dustfoe Air Line Respirator

For Constant Flow Only

Bureau of Mines Approved. This Dustfoe Respirator is a supplied-air protective unit with a formable aluminum facepiece. Its streamlined nylon fitting at the bottom of the facepiece serves as a connector for the flexible corrugated air supply hose. Dual exhalation valves offer minimum breathing resistance, and an air diffuser prevents air blasts on the face. A standard chemical cartridge filter unit gives effective filtering of delivered air. Assembly includes aluminum Constant Flow Control Valve with automatic shut-off and trigger release.

M-S-A Air Line Respirator

with Comfo® Cushion Facepiece
For Constant Flow or Demand Flow

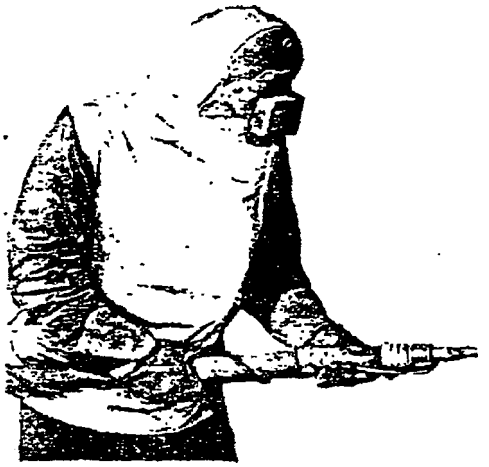
Bureau of Mines Approved. This Comfo Cushion Type Respirator is ideal protection from such hazardous classes of air contaminants as welding and cutting fumes, toxic dusts, paint spray vapors, and fumes from molten metals. The special shape of the facepiece with its floating chin cup and soft rolled edge assures gas-tight seal on the face. Double exhalation valves provide low resistance to air flow. A built-in air deflector assures proper circulation. Complete assembly includes either Constant Flow Control Valve or Demand Flow Regulator, with quick-disconnect coupling.

M-S-A Air Line Respirator

with All-Vision® Facepiece
For Constant Flow or Demand Flow

Bureau of Mines Approved. This "All-Vision" Type Respirator has been exclusively developed by MSA for use in contaminated areas where protection for face and eyes is necessary. The full facepiece assures greater working efficiency. The large lenses for wide-angle vision are fog-proof. The molded rubber body of the facepiece has an airtight seal which does not bind. All-rubber headstraps have swivel buckles for quick adjusting. Complete assembly includes either Constant Flow Control Valve or Demand Flow Regulator with quick-disconnect coupling.

For additional information on M-S-A Air Line Respirators and Accessories write for Bulletin No. 1009-B.



M-S-A Abrasive Mask

Designed for respiratory protection against heavy concentration of harmful dust encountered in shot and sand blasting operations. Durability of this air line respirator has been tested under impact of high velocity abrasives.

Bureau of Mines Approved. The comfortable, lightweight, gas-mask type face-piece is dust-tight and adjusts to fit all facial shapes and sizes. Fresh air feeds into the mask through a corrugated tube, and is deflected from the wearer's eyes to the lens to prevent fogging. Twin exhalation valves circulate air under the hood and form an air-cushion against the impact of abrasives. Wide-vision, safety-glass lens with protective screen can be removed from its hinged rubber frame. Flow control valve has replaceable charcoal cartridge filter to remove objectionable odors, oil and dirt. Abrasive mask assembly includes choice of lightweight or heavy duty hood, cartridge filter, web belt and air hose.

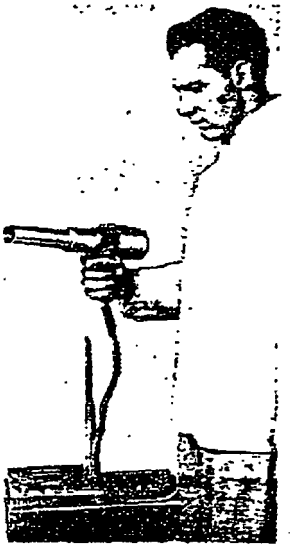
For additional information on M-S-A Abrasive Masks and auxiliary equipment write for Bulletin No. 1009-2.

M-S-A Electrostatic Dust and Fume Sampler

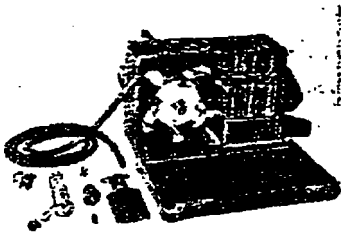
A modern precision instrument for quantitative sampling of airborne particulates including dusts and fumes in the ceramic, steel and chemical process industries, and those industries engaged in the production and use of radioactive materials.

The M-S-A Electrostatic Sampler is a portable unit used extensively by industrial hygienists, health physicists, ventilation engineers, chemists and those engineers concerned with air pollution. It works on the principle of preionization with electrostatic precipitation of airborne particles upon a collecting cylinder for determination by weight, count or chemical analysis. Accurate determination is enabled of very low concentrations of atmospheric particulate matter. The Sampler includes a portable sampling head consisting of ionizing electrode, collecting tube and motor-blower assembly; a power pack transformer rectifier assembly; retractable stand, sampling and collecting tubes, cables and parts, fitted in a sturdy carrying case.

For additional information on the M-S-A Electrostatic Sampler and Accessory equipment, write for Bulletin No. CT-9.



M-S-A Midget Impinger

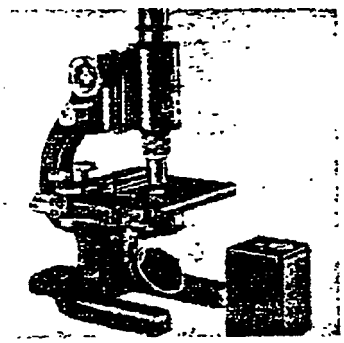


A portable hand-operated instrument for quick and dependable dust sampling in any location. Used in the dust surveys conducted by the U. S. Public Health Service and the U. S. Bureau of Mines.

The M-S-A Midget Impinger is ideal for collecting samples of dust in isolated locations and confined spaces without the use of compressed air or electrical current. This instrument employs the impinger method of drawing air at a relatively high velocity through a small glass nozzle into a liquid, causing the dust particles to be "wetted." The wetted samples are then available for accurate dust particle count under microscope. The impinger is operated by a hand-cranked 4-cylinder pump in conjunction with a vacuum regulator for unfluctuating flow at 1/10 cu. ft. per minute. The apparatus is contained in a convenient carrying case with shoulder strap, complete with flasks and nozzles. Weight is less than 10 pounds.

For additional information on the M-S-A Midget Impinger, write for Bulletin 0820-1.

