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INDUSTRIAL TOXICOLOGY

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PREFACE

This book has been written in response to many requests for information concerning the toxicity of new, as well as old, substances of industrial importance. Its purpose is to serve as a bridge between the rapidly expanding field of toxicological investigation and the application of this knowledge of industrial poisons by the industrial hygienist.

Many of the substances described are not poisons in the usual sense. Indeed, some are not toxic at all. These represent materials newly used in industry, however, and concerning which some doubt has existed. The industrial hygienist is occasionally compelled to search through many periodicals and specialized books to acquaint himself with the current knowledge concerning these substances. All too frequently the toxic effects of a substance are presented in case reports, which are often confusing and detailed, and require careful study in order to define its toxicity. In the present manual this material has been surveyed and summarized with the object of relieving the industrial hygienist of much of this burden. Although this has required the examination of the available literature regarding each substance, only selected and pertinent bibliographical references are given, with such appraisal and interpretation as would serve to give the industrial hygienist an evaluation of toxicity without his having to digest a great mass of literature, or to seek out many obscure and difficultly obtainable publications. A short list of the more pertinent references is given at the end of each substance discussed. The references either relate directly to the subject matter, or are felt to be of general interest to the reader. No attempt whatsoever was made to append a complete bibliography—to do so would only expand the book without adding significantly to its value.

It will be apparent that whatever merit this book may possess is due to the vast fund of information which the many researchers in this field have contributed during the last two or three decades. The author's task has been to examine and compare these investigations and to assess them critically so that the gist of current thought is presented for the toxicological evaluation of a given substance.

The form in which the material is presented was arrived at after much consideration. It is largely based upon the needs of the industrial hygienist and is as brief as was felt consistent with his requirements. Some disparity of space will be found in that certain substances whose toxicity is well recognized are treated in more summary fashion, in comparison with those substances of obscure toxicity or substances which currently are of great importance, which are discussed in more detail.

The purpose in presenting the characteristics of the various substances

under consideration is related to more than mere identification. The physiological response to many substances depends to some extent upon their physical and chemical characteristics, such as volatility, solubility, solvent action, and stability. A knowledge of the outstanding characteristics of a substance is therefore often useful in defining a given occupational hazard. The chemical and physical constants are based upon the best information available for the pure substance and several may differ somewhat from those found in the usual tables. In all cases the most authentic values have been sought for these constants. They are of course particularly useful to the chemist in the examination of materials.

Under the section on industrial uses, an attempt has been made to gather from governmental and other sources such material as would indicate the extent of production and use. In many cases this will give a rough idea of the extent to which exposure may occur in industry.

Because of the diversity of the substances discussed, no attempt has been made to classify these substances in groups based upon either chemical resemblance or similarity of physiological response. Furthermore, it was felt that a simple alphabetical arrangement would facilitate reference. For further simplicity, the substances are broken down into two main groups—inorganic substances and the carbon compounds.

It has not been the author's intention to give detailed analytical instruction with reference to the various substances described in the following pages. Under the heading of analysis, reference is made to standard methods or to recently proposed methods. In a number of cases where no present method of analysis is available, the writer has suggested possible procedures.

The writer is most grateful for assistance from his colleagues in the Industrial Hygiene Division of the United States Public Health Service. However, he assumes full responsibility for the material as presented. Despite every good intention and every earnest and painstaking effort, imperfections and errors may occur; readers will confer a favor by reporting them so that they may be corrected in a future edition.

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INTRODUCTION

In an earlier period of our industrial development workmen were exposed to smoke, dust, fumes, and gases without regard to the possible injurious nature of many of these substances. Certain occupations, such as the mining of mercury, were formerly notorious in this respect. Furthermore, this condition was recognized in only very few instances and exposure to fumes or dusts of a deleterious type was generally recognized as a somewhat disagreeable and accepted condition of many occupations. The fact that some workers in mines, mills, or factories sickened, were forced to leave their occupation, and later died, did not always bring with it the realization that the nature of their work was a factor. In some occupations where industrial disease was prevalent there were often individuals who had been immersed in such an environment most of their working life and yet remained apparently strong and healthy. Such cases would be cited as indicating the general harmlessness of their occupation and those who sickened were regarded as weaklings. Many industrial diseases, such as phosphorus poisoning, were undetected for years, or, as in the case of manganese poisoning, were undetected for generations. The scrutiny of occupation as a factor in disease, although emphasized long ago by Ramazzini, is a comparatively modern development. Industrial physicians only a generation ago were largely concerned with the more external factors of employment—hernia, lumbago, cataracts in the case of glass workers, deafness in boilermakers—or in incidental disease, such as ankylostomiasis among miners or tuberculosis among cotton spinners. The latter were, of course, related to poor sanitation and poor nutrition, respectively, and were by no means peculiar to the occupation of the worker.

Knowledge, however, of the existence of occupational disease became more and more evident and, although locally confined at first, gradually spread. Thus, such terms as "grinder's rot", "phossy jaw", "painter's colic", "chrome itch", and "miner's phthisis" gradually appeared in industry and in the literature. Within the past generation industrial physicians have been alerted to the danger of much of the smoke, dust, or gases to which workers were exposed, scientists have actively investigated the effects of many of these aerial contaminants on animal life, and engineers have instituted control measures in industry to reduce the hazard of exposure.

Poisoning familiarly occurs as the result of ingestion of toxic substances and this doubtless influenced much of the earlier industrial hygiene thought. As a result, stringent sanitary measures were advocated and adopted in certain industrial processes in order to prevent possible entrance of indus-

trial poisons by mouth. These precautionary measures included such matters as a complete change of clothing on entering a shift, careful scrubbing of the hands and face before eating lunch, and a thorough washing and shower at the end of the work period. While excellent in themselves, these measures do not include the sanitation of breathing and instances of industrial poisoning have occurred where workers had observed strict cleanliness yet were exposed to dangerous concentrations of dust, fumes, or gases. Inhalation is now recognized as one of the most dangerous routes of entrance of industrial poisons.

GASES, FUMES, AND DUST

While the greater part of industrial smoke consists largely of carbon and is relatively harmless, it frequently contains gases which are dangerous if continuously breathed or if breathed in high concentration and, in addition, it may contain noxious fumes or toxic dusts from certain processes.

From the point of view of industrial hygiene a gas may be considered to be any aeriform or completely elastic fluid which does not become liquid or solid at ordinary temperatures. Fumes or vapors consist of material in the near-gaseous, or extremely fine particulate form which condense to liquids or solids at room temperature. Dusts, on the other hand, consist of larger particulate matter suspended in air. It must be realized that these definitions are not rigid and that they merely indicate somewhat roughly different stages of attenuation of matter. It is possible, for instance, to have mercury dust suspended in the air of a workroom, due to constant attrition of mercury spilled on the floor and carried into the air as extremely minute droplets following mechanical agitation. At the same time, mercury fume may be present as the result of heating this substance and finally the vapor tension of mercury is such that true, gaseous mercury may also be present to a slight extent. It is important for the industrial hygienist to keep these distinctions in mind. The detection and estimation of mercury gas in air by means of the photoelectric mercury detector depends upon the absorption of ultra violet light of a wave length of 2537 Å and the degree of absorption is a measure of the amount of *gaseous* mercury present. However, this instrument would not indicate the total mercury present in an atmosphere where particulate mercury or dust from any of its compounds was also present.