M-S-A Dust Counting Microscope

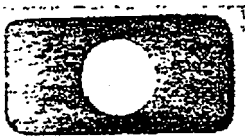


A lightweight, durably-built micrometer screw type instrument especially equipped and precision-adjusted and double-checked at M-S-A laboratories for dust determination. All mechanical parts are of high-grade workmanship and performance.

The M-S-A Dust Counting Microscope is a low-cost, lightweight instrument with optics identical to those offered in more expensive microscopes. It is finished in jet black satin enamel with rhodium-plated bright metal parts. The coarse adjustment is motivated by diagonal rack and pinion, with bearing surfaces provided with oil grooves. Adjustable draw tube fixed at correct tube length gives proper size field for dust counting. Magnification is 100 times. Housed in leatherette carrying case.

M-S-A Dust Counting Cell

A new cell for counting impinger samples, with an accurate depth of 1 mm. Absence of corners makes cell easier to clean and prevents leaks and bubbles.



For additional information on the M-S-A Dust Counting Microscope, the M-S-A Dust Counting Cell and auxiliary laboratory equipment, write for Bulletin No. 0820-1.

DUSTS AND FUMES IN INDUSTRY

M-S-A® . . . AND INDUSTRY

Throughout the many types of American Industry are the products of this Company—the largest commercial manufacturer of approved safety equipment in the world today. In 1914, Mine Safety Appliances Company was formed (as its name would indicate) to serve the mining industry. Today, the Company has far outgrown its original field, to include the design and manufacture of approved safety equipment for every major industry and the public protective services.

The Company's program is founded squarely upon Research—research which is concerned with all industrial hazards to the life and safety of the worker, conducted in MSA's extensive laboratories, by scientists of national reputation. Laboratories for chemical research, dust research, and experimental research join with the engineering departments in translating safety requirements of industry into equipment meeting the highest demands of efficiency, comfort, and practicality.

An important adjunct of MSA Research is its nation-wide network of field representatives, attached to MSA district offices in approximately 76 principal cities. In intimate contact with industrial health and safety problems, these trained men work in effective liaison with industry and M-S-A. A close contact is maintained with the many official bureaus and agencies devoted to furthering the cause of industrial safety.

The broad institutional work of MSA—exampled, in small part, by this particular publication—is undertaken with the fullest sense of responsibility.

Published By

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Pittsburgh 8, Pennsylvania

Dusts and Fumes in Industry

(Sources, Effects and Control)

Introductory Statement

In dealing with occupational diseases and their effects, the attending physician should keep in mind that the etiology of these conditions is present in the industrial environment — if the conditions are to be considered truly industrial in origin.

It is likewise important to understand that rational diagnosis and treatment rests upon a fairly accurate knowledge of the etiology of the disturbances; this is especially true of occupational diseases and allied disorders.

Moreover, employers, industrial hygiene and medical departments are greatly interested in the prevention of various maladies which ordinarily affect employees; this clinical and economic interest on the part of industrial management further emphasizes the need for understanding the etiological factors in the working environment, so that they may be properly controlled.

This booklet on industrial dusts and fumes is the second of a series of which the purpose is to show the most important effects of occupations, show how these occur, give measures for treatment, diagnosis, and prevention, meanwhile considering the source or cause of such conditions. It is hoped that these simple and brief expositions will be of considerable help to industrial physicians, and to private practitioners who have among their patients employees of industrial plants subject to such exposure.

Definitions

Dusts may be defined as solid particles of matter which are ordinarily produced by grinding, crushing, drilling or blasting

processes, the composition of the particles being the same as that of the parent materials. These materials may be rock, metals, ores, or such products, for example, as coal, grains, or wood. The size of the particles varies from what is called ultra-microscopic to those which are visible. Authorities now agree that the hygienic significant particle size of dust ranges from a fraction of a micron to 10 microns. (A micron is 1/25,000 of an inch). The most frequently encountered dusts in industrial processes are mainly those from inorganic minerals, and a few organic dusts, such as wood and flour.

Fumes may be defined as solid particles which are formed by processes similar to combustion, condensation and sublimation. The most common types of fumes encountered in industry are usually caused by the oxidation of a metal, of which typical examples are the oxides of lead or zinc. Particle size is usually smaller than dust and ranges below one micron. In contrast to dusts, fumes tend to flocculate very easily. Many people erroneously speak of fumes when they mean vapors of liquid materials, such as acid solutions, organic solvents, and the like.

Incidence and Etiology

Dusts are the most common type of air contaminants encountered in industrial processes, and can occur whenever various materials, as mentioned before, are subjected to processes which break them up into fine particles; or where powdered or pulverized materials are handled or transported. Inorganic dusts are the prevailing type so far as frequency is concerned, but organic dusts are also found in different industrial operations.

Fumes are usually found wherever there are molten metals such as lead, zinc, and calmium being processed or used in industrial operations. Welding is an operation which has greatly increased in frequency of use and it may give rise to fumes of the metals which are welded or to fumes and gases from the variegated coatings of welding rods.

It is to be borne in mind that metals may produce both dusts and fumes, according to the nature of the operation and the state of the materials when processed. For example, among the heavy metals such as lead, dust may be of equal or greater etiologic importance with fumes — other mineral dusts and metallic dusts also are considered of etiological importance, as the following outline will show:

I. *Organic dusts:*
   1. Non-living organic dusts:
      (a) Toxic and/or irritant dusts; "(Various organic compounds such as trinitrotoluene, mercury fulminate, tetryl)."
      (b) allergy-producing dusts (woods, pollens, and the like).
   2. Living organic dusts:
      (a) bacteria
      (b) fungi

II. *Inorganic dusts:* (mineral dusts)
      (a) toxic and/or irritant dusts; (mainly the dusts from heavy metals and their salts, such as arsenic, lead, manganese.)
      (b) fibrosis-producing dusts, (examples are free silica and asbestos).
      (c) non-fibrosis-producing dusts; (the so-called inert dusts, such as alundum, corundum, emery, limestone, magnesite, marble, plaster-of-paris, and polisher's rouge).*

The most important of the non-metallic, inorganic mineral dusts is free silica, which produces silicosis, if breathed in sufficient quantities over a long period of time. Free silica dust and

* This classification is in accordance with the present state of knowledge. Future studies and experience will probably show some of the so-called inert or nuisance dusts to be harmful.

asbestos dust are the two kinds of mineral dusts which produce disease entities corresponding to the names of the dusts — silicosis and asbestosis. Mixed dusts of various kinds, such as various ores which contain different amounts of free silica, can produce different types of reactions, according to the kind of minerals which are in the ores. Where, for example, there are mixtures of iron, and various calcium-containing compounds such as gypsum, with free silica, a protective influence is exerted against the action of free silica on the lungs. Other modifications of free silica action occur according to the types of minerals present some of which tend to inhibit or restrain the action of free silica.

Another important conception with reference to the etiology of dust and fume diseases which may result from exposure to these classes of materials, is the use of the terms *exposure* and *hazard:* they are not synonymous — exposure merely means contact with some substance which is a *potential health hazard;* whether it is an *actual health hazard* can only be determined by an industrial hygiene engineering survey, in which the amount and kind of exposure is rated, according to accepted standards of concentration, size, and quality of dusts and fumes. Many persons erroneously therefore state that there is a lead hazard, for example, when it is only known that contact is being made with lead; and when there are no data to support the contention that there is an actual hazard present in the working environment.

Methods for the determination of dusts and fumes in the air, also with reference to the types of devices used for such determinations, will be mentioned later. The importance of air contamination will be realized when it is stated that the pathologic effects produced by dusts and fumes are brought about almost entirely by the inhalation of these compounds and materials. There is only one outstanding example, for instance, of the possibility of absorption of lead through the skin, namely tetraethyl lead, and this occurs in the form of a fluid. No dusts, so far as is known, produce any systemic pathological condi-

tions from skin absorption, and it is doubtful that any kind of true fume is absorbed by the skin.

From the etiologic point of view, particularly with reference to pathologic effects, the following outline shows how different types of substances (among the dusts and fumes) act on various systems in the body to produce pathological effects:

1. *General systemic poisoning:*
   Examples: Arsenic, lead, manganese, mercury.
2. *Substances acting on the circulatory system:*
   Examples: Arsenic, lead, mercury, vanadium.
3. *Substances acting on the respiratory system:*
   Examples: Asbestos, free silica.
4. *Substances acting on the gastro-intestinal system:*
   Examples: Antimony, cadmium, lead, mercury, zinc.
5. *Substances acting on the skeleton and joints:*
   Examples: Lead, mercury, mesothorium, phosphorus, radium.
6. *Substances acting on organs of special sense:*
   Examples: Arsenic, lead, manganese, mercury.
7. *Substances acting as fetal poisons:*
   Examples: Lead, mercury.
8. *Substances acting on the genito-urinary system:*
   Examples: Arsenic, lead, mercury, vanadium.
9. *Substances acting on the brain and nervous system:*
   Examples: Arsenic, lead, manganese.

General Diagnostic Methods

The accurate diagnosis of pathological conditions arising from the inhalation of industrial dusts and fumes has tremendous economic significance and also it is of medicolegal importance. Moreover, it is impossible to understand how proper and effica-

ceous treatment may be rendered unless a scientific diagnosis is made. If a claim results from the pathological condition alleged, it is evident that the disease sustained must be directly related to the employment and further, must produce disability to entitle the individual concerned to compensation.

Diagnostic methods with reference to dust and fume diseases need not differ widely, if at all, from those used as routine procedures in general scientific medicine, although there are specific methods as in any other application of general medical diagnostic principles to any special group of persons. New discoveries occur relative to diagnostic methods in occupational disease work and for the most part, reliance must be placed on information which is scattered throughout the current literature in addition to available textbook material which is better now than it was even five years ago.

Emphasis has already been placed upon the vital part played by etiology; it is wise to repeat that etiology forms the bulwark of the diagnostic and therapeutic background.

Among the methods which are used to make a fairly accurate diagnosis with reference to occupational diseases, is the taking of a thorough industrial history; consideration of symptomatology, the physical findings, the results of air examination; and finally, last but not least, a differential diagnosis, ruling out diseases of ordinary life which have no relation to occupation.

The industrial history may be of very great assistance in arriving at an opinion. Most industrial physicians of considerable experience believe that it is desirable to include a complete resume of the kinds of occupations which have been experienced by the individual being examined, daring from the first job to the last one. It is of course clear that the details of actual occupational exposure cannot be evaluated by this method, but important information bearing on the diagnosis may thus be obtained. It is the present practice, therefore, to list the occupations with the dates of occurrence, in the industrial history. It is

furthermore advisable to get more detailed information relative to the performance of the occupation, if that can be obtained. This will at least give information relative to the kind of exposure in industry, although it will give no idea of the severity of exposure.

The symptomatology in many kinds of occupational disease is likely to be confusing rather than useful, because in many instances it so closely simulates that of non-industrial disease that it is quite commonly not characteristic. Especial attention should be given, however, to the recording of the symptomatology and critical attempts made of its proper evaluation. Here again, distinction must be made between symptoms which may be due to non-occupational disease and those symptoms which are specific for occupational diseases — this is where the difficulty arises in adequately assessing symptomatology.

The physical findings, on the whole, are more reliable than the symptomatology, although in certain instances they too are not characteristic and are, therefore, liable to be somewhat puzzling. Where only one portion of the body is affected, as in silicosis, for example, it might be assumed that the physical findings would be more characteristic, but the opposite of this is usually true. In other instances, for example lead poisoning, where different organs and systems are pathologically affected, the picture is liable to be one which demands expert evaluation to differentiate from non-occupational conditions.

Clinical laboratory data, including X-ray findings, are frequently found to be decisive, although here again intelligent appraisal means proper interpretation of the data. When they are decisive, such tests are by the very specificity of their nature, of great value as diagnostic aids, although they should be definitely correlated with other findings.

Differential diagnosis presents perhaps the most difficult problem of all in the realm of diagnosis of occupational diseases. There is good reason to believe that differential diagnosis will

continue to be one of the most difficult phases of the medical ramifications of the medicolegal problems of occupational diseases. The reason which has already been indicated is that the symptomatology and frequently the physical findings imitate so closely the symptomatology and physical findings of non-industrial diseases, that one must make use of specific procedures which are designed especially as diagnostic guideposts. Frequently such specific procedures are not used to any great extent, except by those who are highly specialized in the field and whose work takes them into a review of the current literature containing these developments. For example, one may believe that an industrial employee has lead poisoning, largely because of second-hand knowledge that lead was used in his occupation, more easily than one could demonstrate adequate etiology and the existence of lead poisoning by scientific methods.

Because of all of these obvious and other difficulties, it seems wise to adapt specifically certain examination procedures to industrial problems arising out of occupational or suspected occupational diseases. Most authorities now agree that the examining physician can be of greater service and give more efficient advice regarding either acceptability for employment, continuance in it, opinion on the diagnosis of systemic pathologic conditions and the estimation of disability, if he is conversant with the following points:

1. The nature and extent of the industrial exposure into which the employee will go or where he has been. This can be determined only by scientific methods which will be referred to later.