While dusts have been classified as particles or aggregates of particles of from 150 microns to one micron in diameter, fumes of from one micron to 0.2 micron in size, and smokes as particles less than 0.3 micron in diameter, size alone represents at best only a rough separation of these three classes. The mode of formation must also be considered. Thus, dusts ordinarily result from mechanical attrition and distribution, while fumes and smokes

are formed and carried into the air usually as the result of chemical reaction or the sudden dispersion of a chemically active substance by release of pressure or by explosion.

The disperse systems, or aerosols, in which the dispersion medium is a gas, differ from other disperse systems in the great disparity that exists between the density and structure of the disperse phase and the dispersion medium. The mere fact that two such disperse systems contain amicroscopic particles similar in magnitude does not necessarily mean that the properties of the two systems are identical, although they may have many points of similarity. In the case of dust, a great deal of work is required in order to reduce a solid to fine dust, i.e., to overcome the forces of cohesion that originally held the particles together in addition to the work required in distributing the particles throughout the dispersing medium. Even though a certain degree of uniformity finally is attained by settling or other means, dispersed dust of the finest order of magnitude is less uniform in structure than aerosols produced by the condensation of vapor. These factors have some weight in devising means of sampling and analysis of aerial contaminants.

The evaluation of aerial contaminants is very frequently a matter of importance to the industrial hygienist and a knowledge of the properties of such disperse systems as those indicated above, as well as the properties of gases and vapors in relatively low concentrations, is of particular value with reference to the analytical detection and determination of the constituent contaminants.

The composition of the aerial contaminant to which workmen in a given plant are exposed is of course of paramount importance to the industrial hygienist. This is usually known, or information may be obtainable from the management. In some cases, however, an unknown or unsuspected factor may be present and careful investigation may be necessary before the culprit is revealed. Cases have occurred, for example, where arsenic, cadmium, or selenium existing as unsuspected impurities in the material being fabricated have caused illness and death. An unrecognized by-product of manufacture may cause difficulty, or a change in the formula of basic material used in manufacture may bring about an unhygienic situation. The industrial hygienist is required not only to ferret out the occupational disease hazard but also—very necessarily—to know its characteristics, a proper method of sampling, and the most reliable method of analytical evaluation in terms of air content. His study includes the weighted or average exposure of employees at various stations and occupations. Furthermore, he is required to know something regarding the toxicity of the aerial contaminant in order to define the conditions under which employees are to be permitted to work in such an environment.

The toxic effects of many hazardous materials in industry are well known and it is comparatively easy to define safe working concentrations. There exist many substances in common use in industry, however, which are toxicologically not well defined. Unfortunately, toxicity cannot be evaluated with the ease with which a chemical constant, such as a boiling point, melting point, or index of refraction, may be determined. Even with arduous investigation extending over many months, the toxicologist can at best give only a very general answer regarding the poisonous nature of a given substance. It would be of inestimable benefit of course, if one could—knowing the composition and molecular structure of such a substance—predict its physiological properties.

CHEMICAL CONSTITUTION AND PHYSIOLOGICAL RESPONSE

The possibility of relating chemical constitution and physiological activity has long proved a fascinating field of speculation. The advantages of defining the toxicity of a substance from its constitution or structural formula are, as indicated above, obvious. Unfortunately, however, the matter is not simple. Certain relations exist, it is true, between structure and toxicity. For instance, ethyl and methyl alcohol, although differing in one important respect, are very similar in many of their other physiological properties, and propyl, butyl, and amyl alcohols might be assumed to act similarly. These latter alcohols do, in fact, resemble the lower members but with a progressive increase in toxicity. An analogous increasing toxicity might therefore be anticipated in the higher members of the alcohol series. This reasoning, however, is nullified by the changing physical properties of the higher members. In spite of a similar chemical structure, the higher alcohols become increasingly insoluble in body fluids and as a result there is an overall decreasing toxicity beyond a certain point. A similar increase in toxicity with an increasing number of carbon atoms is noted with sodium acetate, propionate, butyrate, and valerianate. Many other such relationships have been pointed out as more toxicological information has become available.

The replacement of a hydrogen atom with chlorine in the saturated hydrocarbons results in an immediate change in toxicity; the entrance of such a halogen group in the organic sulfur compounds greatly intensifies the toxicity of the resulting compound. When one chlorine group is introduced into ethyl sulfide, which is a weak poison, the resulting monochloroethyl sulfide, is found to be markedly toxic, while the introduction of a second chlorine atom results in dichlorodiethyl sulfide, or mustard gas, which is a very strong poison indeed. However, no such general rule can be applied in other cases. The successive replacement of hydrogens by chlorine in the methane molecule, which of itself is not toxic but merely

an asphyxiant, results in monochloromethane, CH_3Cl , dichloromethane, CH_2Cl_2 , trichloromethane, CHCl_3 , and tetrachloromethane, CCl_4 , respectively. These substances, however, do not follow a pattern of increasing toxicity. For instance, chloroform with its excellent narcotic properties, as well as the attendant possibilities of liver and heart damage, is less toxic in general than carbon tetrachloride on the one hand and much less toxic than methyl chloride on the other hand. Yet dichloromethane, which occupies an intermediate position, is far less toxic than any of the other members of this group. It does not follow therefore that there is any direct correlation between the number of chloro groups and the toxicity.

The relatively inert and inoffensive hydroxyl group when introduced into an organic molecule frequently results in an increase in toxicity. Thus, methanol, CH_3OH , has pronounced toxic properties compared with the parent substance and monohydroxybenzene, $\text{C}_6\text{H}_5\text{OH}$, or phenol, has marked poisonous properties over and above those of benzene. Increasing the number of hydroxyl groups may also increase the toxicity of the aromatics. For example, the introduction of a second hydroxyl group in the benzene ring yields resorcinol, $\text{C}_6\text{H}_4(\text{OH})_2$, which is more toxic than phenol, while the introduction of a third group yields phloroglycinol which is the most toxic of the three.

The entrance of an alkyl group into the molecule of a substance may also intensify its poisonous quality. Dimethyl resorcinol, $\text{C}_6\text{H}_4(\text{OCH}_3)_2$, is more toxic than resorcinol, $\text{C}_6\text{H}_4(\text{OH})_2$. On the other hand, an alkyl group may diminish the toxic effect in other substances. Dichloromethyl arsine, $\text{As}(\text{CH}_3)\text{Cl}_2$, is very toxic, while the introduction of a second methyl group, as in dimethyl chloroarsine, $\text{As}(\text{CH}_3)_2\text{Cl}$, yields a substance of weaker toxicity.

While the introduction of a chlorine group in aliphatic hydrocarbons increases the toxicity in general, this is not necessarily true in the case of the aromatic hydrocarbons. Thus, monochlorobenzene is less toxic than benzene itself and has been of no particular significance as an industrial poison.