2. The effect of this specific exposure upon normal and abnormal physical states of employees, as interpreted by the physical status of each individual on examination. (This refers specifically to etiology in the industrial environment and the effects which such etiology may or could produce.)

3. The nature of the protective devices supplied by the employer, if a hazardous exposure cannot be otherwise controlled, and as to whether these devices are functioning efficiently. (Reference will be made later to these devices, especially designed for protection against dust and fume exposures.)

It is realized that these procedures are somewhat removed from the usual program of physical examinations, particularly with reference to the work of part-time industrial physicians, but it is also expected that the physician will be called upon increasingly to know and appreciate such information so that he may function better as a diagnostic medium.

Because the most frequent and most significant exposures in industrial processes are free silica dust and lead and its compounds, it seems advisable to include in the next section some clinical examples, namely silicosis and lead poisoning; there will also be included a brief discussion of metal fume fever.

Clinical Examples

Simply stated, silicosis is a disease of the lungs, caused by the continuous inhalation over a long period of time of very finely divided, microscopic, particles of free silica dust. There have been various standards set up by different agencies, based upon studies of silicotic workmen and their exposure to free silica dust. The National Silicosis Conference Report on Medical Control of Silicosis says: "There is evidence that for prolonged exposure a concentration of more than five million particles per cubic foot of a highly siliceous dust is dangerous. Therefore, it is now considered good practice to hold concentrations of highly siliceous dust at 5 million particles per cubic foot, or less."

Because of the fact that industrial exposures, involving high free silica content in which the concentration of dust should be restricted to five million particles per cubic foot, are relatively infrequent in industry, and also because the opposite usually

occurs (an exposure to "mixed" dusts which contain appreciable amounts of free silica but also contain considerable amounts of other types of material) it is believed wise to refer to the formula* developed by the National Silicosis Conference with reference to a working criterion with respect to satisfactory or unsatisfactory conditions, when exposure to 'mixed' dusts occurs.

It is to be borne in mind, however, that any unprotected industrial operation, which involves the production of a lot of fine dust particles of material containing a relatively large percentage of free silica, offers a potential silicosis hazard.

The incidence of silicosis is not known with any degree of accuracy, although various estimates have been made. One of these made some time ago by the American Institute of Mining and Metallurgical Engineers indicated that in the United States the number of industrial workers exposed to the silica dust hazard is in excess of a half million. Figures quoted by the Committee Reports of the National Silicosis Conference disclosed estimates that approximately 2% of the working population of the United States or about one million workers are exposed in some way to the potential hazard of silicosis, and that perhaps half of this number, 500,000, or 1% of the total, are exposed to a serious hazard. It is furthermore believed that approximately 110,000, or 0.2 of 1% of the total, have silicosis to some degree, but likely only 4000 to 5000 (a small fraction of 1% of the total) suffer any degree of disability from the disease. (These figures are based upon prewar estimates.) It can readily be seen that silicosis, although not a rare disease, is not common and furthermore not as common as may ordinarily have been supposed by many persons.

Recent conceptions with regard to the contraction of silicosis have established the belief that approximately 25% of those exposed to a real silica hazard will eventually develop silicosis.

* FORMULA A: "Multiply the percentage of free silica by the total dust count. If the result is under 5 million, the condition may be considered permissible. If the result is over 5 million, the condition may be considered too high. For example, 10 per cent free silica with an average total dust concentration of 30 million particles per cubic foot would give 0.10 times 30 million, which equals 3 million (good practice) 50 per cent with an average total dust concentration of 30 million, would equal 0.3 times 50, or 15 million (unsatisfactory). This formula is not applicable to any dust containing less than 5 per cent free silica."

With respect to the occurrence of silicosis in the foundry industry, a prewar survey by the Division of Industrial Hygiene of the New York State Department of Labor, based on a study of 311 foundries, of which 80 plants included X-ray examinations involving 4,754 employees, provides some interesting figures: the X-ray summary showed that 3,259 or 80.2% were negative; 185 or 4.5% showed fibrosis; 110 or 2.7% showed silicosis with and without infection; 485 or 11.9% showed healed primary and re-infection types of tuberculosis; 27 or 0.07% showed clinically significant tuberculosis. It was concluded, as stated in the report, that it would appear that the silicosis hazard in the foundry industry in New York State, while existent, is of mild degree of severity and may be expected to yield satisfactorily to appropriate measures of control. The report further indicated that tuberculosis as manifested by X-ray films of the chest was found to be more prevalent among silicotics than among non-silicotics in foundrymen.

The occupational history, as previously indicated, is of considerable importance. Forms for the recording of occupational records have been devised by various organizations and groups and by physicians and insurance companies. One of the most complete and longer forms has been devised and used by the Saranac Laboratory, Saranac Lake, N. Y., in connection with silicosis studies and symposia on silicosis which have been held by this group. It is suggested that the physician, if interested, should contact the industrial hygiene bureau of his state health department through which detailed forms may be secured or information relative to them.

The symptoms and physical signs are not characteristic; they may be confused even by skilled diagnosticians in other diseases, with the symptoms and signs which commonly occur in both minor and the more serious types of the well-known respiratory infections. Characteristically, shortness of breath on exertion and decreased chest expansion frequently occur in advanced cases of silicosis, but these findings must also be differentiated as

to their casual relationships and their possible association with other diseases.

The clinical confirmation of the diagnosis of silicosis rests mainly on the characteristic X-ray appearance of the chest. This shows the typical lesions of silicosis, hyaline fibrous nodules which are scattered as opaque bodies throughout both lung fields, the density of these nodules being somewhat less than calcified bodies, but discrete in outline, all about the same size in the early stage (so-called millet-seed size) and evenly distributed throughout both lung fields. These nodules appear as the first indication of silicosis, later become somewhat confluent and in the latter stages, there are large masses of opaque material found near the periphery of the lung fields. In the later stages of silicosis, the disease is frequently complicated by infection, usually tuberculous in nature. As an aid to interpretation of the X-ray films of the chest where silicosis may be suspected, it is suggested that the physician refer to a tabulation of the X-ray changes in silicosis, with the underlying histological conditions which cause them, published in the transcript of the Second Silicosis Symposium, Saranac Lake, N. Y., 1935.

Diagnosis rests upon proof of occupational dust exposure sufficient to produce the disease, and the presence of characteristic nodular lesions in the X-ray film of the chest. The mere mention of occupation is not sufficient for diagnostic purposes — adequate knowledge concerning the quality and quantity of dust exposure, as well as the length of the employment period, is necessary to establish a distinct causal relationship.