It appears to be a general rule that *iso* compounds are somewhat less toxic than normal compounds. *iso*Propyl alcohol has a somewhat lower toxicity rating than normal propyl alcohol; *isobutyl* alcohol than normal butyl alcohol. A most interesting difference in toxicity has been found to exist in the benzene hexachlorides which have recently received attention as insecticides. In this case, the gamma derivative of 1,2,3,4,5,6 hexachlorocyclohexane, $\text{C}_6\text{H}_6\text{Cl}_6$, has been found to be especially lethal in action compared with the other four known isomers (1). Woodard and Hagan (2) found the gamma isomer to be as much as sixty times as toxic for certain warmblooded animals as other isomers of benzene hexachloride. In the

case of many other optically isomeric substances, great differences may also be found in toxicities. For instance, 1-hyoscyamine is twice as active physiologically as dl-hyoscyamine (atropine) and moreover the laevo compound is twelve to twenty times as active as the dextro compound (3). Usually, but not invariably, the laevo compounds are more active than dextro compounds. Thus, Cushny found 1-hyoscine to be sixteen to eighteen times as active as d-hyoscine and twice as active as dl-hyoscine. Similarly 1-adrenaline was twelve to fifteen times as active as d-adrenaline in its vasoconstrictor action and twice as active as dl-adrenaline. On the basis of rather indirect evidence, it is possible that the difference in physiological behavior of such stereo-isomers is due to the ability of one or the other to combine with some protein or other constituent of the cell. However, sufficient quantitative data are not available to define clearly the mechanism of physiological activity and stereochemical configuration. The problem of relating chemical constitution and physiological action is even more confusing when it is recalled that substances of diverse chemical nature may produce similar physiological effects. The aliphatic narcotics, for example, include a large variety of structural type, such as hydrocarbons, alcohols, ethers, amines, and sulfones. According to Ing (4), these substances appear to achieve their effect by modifying the physico-chemical conditions of the cells due to certain physical properties shared by all classes of these compounds and not by the presence of certain pharmacodynamic groups.

It will be apparent therefore that while the temptation to rationalize regarding the prediction of toxicities may be great, the evaluation of toxicities of new substances is fraught with considerable uncertainty. Since no great rational scheme is available to aid, except rather sketchily, in deciding upon the toxicity of a given substance, it is inevitably necessary to carry out experimental work with animals.

EXPERIMENTAL TOXICOLOGY

Experimental industrial toxicology does not differ widely from that of experimental pharmacology, since they both use the resources of chemistry, physics, physiology, and pathology to achieve their purpose. However, the pharmacologist is primarily interested in therapy rather than toxicity. Furthermore, he is interested in the effects of administration of a substance by mouth, or by intravenous or subcutaneous injection and rarely in the effects of inhalation, except in the case of inhalation anesthetics. The refinements of approach to certain physiological reactions adopted by the pharmacologist, however, often yield data of fundamental importance within a relatively short space of time. The toxicologist on the other hand may be compelled to follow an intricate procedure which is arduous and

time consuming in order to define a given physiological response. Moreover, toxicological studies frequently depend upon pathological changes following the administration of small amounts of such toxic materials as gases, fumes, or dusts and these changes usually occur very slowly.

Toxicological investigations are in general based upon animal experimentation for human experimentation is, of course, indefensible. It is true that animal experiments provide only indirect evidence of the probable action of toxic substances on man, yet they are none-the-less of the greatest value. They not only afford information regarding upper toxic limits, but by long-continued study, reveal changes in lower concentrations which are of the greatest importance. In experimental work of this character, a considerable amount of interpolation is necessary and evaluation of the experimental results requires both acumen and careful judgment. Moreover, there are many poisons which produce completely different effects in different species of animals. Mice and guinea pigs, for example, are more sensitive to poisoning by trichloroethylene (acetylene trichloride) than cats, while rabbits are less sensitive than cats. On the other hand, cats are more sensitive to lead poisoning than dogs and the latter more so than rats. Although such qualitative and quantitative differences may exist, nevertheless there can be no doubt that animal experiments are of value since they give an indication of the type of toxic action, as well as the relative toxicity of the substance under investigation.

The great advantage of animal experimentation is, first of all, the control of dosage and, secondly, the degree to which poisoning can be carried. This yields most useful information in tracing similar effects in humans accidentally poisoned and in warning against human exposure beyond a certain degree.

Although the effects of exposure by inhalation are of paramount importance, animal studies also usually include other forms of administration, such as ingestion, intravenous injection, intraperitoneal injection, and subcutaneous injection. The latter form of administration is perhaps the least used as absorption is frequently slow and has less significance than the other forms. Intravenous injection provokes the most immediate response and is often useful in studying the immediate effects of substances upon the hemopoietic system. Intraperitoneal injection is one means of following the slow absorption of relatively insoluble substances, but is of particular value in studying the physiological response of various substances. For instance, when pure silica suspensions are intraperitoneally injected, a reaction occurs which is typical and fairly constant (5). Sayers and Miller found three types of reaction occurred with various dusts—absorptive, inert, and proliferative. The absorptive reaction occurs when fine dusts, such as calcite, limestone, gypsum, and cement, are injected. After suffi-

cient time, this material disappears from the peritoneal cavity without the formation of any scar tissue. In the case of the inert reaction, the dust remains distributed about the peritoneum by the action of phagocytes, sometimes forming flat nodules, which do not tend to progress or form fibrous scar tissue. Soapstone, carborundum, and coal dusts exhibit this inert reaction. In the proliferative reaction, nodules form which continue to increase in size with the formation of fibrous or scar tissue. Quartz, chert, and flint dust produce this reaction. These three types of reaction correspond very closely with the results obtained by the inhalation technique. While this method perhaps requires further exploration, it is useful in the approximate classification of dusts.

The inhalation technique with reference to dusts, fumes, and gases is by far the most useful method used in experimental toxicology. In the case of gases, the various concentrations to which animals are exposed may be regulated with accuracy. Hence, the exposure may be clearly and accurately defined. With fumes and dusts, the concentration of fume or dust in the air may be determined by exact chemical analysis. Whether or not the animals always breathe in the amount in the atmosphere may not be so accurately defined in many cases as small animals, such as guinea pigs, tend to huddle together and may be able to filter out some of the dust or fume to which they are exposed.

For the purpose of exposure, gas-tight or dust-tight exposure chambers of large size are usually used and animal cages may be placed directly in the exposure chamber. The gas may be introduced in known amounts and rapidly distributed throughout the exposure chamber by means of small fans. Substances which are sufficiently volatile, such as solvents, may be introduced in known amount into a constant current of air and the breathing concentration may be accurately calculated. In addition, of course, samples of the exposure atmosphere may be taken in most cases and analyzed chemically.

Fumes, such as metal fumes, may be generated by arcing between electrodes of the metal carrying 110 a.c. current and an appropriate resistance. In order to prevent the formation of metallic oxide fume, it is usually necessary to generate this fume in an atmosphere of inert gas, such as nitrogen or helium, and "bleed" it into an incoming air current. Oxide fumes are readily formed by arcing in air or in an atmosphere of oxygen. Fumes of many organic substances may be formed by heating the material to the volatilization temperature in the incoming air stream.

Dusts must be suspended in air in very finely divided form in order that they may remain in suspension as long as possible and so that the material is of sufficient degree of fineness to be carried deeply into the animals' lungs. An elutriating device is useful for this purpose (6), since it permits

constant flow of very finely divided dust of more or less uniform size. In the case of dust exposures, it is difficult to control the amount of dust in the atmosphere and hence it is necessary to draw small samples from time to time for analysis. Even though the dust concentration is accurately known, the amount carried into the animals' lungs is questionable as a variable amount is removed by the filtering action of the nose, part of the dust remaining in suspension is removed before the air enters deeply into the lungs. The ciliated epithelium found throughout the extent of the air-passages and their prolongations constantly sweeps out air-borne particles and only the very finest material penetrates to the lung alveoli.

Description of the means of evaluating the effects of various industrial poisons will be found given in detail in the various publications referred to in the succeeding text. It will be noted that a considerable latitude of experimentation is necessary in order to study the effects of a given industrial poison.

The degree of exposure (that is, the total amount of substance to which an animal has been exposed for a given length of time) is, of course, only preliminary to the evaluation of the toxicity of the substance. The behavior of the animal must be observed for this is an index of the manner in which the poison acts. Some substances may cause death immediately or within a few minutes—for example, hydrogen cyanide, hydrogen sulfide, or carbon monoxide. Other substances may act as irritants with symptoms of pain, salivation, vomiting, and purging. Ammonia gas, cadmium oxide fume, and chloropicrin, respectively, produce these effects. Other substances affect the central nervous system and produce characteristic symptoms, such as narcosis, convulsions, and paralysis.

The various symptoms, in general, serve at best only as a rough classification of toxic substances. The effects of poisons are often subtle and a variety of symptoms may be displayed. In chronic poisoning, or in the case of a cumulative poison, anatomical changes may be produced in certain organs or tissues which are characteristic for the poison. Blood changes may occur with an increase or decrease in hemoglobin, in red or white cells, and often with characteristic morphological changes in either or both the erythrocytes and leukocytes. All these and many other effects must be studied with care in the evaluation of the toxicity of a substance. It will be apparent therefore that the term *toxicity* does not refer to a fixed quantity, such as, say, a constant of nature. On the contrary, it is a descriptive term and is often more clearly understood when applied in relation to other analogous substances. When the toxicity of a substance is evaluated in the broad sense described above, however, the industrial toxicologist is able to advise somewhat definitely the limitation of possible exposure of human beings to industrial poisons.

It should be clearly indicated that, while such procedures as those briefly indicated above serve to establish useful limits with regard to industrial poisons, the more fundamental aspects of toxicology involve far more extensive and difficult physicochemical and physiological investigation. Various physical factors such as surface activity, solubility, dispersibility, polarity, partition coefficient, particle size, and electrophoretic properties may require study. The mechanism whereby many substances produce toxic effects in man is important and requires careful exploration. The fate of these substances in the body—the changes which organic compounds undergo and the metabolites formed—serves to indicate the means by which the body attempts to reduce their toxicity and thus protect itself against their poisonous effects. This is especially true of organic compounds completely foreign to the body. In their passage through the animal organism these substances interact with the normal biochemical systems which they encounter. The enzymic systems which carry out the oxidations, reductions, hydrolyses, and syntheses in the body may be variously affected. The metabolic processes involved reflect an attempt to reduce or abolish the toxic action or damage to the organism. However, it is not true that the product or products formed are invariably less toxic than the parent substance. For example, Channon and his associates (7) found that 2,4,6-trinitrotoluene (α -TNT) is partly converted in the animal organism to 2,6-dinitro-4-hydroxylaminotoluene which is excreted as such in the urine. Furthermore, the biological reduction product of picric acid is picramic acid. In both these cases the end-products are more toxic than the original substances. Nevertheless in the case of many other poisons the toxic substance is converted to harmless metabolites and eliminated. Fundamental investigation of the toxicity of a given substance therefore includes study of the course of the toxic agent through the body, its effects on various organs and tissues during its passage, and the changes produced in the substance by the detoxicating mechanism of the body as indicated by the metabolites formed.

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ASBESTOS

Characteristics

Asbestos, amianthus, earth flax, stone flax, mountain cork, is a characteristically silky, fibrous mineral, the composition of which varies with its source. The form known as chrysotile is derived from serpentine and is a hydrous magnesium silicate containing from 12.5 to 14 per cent water of crystallization. About 95 per cent of commercial asbestos is chrysotile. Chrysotile has the silkiest and strongest fiber and can be spun. The fibers may be as long as six inches in length. Asbestos derived from amphibole occurs as a variety of minerals consisting of iron, calcium, and magnesium with little water of constitution. The latter type of asbestos consists of short fibers and is usually inferior to that derived from serpentine. Amphibole asbestos (anthophyllite) while not so suitable for spinning is more stable chemically than chrysotile and more resistant to acids and heat.

Canada has been an important source for the chrysotile asbestos used largely in the United States. The Canadian asbestos industry is centered in the Thetford Mines area of the Province of Quebec. A new source for chrysotile type asbestos is a large quarry on the eastern shoulder of Belvidere Mountain in Vermont which was opened in the summer of 1944. This deposit is of importance because it represents the only large source of long-fibered chrysotile so far found in the United States. Deposits of amphibole asbestos are mined in Georgia and North Carolina.

Industrial Uses

Asbestos is an important substance in industry and consumption in the United States for 1945 amounted to 377,875 short tons (1). Due to its fibrous nature, flexibility and heat resistant properties, it is used extensively for valve packings, gaskets, boiler lagging, and pipe covering in industrial plants and as friction material in the automotive industry. A considerable market exists in the building industry for asbestos-cement products, heat insulation, and fire-proofing. The utilization of asbestos for fibers for spinning in the manufacture of asbestos clothing for fire fighting is an im-

portant use for asbestos. The largest single outlet for asbestos in manufactured products in 1944 was for clutch facings, and next in quantity of output were brake linings. Asbestos roofing consumed the third largest amount of asbestos in that year.