Although considerable diagnostic reliance is usually and properly placed upon the history of adequate occupational dust exposure and the characteristic chest X-ray appearance, a thorough physical examination with appropriate clinical laboratory examinations should be done, especially with reference to the ruling out of other respiratory diseases. Diagnosis becomes essentially a differential diagnosis, as before explained, one which is likely to be difficult, necessitating special experience

and knowledge. Some of the conditions requiring differential consideration here are miliary tuberculosis, multiple calcifications of healed tuberculosis, cancer of the lungs, and fungous growths of the lungs.

Within recent years, superimposed infection has come to be the accepted cause for disability. The outlook in simple silicosis, therefore, (that is, without infection) is more favorable than was formerly believed. It is obvious that the chief problem for the silicotic is to avoid superimposed infection, which is usually tuberculous.

There is no one specific, efficacious treatment for silicosis, although in recent years, the inhalation of metallic aluminum powder has shown some promise. In cases where tuberculosis complicates the picture, it is obviously to be treated by the best known methods for that disease. There is some recent evidence that surgical therapy as used in ordinary forms of tuberculosis is also a help in the type of tuberculosis which follows silicosis. The evidence for this is not conclusive, but there are good results on record.

In general, recommendations for the treatment of cases of silicosis vary with experience and with the findings of each case. It is believed, for example, that no treatment is necessary in the case of an individual without symptoms, whose X-ray film of the chest (taken perhaps in a routine industrial survey) shows only widespread discrete nodulation without localized shadows indicating infection. However, the employee should have serial X-ray examinations with physical examinations of the chest at least once a year and should be removed from further silica exposure to some other job, since the disease is very slowly progressive and there is at this stage no disability which precludes continuation in employment. A person with localized conglomerate lesions may have symptoms and usually cannot do as heavy work as formerly; therefore, a lighter job should be found. In the presence of active infection where there are symptoms or incapacitation, the individual cannot obviously continue in his

former employment. If the case is one of open tuberculosis with positive sputum, it is evident that such an employee is a source of danger to his fellow workmen and should be placed in a sanitarium or hospital. In cases of inactive infection with no incapacitation, lighter work should be provided, without further dust exposure.

Dusts containing silica in combined form do not cause silicosis; however, one such dust, asbestos, a magnesium silicate, will produce a characteristic fibrosis which is called asbestosis. This disease is quite different in its pathology from silicosis, and has a characteristic 'ground-glass' appearance on the X-ray films of the chest. Animal experimentation indicates that the action of asbestos is probably mechanical, since it cannot be produced in other than the pulmonary tissue. Asbestosis may cause disability and a few fatal cases have been recorded, but in those cases showing marked disability, other organic disease is usually also present. The symptomatology and physical findings include cough, dyspnea, pallor, clubbing of fingers, and diminished chest expansion, with a characteristic X-ray appearance as previously mentioned.

Lead Poisoning

Lead poisoning offers a good example of an industrial poisoning which is contracted by the inhalation either of a toxic dust or fume, and has been stated to be one of the most frequent and important types of metallic poisoning in industry, on account of the widespread use of lead and its compounds.

Although there are no reliable statistics available with respect to the national incidence of lead poisoning, it is believed that lead poisoning is very prevalent and that most of it is mild. The incidence of severe types of cases, particularly lead encephalopathy and the more severe types of nervous system involvement are not seen with as great frequency as in former years. Many physicians who are used to handling industrial cases now rarely see a case of wrist drop due to lead poisoning, whereas 20 to 25 years ago it was a rather common phenomenon, in cases of lead intoxication.

The industrial history is of great importance, as previously indicated, and a thorough consideration of the nature and extent of the exposure to lead and its compounds is an essential point in the etiology. It is now generally accepted that the standard of the U. S. Public Health Service and the American Standards Association of 1.5 milligrams of lead per ten cubic meters of air, is the maximum allowable concentration. Because it is now conceded that practically all industrial lead poisoning is contracted by the breathing of lead dust and fumes, it becomes of considerable importance in working out the etiology to ascertain the amount and quality of lead dust and fumes in the air, during the occupation of the involved individual. This point will be discussed later with reference to prevention.

Formerly, the nature and variety of non-industrial sources of lead intake made it particularly difficult to establish industrial etiology on a sound and competent basis. More recently, however, non-industrial lead intake has been measured by scientific methods and with the exception of an occasional difficulty with various types of homemade beverages stored in utensils glazed with a lead compound, the non-industrial lead intake is not believed to be a considerable factor in the production of lead poisoning in adults. There are, however, some exceptions and these should be taken into account, as indicated.

The diagnosis of industrial lead poisoning is not a simple matter on account of the fact that clinical symptoms and signs are protean and are liable therefore to simulate other diseases. Great increase in our knowledge, however, has occurred within the past ten to fifteen years, and it is now known that there is a close relationship between the action of lead and its compounds within the body and the calcium metabolism, and further, normal and abnormal excretion of lead has been worked out on a scientific basis. It is now an accepted fact that persons without industrial exposure normally excrete a certain amount of lead in the urine. It has been stated that definite clinical findings of lead poisoning are practically always associated with abnormal urinary lead

excretion — the reverse, however, is not true — that abnormal urinary excretion of lead in the absence of characteristic clinical findings indicates lead poisoning.

Other laboratory findings are important, particularly the examination of the blood, some investigators and clinicians using the increase in basophilic stippled red cells as an indication of positive findings and others using what is known as the basophilic aggregation test. Both are found to be successful, the former more adaptable in cases of frank poisoning, the latter more useful in cases of lead absorption and suspected incipient poisoning.

Although as indicated, the clinical symptoms and signs of lead poisoning are protean, the principal effects are expressed in the blood, the gastrointestinal system, and the nervous system, named in order of their usual chronological occurrence. In brief, the most consistent findings are changes in the color and form of the red cells of the blood, colic and constipation, and weakness and atrophy of the extensors and flexors of the muscles of the wrists, with partial paralysis in the more advanced types of cases.

The differential diagnosis, as may be obvious, has to do mainly with differentiating industrial lead poisoning from non-industrial diseases which may involve the blood, the gastro-intestinal system, and the central and peripheral nervous systems, producing changes which are similar to those observed in frank lead poisoning. Such non-industrial diseases show a considerable variety of pathological processes affecting the above-named systems.

The treatment of lead poisoning has undergone considerable change within the past ten to fifteen years. In general, the treatment consists mainly of appropriate therapy for the toxic episodes and the mobilization of stored lead in the long bones. It is usually agreed that it is wise to facilitate the storage of lead by an excess calcium intake rather than to set lead free in the blood stream; after the passing of acute toxic episodes, the elimination of lead may be assisted by means of low calcium intake, the administra-

tion of weak organic acids and ammonium salts, sodium bicarbonate, and potassium iodide. Care should be exercised in using such forms of treatment on patients who have nephritis.