Industrial Injury

The inhalation of asbestos dust produces a condition known as asbestosis. While certain other minerals of minor importance have been shown to produce lung fibrosis, asbestos is the only important silicate apart from talc and mica which does not contain free silica and yet produces pulmonary lung fibrotic changes leading to disability and death. Asbestosis occurs chiefly in industrial plants where asbestos is fabricated. The spinning and weaving of asbestos in combination with other textiles results in exposure of workmen to asbestos dust. The long continued inhalation of asbestos dust results in a form of pneumoconiosis. The primary effect of inhalation of asbestos dust is an interstitial pulmonary fibrosis. On an X-ray film the shadows cast by this type of fibrosis resemble ground glass in appearance and usually extend over the lower portions of the lung fields, frequently being heavier on the right side (2). Unlike silicosis, nodular fibrosis has not been detected in asbestos workers (3). A progressive dyspnea, variable cough, substernal chest pains, decreased chest expansion, weakness, emaciation, clubbed finger tips, and curved fingernails are the chief symptoms of asbestosis, as in silicosis. A characteristic finding in asbestosis is that of asbestos bodies in the lungs and in the sputum (4). The so-called asbestos bodies are apparently formed only in the lungs and may be demonstrated microscopically on sectioning lung tissue or in the sputum. The core of the body is an asbestos fiber which is surrounded by protein deposits. Unstained specimens are golden yellow or golden brown. They are not stained with ordinary histological stains but may be demonstrated by the Prussian blue staining procedure. The reaction to the fibrous needle in the tissue which becomes manifest in exudate cell infiltration is accompanied by numerous giant cells containing foreign particles and an increase of diffuse interstitial connective tissue and fibrosis. While the essential reaction to asbestos particles is considered to be chemical by many investigators others consider the pathogenesis of the disease to be mechanical (5) in nature. When the lungs are examined by the naked eye after death, they are large and densely fibrotic. Often the lung is completely adherent to the chest wall and, in advanced cases, to the diaphragm with the formation of a thick and extremely dense layer of fibrous tissue. Four main complications and sequelae of pulmonary asbestosis are purulent bronchitis, bronchial pneumonia, pulmonary tuberculosis, and emphysema (6). Several cases of asbestosis have been reported which progressed to a fatal termina-

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tion with heart failure and without evidence of infection or other complicating disease (7). Any appreciable decrease in the amount of asbestos dust will cause a decrease in the incidence and severity of asbestosis (8). It would appear that if the dust concentration in asbestos factories can be kept below five million particles per cubic foot, new cases of asbestosis would not arise (2).

Analysis

While the analysis of asbestos dust as an aerial contaminant is not of particular importance, its microscopy and above all the evaluation of the number of particles per cubic foot of air is of paramount importance. Air samples may be secured by the impinger method using 25-50 per cent alcohol as a collecting medium and dust counts made by the usual method. Microscopic examination of the dust reveals typical asbestos fibrous particles which may be accompanied also by cotton or other textile fibrous materials in samples taken from the air of weaving factories. The index of refraction being only slightly greater than that of Canada balsam, the relief is low. Other forms of asbestos than chrysotile have somewhat higher indices of refraction. Extinction is parallel except in the case of tremolite which has oblique extinction. The birefringence of chrysotile is moderate $n_x - n_z = 0.013$. The maximum interference color is bright yellow of the first order. The air sampling of asbestos dust both by the impinger method and by the electrostatic precipitator method is discussed in detail by Fehnel (9).

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SILICA

Silica (SiO_2) or silicon dioxide, is the most abundant of all the minerals and rocks that form the earth's crust. It is characterized by its hardness and chemical resistance to reagents. It is slightly soluble in alkalis but the finely particulate material is only very slightly soluble in water (1, 2).

Silica fractures into very minute angular particles. In the crystalline form it occurs as quartz but two other forms are also known which are tridymite and cristobalite and each of these exists in a number of modifications. Sand, flint, and agate are familiar forms of silica and diatomaceous earth, which is occasionally found in nature in large deposits, is composed of the silicious skeletons of diatoms. In view of what is known about the disease called "silicosis", it is important to distinguish between silica in the free state, as SiO_2 , and silica in the combined state, such as the various silicates. The silicates are still considered innocuous when inhaled as dust with the exception of talc, mica, and the fibrous silicates which are known as asbestos (3). The percentage of free silica in the various dusts which have been analyzed in connection with health studies of workers in dusty trades has been shown to range from 54 per cent to a trace (4). Silicosis is a chronic disease caused by the inhalation of particulate matter containing free or uncombined silica. It is characterized anatomically by generalized fibrotic changes with miliary nodulation in the lungs. Clinical signs are shortness of breath, a lowered vital capacity, a lowered capacity for work, increased susceptibility to tuberculosis, and a characteristic X-ray appearance of the lungs.

Silicosis is found to occur in such occupations as mining, the cutting of sandstone and granite, the coal industry, the smelting, refining, and grinding of metals, the manufacture of certain abrasives, the pottery industry, and the processing of the various forms of free silica. The number of workers exposed to dangerous amounts of silica dust has been estimated at more than one million (5).

Because of the disabling nature of silicosis (6), the extent to which exposure occurs in industry and the number of workers involved, many surveys have been made and these investigations have resulted in the setting up of efficient protective measures in working establishments. Dust control measures, taking into consideration the chemical composition of the dust and the size of the silica dust particles, as well as the concentration of the dust in the air, have been instituted. There is more or less general agreement that it is desirable to avoid concentrations of more than five million particles per cubic foot of air in working places where the dust contains a high percentage of free silica. Granite dust, which contains about 35 per cent free silica, when in concentrations of ten to twenty million particles per cubic foot has been found not to cause disabling silicosis in a working lifetime, while anthracite dust, containing less than 5 per cent free silica, has been found not to cause anthracosilicosis in concentrations of less than fifty million particles per cubic foot (3). Because air-borne dust may differ markedly from that of the source material from which it arises, it is common practice in appraising dust hazards to determine the

free silica content of samples. Furthermore, it has been indicated that toxicity increases sharply with silica dust below 3 microns in size (7). Since particles too large to be significant in silicosis production often contain a much higher percentage of free silica than fine particles of significant size, Holden and his associates (8) describe a procedure to eliminate the oversize particles before analysis.

The mechanism and pathology of silicosis have been very adequately described. It is well recognized that very fine particulate silica is carried to the air sac or alveolus which represents the terminal dilatation of the bronchioles in the lungs. The alveoli are in intimate contact with blood vessels through which the oxygen-carbon dioxide interchange phenomena of respiration occur and they are also in contact with lymphatics which are important for the removal of foreign or irritant material. This defense mechanism is motivated by the activity of phagocytic cells which carry off the minute silica particles to the lymph nodes. There an ineffective accumulation of this material occurs and fibrotic changes take place resulting in areas which constitute the silicotic lung marking revealed by the chest roentgenogram. Just why the phagocytic cell is affected by the silica—whether, in fact, silica has a direct toxic action, or whether it initiates other effects—is still in the realm of speculation.

Certain substances, such as aluminum, iron, and magnesium dusts, when used with quartz, as well as coal and cement dusts, have apparently been found to decrease the pulmonary changes associated with free silica alone. Outstanding in this respect is the work of Denny, Robson, and Irwin (9), who have shown that small quantities of metallic aluminum powder almost completely inhibit the solubility of silicious material. While this work represents an interesting and significant development in silicosis research, Heffernan (10) has recently pointed out that the surface activity of the silica particles is the important factor in the production of silicosis rather than solubility of the silicon dioxide. Recent investigation of the use of aluminum and alumina has been made by Policard (11), who confirms the view that the aluminum dust itself is innocuous and by King (12), who, in a critical discussion of the solubility of silica, reports that a mixture of one per cent of aluminum hydroxide with the quartz fractions lowered their solubility but did not prevent the formation of fibrosis in animals. The phase of silicosis therapy indicated above, however, is still in the stage of experimental investigation.

Since free silica or quartz is the important factor in causing silicosis, the identification of this substance and its quantitative evaluation as a dust constituent are of prime consideration. The microscopic examination of such dusts yields especially valuable information. Of the six distinct silica minerals, quartz, chalcedony, and opal are the more common. Tridy-

mite and cristobalite are more or less of volcanic origin and are also found as constituents of silica bricks, while lechatellierite is exceedingly rare. Quartz and chalcedony have refractive indices near that of Canada balsam and a birefringence of about 0.009. The other silica minerals have lower indices of refraction and weaker birefringence. The microscopic examination of such industrial dusts as are known to contain free silica can only be satisfactorily accomplished by the use of the petrographic microscope and a considerable amount of experience is necessary. Quantitative evaluation of the quartz content of a dust may be made microscopically by examining the dust and counting the dust particles in two different immersion media (13) having different indices of refraction. In one of these media all the particles are visible; in the other, having the same index of refraction as quartz, only the nonquartz particles are visible. The difference between these two estimations gives some approximation of the silica content of the dust. The method is not suitable for very fine particles and estimation based on particles of less than 10 microns in size is not good petrographic practice. Chemical methods for the estimation of free silica, such as the hydrofluosilicic acid method (14) and the fluoboric acid method (15), are tedious, time-consuming, and frequently yield information of no more quantitative value than less lengthy procedures. The X-ray diffraction method of dust analysis has become of increasing importance and is especially commendable from the point of view of speed, while it appears to yield results of as great accuracy as is obtainable by other methods of analysis.

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THE SILICONES

Characteristics

Silicon forms a large number of compounds which are analogous to those of carbon. The simplest of these is silane, SiH_4 , which corresponds to methane. While these compounds have been known for some time, they were, however, of academic interest only and received no practical application until about 1940 when the unique properties of the silicones caused them to assume considerable industrial importance. The silicones, R-SiO-R , correspond to the ketones but differ from the latter in most of their properties. The methyl silicones occur as oils, resins, and elastomers or rubber-like substances. The methyl silicone oils are soluble in benzene and the lighter hydrocarbons but are only partially soluble in alcohol. Many very complex alkyl and aryl silicones have been reported and studied within recent years. Some of these substances, such as methyl silicone oil, are characterized by their chemical inertness, low temperature coefficient of viscosity, wide liquid range, and low freezing point. Their high boiling points and heat stability are important properties for many industrial applications.

Industrial Uses

The uses of the silicones are as varied as their properties. Certain of the liquids are used as damping fluids for instruments and machinery. Other liquids find application as special lubricants for high temperature instruments, while others are used as high viscosity heat transfer liquids and as heavy diffusion pump oils. Minute amounts prevent foaming in fuel oils—an important problem in Diesel engines—and also the foaming of aqueous solutions. The relative insolubility of methyl silicone oil in petroleum oil may account for the success of this substance in suppressing foam. Silicone greases are used as hot valve lubricants, as insulating

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BENZENE

Characteristics

Benzene, benzol, C_6H_6 , boiling point $80.094^\circ C.$, melting point $5.51^\circ C.$, density $D_{4^0}^{20} 0.87895$, and index of refraction $n_D^{20} 1.50124$, is a colorless liquid with a characteristic odor. It is soluble in water only to the extent of 0.06 part per hundred at $20^\circ C.$, but is miscible with organic solvents. Its flash point is from $-12^\circ C.$ to $-10^\circ C.$ and ignition may be caused by open flames or sparks from electrical appliances. The "light oil" from the fractional distillation of coal tar consists principally of the hydrocarbons—benzene, toluene, xylene, and "solvent naphtha". On nitration, benzene yields both nitrobenzene with a boiling point of $209^\circ C.$ and *m*-dinitrobenzene with a melting point of $90^\circ C.$ On bromination, benzene yields bromobenzene, boiling point $157^\circ C.$ and *p*-dibromobenzene, melting point $89^\circ C.$ When heated with bromine plus iron catalyst, it yields mainly

p-dibromobenzene. It forms a picrate of colorless needles, melting point 83.9° C. With ClSO_3H , it yields benzenesulfonyl chloride, melting point 14° C., boiling point 251° C., and diphenyl sulfone with a melting point of 128° C. It is slowly sulfonated with boiling concentrated sulfuric acid.

Industrial Uses

Domestic production of benzene has more than tripled during the past decade, chiefly due to the increased demand for the manufacture of phenol and for a motor fuel additive. The production of benzene amounted to 36,555,643 gallons for all purposes except motor fuel in 1940. Nearly three times as much (101,140,079 gallons) was produced for use as motor benzene alone in that year (1). During the war, however, the use of benzene for chemical purposes increased considerably, while its use for motor fuel dropped correspondingly. Thus in 1945, the total production of benzene for motor fuel amounted to 32,151,532 gallons, while the production of benzene for all other purposes corresponded to 159,013,084 gallons (2). Apart from motor fuel, benzene is mostly used in manufacture of rubber, chemicals, dyestuffs and intermediates, explosives, aniline, phenol, resorcinol, benzidine, picric acid, and miscellaneous organic preparations. It is used as a solvent for oils, fats, waxes, gums, natural and synthetic resins, cellulose esters and ethers, and a large variety of varnishes, airplane dopes, as well as in paint and varnish removers.

Toxicity

On inhalation in high concentration, benzene is narcotic in action producing successively euphoria, hypermotility followed by sleepiness, and fatigue. The preliminary excitement is greater than with chloroform and as a general anesthetic, its action is more of a convulsive character. In concentrated form, it is especially toxic and may produce death. In dilute form, the vapors produce headache, vertigo, ataxia, twitchings, convulsions, and coma. Chronic poisoning in industry is characterized by leukopenia and aplastic anemia. Bowers has recently described a chronic case of benzene poisoning with fatal termination characterized by an intractable anemia, leukopenia, and thrombocytopenia following long continued exposure to benzene. In this case, the bone marrow was grossly hyperplastic with extensive areas of extramedullary hemopoiesis (3). It should be emphasized that blood changes alone cannot be accepted as evidence of benzene intoxication in the absence of estimations of benzene as an atmospheric contaminant (4). Benzene is absorbed through the skin but clinically detectable benzene poisoning has not been found from this source (5). However, absorption from the skin is of far less importance than absorption from inhalation. A test for detecting active absorption

of benzene may be made by determination of the ratio of the total urine sulfates to the inorganic sulfates (6). Since serious trouble may develop following exposure to benzene without any warning symptoms, the significance of neglected disorders is often apparent when too late. Lambin (7) has indicated a procedure for the detection of benzene poisoning applicable to large groups of individuals so exposed. The 1949 American Conference of Governmental Industrial Hygienists has accepted 35 parts of benzene in air as a maximum allowable concentration in workrooms.