Two schools of thought with widely divergent views exist with reference to deleading the body: there are those who feel that in the majority of instances deleading will accomplish good results, while there are others who adopt a conservative attitude and because of the possibility of precipitating acute symptoms, feel that it is safer to produce artificial deleading by slow methods, or to resort to the natural process of letting the body delead itself over a period of time. The choice in the matter depends upon the individual case and the previous results which have been achieved at the hands of the physician in charge of the case.

Storage of lead may be facilitated in acute cases by a high calcium diet or by the slow intravenous injection of 10 cc of 20% calcium gluconate; variations with relationship to this form of treatment have been devised by various physicians. In the deleading process, low calcium diet together with oral administration of ammonium chloride has been found to be effective, whereas in certain cases parathyroid extract and sodium bicarbonate also have been useful. Within recent years it has been established that large doses of viosterol have been instrumental in increasing the excretion of lead. Other investigators have found that an adequate phosphorus intake is essential for the depositing of lead in the bones and for the removal from the bones of the excess cations, as the insoluble phosphate salts in the feces; it has therefore been recommended that deleading may be accomplished by giving a diet low in calcium and relatively high in phosphorus, using viosterol 20 minims daily.

There are also variations of treatment with reference to the amount of intoxication, and where there has been high, medium or low lead intake in industrial occupations. In all these types of treatment, deleading should be done in the hospital where the patient may be closely supervised. Some investigators advise

against deleading more often than once in four weeks and the course should not be prolonged for more than three or four days; some physicians believe that it may take from six to fifteen courses of deleading to free the individual of his most loosely-combined lead, which of course is the most dangerous type of lead. It is again warned that deleading should not be undertaken in the presence of acute symptoms; it is further agreed by all investigators that calcium therapy is considered to be the most specific at the present time.

The outlook in cases of lead poisoning is much better now than formerly because of the occurrence of moderate types of effects, the severer forms being a great deal less frequent than in former years.

Metal Fume Fever

This name is applied to the condition which results from breathing fairly heavy concentrations of freshly formed metallic oxide fumes, principally zinc and one of its alloys, brass; although the condition may result from the inhalation of other metallic oxide fumes. Metal fume fever is not a systemic poisoning, but it should be borne in mind that some of the metallic fumes that can cause it, such as lead, cadmium, mercury, and manganese, may also cause systemic poisoning.

The symptoms come on some hours after exposure, usually after leaving work, a dryness of the throat being first noticed, which may be followed by a dry cough and a feeling of heaviness in the chest. Rise of temperature, accompanied by sweating, and leucocytosis and elevation of the pulse rate and the blood pressure usually follow, sometimes with chills. The symptoms ordinarily pass off in 12 to 24 hours and the usual experience is that the employee returns to work the following day. A natural immunity is conferred in certain types of workers, which seems to be lost rapidly after removal from continuous exposure.

Diagnosis is made usually on a clear history of industrial exposure to metallic oxide fumes. It must be remembered that

the effects and symptoms of metal fume fever are very similar to those of influenza and that the malady is more often noticed in the fall and winter, when influenza usually occurs. The industrial history is, therefore, important in diagnosis, particularly with reference to a causative exposure to metallic oxide fumes.

The outlook is good for complete recovery in a short time and usually there is no time lost from work, although there may be temporary incapacity in terms of hours.

The treatment is symptomatic and especially directed toward comfort because the patient usually recovers within a relatively short time in all events. Rest in bed with adequate warmth and protection from drafts are recommended. However, all suspected cases of metal fume fever should be attended by a physician because the symptoms may closely simulate influenza which makes the differential diagnosis important.

Other Dusts and Fumes

It must not be concluded that although clinical examples were given of silicosis, lead poisoning, and metal fume fever, there are not other examples which are of importance. These subjects were selected because of their frequency of occurrence and because of the fact that they are commonly encountered as examples of dust alone, dust and fumes, and fumes.

Among the other metals which may be mentioned are arsenic, antimony, cadmium, manganese, mercury, radium, selenium, silver, tellurium, thallium, and vanadium. Not all of these are equally important with reference to toxic effects.

Probably the most important of these from a toxic point of view are arsenic, mercury, manganese, and cadmium. Arsenic produces chronic poisoning chiefly from breathing over a long period of thin air containing dusts or sprays of arsenic compounds. The compounds of arsenic such as lead arsenate, paris-green, arsenic oxide and arsenic sulphide are among the more dangerous forms.

Where dusts of mercury are present, they will be in the form of either mercurous or mercuric compounds, where metallic mercury is used in the manufacture of such items as thermometers, mercury switches, mercury vapor lamps, mercury boilers, etc., the hazard is from the vapor since mercury has a considerable vapor pressure even at normal temperatures. Mercurialism is characterized by three features: inflammation of the mouth, muscular tremors, and psychic irritability — sometimes all of these are encountered and sometimes only two or only one. In the more chronic or slow form of poisoning, a characteristic tremor is the commonest symptom, coming on late, and has been noted to occur particularly among mercury miners.

Manganese poisoning is an old industrial poisoning, having been described more than 100 years ago, but has receded in incidence in American industry. Characteristically it produces a peculiar slapping gait with phenomena of retropulsion and propulsion; masklike faces; monotonous voice with economical speech; muscular twitching varying from a fine tremor to gross rhythmical movements of the extremities; languor and sleepiness; cramps in the calves of the legs with stiffness; slight increase in the tendon reflexes without any disturbance of the deep or superficial sensation. Studies within the past five years indicate that dust is the most important type of exposure.

Cadmium is encountered in the smelting of ores, the handling of socalled 'blue powder' compounds, the spraying of pigments, the welding of alloys, and the melting of cadmium metal itself, as well as certain non-industrial exposures where utensils have been plated with this metal. In recent years there have been several outbreaks of cadmium poisoning among non-industrial users of such utensils. The use of the metal has increased tremendously and therefore, there is more industrial exposure. Two types of effects have been observed, those with reference to the lungs and also those with reference to the gastro-intestinal tract, the former being more important and more frequent. Pulmonary involvement is characterized by a feeling of constriction in the

chest, edema and congestion of the lungs, sometimes with hemorrhage, proliferative interstitial pneumonitis; while the latter is characterized by nausea and vomiting and a general irritative reaction of the gastro-intestinal tract, with diarrhea, hiccough, and oliguria.