Analysis

Methods for the determination of minute amounts of benzene are at best, methods of approximation only. The chemical methods are colorimetric in character and depend upon nitration and colorimetric evaluation of the reaction products. Various physical methods have been developed for the determination of benzene, such as the gas interferometer method and the combustible gas detector, and are useful when the air contaminant is solely benzene vapor. In contact with a nitrating mixture (equal parts of concentrated sulfuric acid and fuming nitric acid of d. 1.50) in the cold for 30 minutes, benzene is converted into a mixture of the *ortho*, *meta*, and *para* isomers of dinitrobenzene. The mixture contains about 95 per cent of the *meta* isomer. With aldehydes and ketones, the mixed dinitrobenzenes give a violet color in alkali solution. The *meta* isomer in particular gives a color with acetone. In low concentrations of benzene, the color develops within 30 minutes, is stable for three hours, and follows Beer's law. Schrenk and his associates (8) have adapted the nitration method to the determination of small amounts of benzene vapor as an air contaminant, using methyl ethyl ketone as reagent. A combined absorption nitration bubbler tube is used in which the benzene is converted to *m*-dinitrobenzene which is then estimated colorimetrically in saline solution with methyl ethyl ketone. Both Baernstein (9) and Dolin (10) have published methods for the determination of benzene in the presence of toluene and xylene, which depend upon oxidation of the nitro derivatives of the two latter substances. The oxidation products of the nitro derivatives of toluene and xylene are colorless under the conditions described and therefore do not interfere with the benzene determination.

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BENZENE HEXACHLORIDE

(Gamma Hexachlorocyclohexane)

Characteristics

Benzene hexachloride, hexachlorocyclohexane, $C_6H_6Cl_6$, one of the newer insecticides also known as "BHC" or "666", exists in five isomeric forms—the *alpha*, *beta*, *gamma*, *delta*, and *epsilon*. In the pure state, the isomers are well-defined colorless crystals, practically insoluble in water, soluble in organic solvents, and have a bitter taste and practically no odor. The melting points of the isomers are as follows: *alpha*, 157.5–158° C.; *beta*, 309° C.; *gamma*, 112.5° C.; *delta*, 138–139° C.; and *epsilon* (1), 218.5–219.3° C. At 40° C., the vapor pressure of these isomers are *alpha*, 0.06 millimeter of mercury; *beta*, 0.17 mm. Hg.; *gamma*, 0.15 mm. Hg.; and *delta*, 0.09 mm. Hg. The solubilities of the isomers of benzene hexachloride have been determined in each of forty-five organic solvents and the con-

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THE OCCURRENCE OF PULMONARY FIBROSIS AND OTHER PULMONARY AFFECTIONS IN ASBESTOS WORKERS*

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INTRODUCTION

PRIOR to the commencement of this inquiry, in February, 1928, definite knowledge existed of only two deaths of asbestos workers about whom there was expert opinion that the inhalation of asbestos dust had at least contributed to, if not caused, the fatal outcome.

The first of these, in retrospect the most suggestive, only came to light some years after the occurrence, when full information was unobtainable. All that is known of this case, now referred to as "the Montague Murray Case," is contained in the evidence given by Dr. Montague Murray before the Departmental Committee on Compensation for Industrial Diseases in 1906 (1). From this source, we learn that:

The patient, a male aged 33, came under the care of Dr. Montague Murray at the Charing Cross Hospital in the beginning of 1899. He had worked with asbestos for "some 14 years," 10 years as a cardroom hand, and the remainder in some other room of the factory, "where there was much less dust." He volunteered that, of the 10 people working in the cardroom when he went into it, he was the only survivor, and that all the others had died somewhere about 30 years of age. There is no note as to the nature of his work, previous to that in the asbestos factory.

He was treated in the Charing Cross Hospital for two months, and then returned to work. After a few months, however, he became ill again, and was re-admitted to the Hospital in April, 1900, where he died. The post-mortem examination confirmed the clinical diagnosis of extensive pulmonary fibrosis. There was no evidence of pulmonary tuberculosis, and examination of the sputum for *B. tuberculosis* was negative.

The second case was reported by Dr. W. E. Cooke in 1924, eighteen years later (2):

The deceased, a woman, aged 33, who died in 1924, had worked in asbestos for 18 years, but intermittently for the last 5 years, owing to periods of ill-health. The post-mortem examination revealed, not only extensive fibrosis of the lungs, but also much change due to pulmonary tuberculosis.

Although Cooke (3) and Stuart McDonald (4) were conclusively of the opinion that in this case the lungs showed a progressive dust fibrosis, together with a chronic tuberculous infection, the etiologic relationship between the inhalation of asbestos dust and fibrosis of the lungs would have been strengthened by the absence of a tuberculous infection.

Cooke's case is, however, of outstanding importance, not only because of the discovery of "curious bodies" in the lungs—discussed later—but also, and of more importance generally, be-

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cause its publication again directed attention to the possibility that inorganic dusts containing little or no free silica may be productive of extensive pulmonary fibrosis. Of these dusts, the silicates form a very large class, of which asbestos is but one example. Many other members of the class, such as the feldspars, kaolin, French chalk, and pumice, are extensively used in industry. The importance, therefore, of delimiting the potentialities of the class as producers of pulmonary fibrosis is clear.

Cooke's case was the first to be generally reported in the medical press; the facts of the Montague Murray case, although contained in the evidence presented before the Departmental Committee in 1906, and published in 1907, were liable to be overlooked in the mass of important material on industrial diseases elicited by that Committee.

Since Cooke's case, Dr. I. M. D. Grieve has made a careful study of a group of asbestos workers in his practice, and has courteously allowed access to his records.

In February, 1928, Dr. MacGregor, Medical Officer of Health for Glasgow, drew my attention to an asbestos worker who was receiving treatment in one of the hospitals in that city. This case, the details of which have been reported by H. E. Seiler (5), presented, both clinically and radiologically, signs of a diffuse pulmonary fibrosis, with no evidence of a tuberculous infection. On further investigation into the patient's industrial and medical history, no presumptive cause, other than the inhalation of asbestos dust, was found to account for the existence of the fibrosis.

This case, at that time the third of which the Factory Department had knowledge, was, however, the first in which the four essential conditions, necessary to establish a relationship between the inhalation of asbestos dust and the development of fibrosis, could be demonstrated. These conditions are:

1. Work involving exposure to asbestos dust.
2. The existence, demonstrable clinically and radiologically, of a definite pulmonary fibrosis.
3. The absence of previous or present infections known to cause pulmonary fibrosis—e.g., tuberculosis, influenza, or pneumonia.
4. The absence of previous or present work involving exposure to other dusts, which might cause pulmonary fibrosis.

These conditions being fulfilled, a relationship between the inhalation of asbestos dust and the development of the pulmonary fibrosis may be presumed.

The importance of establishing whether the supervention of this disease in an asbestos worker was an exceptional occurrence, or evidence of a grave health risk in the industry, was now apparent, and steps were taken, forthwith, to obtain *prima facie* evidence in proof, or disproof, of the existence of such a risk.