If further details are desired on the clinical aspects of the metals mentioned, standard works on industrial hygiene and occupational diseases should be consulted.

Prevention

The general principles of prevention involve first the identification of industrial exposures and the evaluation of such exposures by proper scientific methods to determine whether or not they represent actual health hazards. This is done by sampling the air and determining the amount of air contaminants and the quality of them in each type of occupation or problem involved, and comparing the results with the accepted standards of maximum allowable concentration that have been adopted by official bodies. Second, after it has been determined that there is an actual health hazard represented by the occupation, control measures, both engineering and medical, are instituted to control the hazard, providing it cannot be entirely eliminated.

In discussing the methods of engineering control, we are concerned with three basic principles:

1. Investigation of occupational conditions.
2. The measurement of the occupational exposure and the rating of the hazard, if one exists.
3. Putting into effect the specific measures of protection which have been indicated by information secured under (1) and (2).

Among the methods of approach for engineering control measures may be mentioned:

1. Physical and hygienic survey of plant conditions.

2. Occupational history and analysis.
3. Adequate information concerning materials and processes used.
4. Atmospheric and gross sampling of materials, combined with quantitative physical and chemical determinations.
5. The rating of the hazard by comparing the findings in the given instance with accepted standards.
6. An appraisal of the efficiency of protective apparatus and devices.
7. An adequate and proper interpretation of the information so that it can be applied practically to the industrial problem involved.

When the foregoing methods of approach to engineering control have been carried out and information secured from them, the information can indicate the necessity for using specific methods of protection which have been found useful in different combinations:

1. The use of mechanical devices, such as exhaust ventilating systems, respirators, masks, and the like.
2. The changing of processes (segregation, enclosure, wetting, etc.).
3. Substitution of nontoxic materials for those of proved toxicity.
4. Educational programs for management, workers, engineers, and physicians.
5. Continuous maintenance and checking of the efficiency of protective equipment.

Atmospheric Sampling

The best method for collecting a sample of a dust or fume from the air will depend somewhat upon the type of contaminants to be sampled. The degree of hazard of fibrosis-producing dusts is measured in terms of particles per unit volume of air (example:

10 million particles of silica dust per cubic foot of air) while toxic dusts and fumes which may cause systemic poisoning are usually measured in terms of weight per unit volume of air. (Example: lead dust 1.5 milligrams per 10 cubic meters of air.)

Since results may vary with the type of air sampling device used, it is important to select a type accepted through general usage in the practice of industrial hygiene.

The larger Impinger which samples one cubic foot of air per minute by impinging the air containing the dust particles from a nozzle submerged in water or alcohol against a plate or bottom of the flask was used in all of the original studies in the United States on fibrosis-producing dusts. The large Impinger, however, has the disadvantage of being bulky and requiring electric current or compressed air for operation of the motor-driven air pump, which produces the suction stream for air sampling.

A portable Midget Impinger has been developed which has been proven to give results as reliable as the large Impinger. This apparatus samples at the rate of 1/10th of a cubic foot per minute and is operated by a hand cranked, positive action, four cylinder pump, in conjunction with a vacuum regulator which maintains an unfluctuating suction regardless of minor variations in rate or smoothness of cranking.

After the sample has been collected in the Impinger flask, a portion is placed in a special dust counting cell and counted under a microscope with an eyepiece having a special ruling similar to a blood count ruling.

Further analyses are necessary with reference to the amount of free silica in the dust and this is accomplished by the use of specific chemical, petrographic and X-ray diffraction methods.

The Impinger method of sampling is not entirely suitable for collecting fumes or dusts of toxic materials such as lead, cadmium, and others because the efficiency of the Impinger is not adequate when sampling the very fine fume particles and since the amount

or concentration of such materials in air which may be injurious is much smaller than the safe amounts of fibrosis-producing dusts, a correspondingly large air sample should be taken for reliable results.

The Electrostatic Precipitator is widely used in the collection of toxic dusts and fumes, particularly of metals and their compounds. A Portable Electrostatic Precipitator has been developed which operates from 110 volts, 60 cycle current, and by high voltage electrical precipitation collects the sample of the particulate matter dry on the inside of aluminum or glass cylinders. This device samples at the rate of 2 cubic feet per minute. After the sample is collected, a quantitative chemical analysis is made in the laboratory. This type of apparatus permits determinations of very low concentrations of many different kinds of atmospheric particulate matter.

Where equipment or personnel is not available for atmospheric sampling, state industrial hygiene bureaus or private testing laboratories may be engaged. Many insurance companies also have facilities for such work. The Industrial Hygiene Foundation in Pittsburgh, Pa., an association of industries for the advancement of healthful working conditions, also have an excellent staff of technicians for industrial hygiene surveys or consultations in this field.

Respiratory Protective Equipment

It frequently happens that because of the nature of the operation or process in an industry, it is not possible to thoroughly control a hazardous exposure resulting from the evolution of dusts and fumes. In such instances, it is considered wise to provide employees with personal respiratory protective devices. These vary with the kind of exposure and with the amount of materials in the air.

For ordinary conditions in which only dusts and fumes are present, simple filter type respirators are available which will

protect the worker against the hazards of breathing these dusts and fumes.

Such respirators normally consist of a facepiece covering the nose and mouth, made of some pliable material such as rubber which will conform to the face and provide a comfortable and dust-tight fit, and to which is attached a filter made of treated paper or felt through which the air is inhaled. If the filtering material is of the correct type and density, it will effectively filter particles down to less than a micron in size from the air.

Comfort is a most important consideration in a device of this type. Since a high breathing resistance will materially affect the comfort of the wearer, the filters should be of large area so that the inhalation resistance will be as low as possible. Valves should be provided which open on exhalation and further lessen the exhalation breathing resistance.

The particular type of filter employed will depend upon the kind of contaminant present in the air. Since the amounts of such toxic materials as lead which will affect the wearer are much smaller than the harmful amounts of fibrosis-producing dusts, a filter will have to be more effective when used against toxic dusts than against fibrosis-producing and nuisance dusts.

For special operations such as sandblasting, a mechanical filter type respirator is not practical because of the extremely high concentrations of dusts present. In such conditions, special air-supplied masks or respirators should be used in which air from a compressed air line is supplied through a hose to the facepiece for breathing. Such devices may also be employed if desired as protection in less severe and hazardous operations. The advantage of an air-supplied respirator is that there is no breathing resistance and the wearer is protected against any and all dusts and fumes in the surrounding atmosphere. However, the restriction by the air hose which must be attached to the wearer at all times between his facepiece and the air supply, prevents the use of this device in many operations where the worker must move freely

over a considerable area. Furthermore, caution must be taken to see that the air supplied to such devices is free from all injurious or objectionable odors and contaminants and that the humidity and temperature of the air is such as to provide reasonable comfort.

The United States Bureau of Mines (Department of the Interior) tests respiratory protective devices against various air contaminants under carefully controlled conditions. Devices meeting its tests receive that Bureau's official approval, and this is indicated by approval numbers on the device and a reproduction of the certificate on the package. Equipment approved by the U. S. Bureau of Mines for the particular contaminant or class of contaminants in the air should be used.

MEDICAL CONTROL

It is of course obvious that medical control measures are futile after a disease has been contracted because of the exposure to dusts or fumes except when the information is adapted to prevent additional cases. However, before the diseases are contracted, there are a number of things that can be done by physicians to prevent such occupational diseases, which may be summarized as follows:

1. The intelligent use of preemployment physical examinations, including chest X-ray films, and appropriate clinical laboratory tests as indicated.
2. Similar use of periodic physical examinations, chest X-ray films, and clinical laboratory tests on employees in occupations where typical hazards have been proved to exist, by industrial hygiene investigations.
3. Responsibility for seeing that hazardous exposures are properly taken care of, by various efficient methods of protection, including the supplying of personal respiratory protection, where industrial conditions make it impossible to control the hazard by other means.

4. Prompt transference to non-hazardous occupations of employees found to have symptoms of an occupational disease associated with their exposure.
5. Prompt removal from work of all persons suffering from active occupational diseases, when such persons are a menace to themselves, their fellow workmen or the employer.
6. Adequate provision for treatment, rehabilitation, and prompt return to work, of employees having disability resulting from occupational diseases.

Summary

In this brief discussion of dusts and fumes in industry, the sources, effects, and control have been considered under the following headings:

1. Definitions.
2. Incidence and etiology.
3. General diagnostic methods.
4. Clinical examples.
5. Prevention — general principles, including specific methods of air sampling and apparatus for this purpose; specific methods of protection, including mention of respiratory protective equipment available; and other general methods.

It should again be emphasized that industrial managers are interested in the prevention of trouble that may arise from exposure to dusts and fumes in industry, and that this principle is particularly important nowadays because of the need for every person in active production, and in addition, because so many physicians are being used in the armed services.

It is believed that if the suggestions made in the foregoing pages are followed, the problems arising from dusts and fumes in industry can be successfully managed.

APPROVED

M-S-A EQUIPMENT

FOR

Dust and Fume Protection

MSA Protection for men and women workers against harmful dusts is the result of many years of research into all phases of every industry's respiratory protective problems. In a well-equipped Dust Research Laboratory, which is a special division of our Research Department, continual study of dust hazards is maintained, and from this research group have come many of the most important improvements in dust protection—dating from the first U. S. Bureau of Mines-approved dust respirator, the famous M-S-A "Comfo." The M-S-A trademark on the products you recommend is a solid assurance of excellence in design, manufacture and performance.


PERMISSIBLE RESPIRATOR
FOR
DUSTS
APPROVAL NO. 2133
ISSUED TO THE
MINE SAFETY APPLIANCES CO.
Pittsburgh, Pennsylvania, U. S. A.
CAUTION

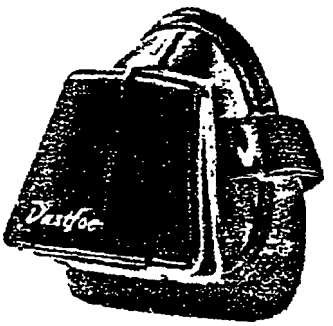
All applicable M-S-A personal protective equipment against harmful dusts or fumes is approved by the highest authority—the United States Bureau of Mines, official testing agency of the United States Government for safety equipment. The approval certificate of the Bureau signifies that each product has passed the most stringent impartial tests—fit for the most exacting service in industry.

We will be glad to send you, upon request, descriptive bulletins on any or all of the products mentioned in this booklet—without obligation.

M-S-A® Filter Respirators

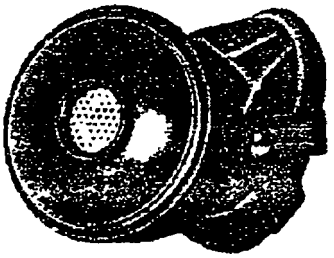
Designed for protection against harmful dusts and fumes by the use of interchangeable filters. Each of these Respirators answers all respiratory protective requirements against pneumoconiosis-producing, nuisance or toxic dusts.

M-S-A® Dustfoe 66



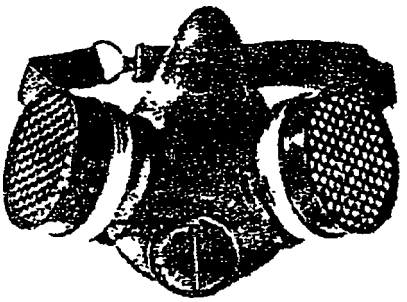
Bureau of Mines Approved. An improved compact and lightweight Respirator with larger exhalation valves reducing resistance to a minimum. These spring-retained valves eliminate disturbing variation in breathing resistance, and prevent accidental *removal* of valves. Facepiece cushion of neoprene sponge is contoured to provide automatic fit, great wearing comfort, and air-tight seal without adjustment. Electrostatically charged resin-treated filters supplement mechanical holding action of filter's fibers. Simple construction assures quick and easy filter changing and cleaning of Respirator. Weight, including headband: 3 ounces.

M-S-A Dustfoe *Ultra-Filter* Respirator



This compact and lightweight Respirator of aluminum is designed for unusually toxic dusts and extremely fine particles. Its filter holder allows for good field of vision and freedom of head movement. Facepiece forms to contour of wearer's face. Neoprene sponge cushion assures air-tight seal with minimum adjustment and maximum comfort. Neoprene stays soft and pliable: will not harden with age. M-S-A Type H Ultra-Filter protects against toxic aerosols, radioactive dusts and fine particle smoke; also effective against airborne bacteria and certain viruses. Construction is simple for easy filter changing and cleaning.

M-S-A Comfo Filter Respirator



Most versatile of the M-S-A Respirators. Facepiece is made of a special new soft compound for improved wearing comfort and greater durability. Provides dependable air-tight seal with wearer's face, and will not harden or crack with age. Positive-action exhalation valve offer little resistance to air. The Comfo features interchangeability of three filters: Type F (Bureau of Mines Approved) for dusts not significantly more toxic than lead; Type S (Bureau of Mines Approved) for dusts, mists and metal fumes; and Type H Ultra-Filters for toxic aerosols, *extremely* fine particles, radioactive dusts, bacteria and certain viruses.