A number of workers in asbestos were selected and examined clinically and radiographically. The findings invited further investigation throughout the industry, with the result that a comprehensive inquiry involving the investigation of the different processes in relation to the evolution of dust, and the examination, both clinical and radiologic, of workers, was undertaken during the year 1928, commencing

with the carding, spinning, and weaving processes of the industry.

In the meantime, in March, 1928, the death of an asbestos worker (one of Dr. Grieve's cases) occurred in another part of the country, and, on post-mortem examination, a condition of widespread fibrosis of the lungs, without tuberculosis, was revealed. The microscopic examination also disclosed the presence of the curious bodies.

In 1928, also, Dr. F. W. Simson (6) reported a fatal case of fibrosis of the lungs occurring in a native working in an asbestos mill in southern Rhodesia.

In order that this brief survey of the events leading up to the present inquiry may be complete, it is necessary to review the investigation made in 1910 to 1911 by Dr. Collis and Miss Whitlock—the only previous inquiry into the subject.

In May, 1910, the Registrar-General drew attention to a death which had been certified as "acute pulmonary phthisis in an asbestos worker," and to a statement (for which he was unable to vouch) that seven other deaths from phthisis had occurred in the same factory.

Following this, extensive inquiries were made, a medical report on all the employees in the factory concerned was obtained, and Dr. Collis and Miss Whitlock investigated generally the various processes in the industry with respect to the evolution of dust, methods of ventilation, lost time due to sickness, etc. Also inquiries were made of the Canadian government as to the conditions in the asbestos quarries and mills in that Dominion, evidence of increased sickness and mortality rates among the workers,

and any methods of ventilation which had been found of especial value.

The result of this investigation did not conclusively prove that asbestos possessed injurious properties, but it pointed to the probability that such was the case. It is interesting to consider why it was that no conclusive proof one way or the other could be obtained, then, as to the injuriousness of asbestos dust. Some, if not all, of the factors which affected the position then are, in varying degree, continuing factors, and have an important bearing on the present inquiry. Their operation, while providing a solution to this query, also affords an explanation to another pertinent question: Why is it that this industry, founded in antiquity, has only recently excited attention, by reason of its raw material becoming suspected as a cause of industrial disease?

The answer appears to be that in the past it was not practicably possible to obtain proof of the injuriousness of asbestos dust, considering the limitations imposed by the state of the industry, and the point reached by research work into the relationship between dust inhalation and diseases of the lungs.

The industry itself, not a large one today, was then (1910) considerably smaller. Certainly it had begun to grow rapidly, but the number of workers who could have been employed for a period of time long enough to allow of the development of definite physical signs of pneumokoniosis must have been quite small, and dispersed over the country.

Precise knowledge of the morbid affections of the lungs produced by the inhalation of dusts was more fragmen-

tary than it is today. The position was clearly stated two years later in evidence placed before the Royal Commission on Metalliferous Mines and Quarries. This Commission, when reporting in 1914 (7), stated "we do not know whether other dusts besides those containing free crystalline silica induce a pathological condition in the lungs, though the experiments of Professor Beattie in animals suggest that this may occur." It is only in the last year or two that research has produced some definite evidence as to the precise effects of some of these dusts on the lungs (8) (9) (10) (11) (12).

In 1910, radiography of the lungs, in both its technical and its interpretative aspects, was still in its infancy and, so far from having attained its present status of being an indispensable aid to the diagnosis of the dust diseases of the lungs, was an unused ally.

In addition, the existence of a measure of exhaust ventilation in the most dusty processes of the industry, incomplete as it was, had important results in modifying the onset, course, and duration of any pathologic lung conditions resulting from the inhalation of the dust. This influence has been much more pronounced in the period intervening between 1912 and the present inquiry, and will be referred to again.

Another factor which has tended and still tends to obscure the possible deleterious effects of the non-silica dusts, is the general use of the phthisis mortality rate as a comparative index of the degree of injuriousness of the dust encountered in the various dusty occupations. This rate, while of great value in separating dusty industries into two great groups—those which

show an excess mortality from phthisis, and those which do not—as well as being a comparative index of the industries belonging to the former group, not only is of little value as a means of classification in the latter group, but also tends to distract attention from it, and to result in the associated dusts being dismissed as more or less innocuous.

Evolution of dust is only one factor, though an important one, which may cause variations in the phthisis mortality rate in different industries. Wages, hours of work, aggregation of workers, amount of food, housing, and other social and environmental conditions are, however, powerful influences in the same direction, and are not necessarily comparable as between the workers in any two industries.

In thus reviewing some of the influences which have retarded recognition of the baneful effects of some dusts upon the lungs, the singular attributes of pulmonary fibrosis, the most important of the diseases caused by the inhalation of dust, must not be overlooked.

This disease, insidious in its onset, stealthily advances with but faint warnings of its progress; inexorably it cripples the essential tissues of the lungs, yet for a considerable period causes almost no inconvenience to the worker. As time goes on, however, the lungs find more and more difficulty in re-aerating the blood; and breathing is quickened on slight exertion. Still the worker is able to remain at work, but is aware of his undue shortness of breath on extra effort. Usually, however, he ascribes it to causes other than the dust he is inhaling.

As the disease progresses, if no acute illness has caused a fatal termination,

a stage is reached when the lungs can do little more than maintain life; and the shortness of breath is extreme. Even in its terminal stages, the disease, deceitful to the last, may masquerade as chronic bronchitis, pulmonary tuberculosis, bronchopneumonia, or the like.

While more or less acute cases of fibrosis closely simulating miliary tuberculosis have occurred, even in this country (13), they have all been associated with the inhalation of dense concentrations of free silica dust, and are, fortunately, the exception rather than the rule.

The difficulty of diagnosing pulmonary fibrosis, especially in its early stages, or if complicated by tuberculosis, has been stressed by a number of authorities, and has, undoubtedly, contributed to impede the attainment of precise knowledge of the extent to which the various industrial dusts affect the lungs.

Difficulties and obscurities still impede, though to a less degree than in the past, any investigation into the effects of an industrial dust upon the lungs of those exposed to it; but prior to the War, although there were certain slight indications that asbestos, in common with some other dusts, might produce permanent pathologic changes in the lungs, it was not possible to obtain evidence sufficient to prove or disprove this hypothesis.

At the outset of the present inquiry into what, if any, pulmonary diseases workers exposed to the inhalation of asbestos dust are more prone to contract than the general population, it was considered essential to view the problem afresh, and with complete detachment, because of the fact that

asbestos dust has a totally different physicochemical constitution from that of dusts containing much free silica and causing silicosis. It was felt that although much valuable guidance could be obtained from the methods of investigation of silicious dusts, care had to be taken to keep an open mind, so as not to be unconsciously biased in the direction of assuming that the effects of the dust, if any, must be comparable in some degree to those of crystalline silica.

ASBESTOS AND THE ASBESTOS INDUSTRY

Asbestos

The term asbestos is a collective name, of no definite mineralogic significance, which has been applied to a variety of silicate minerals, which differ from one another in chemical composition and physical properties, but which resemble one another in their finely fibrous feature and flexibility (14). Their value depends on the facility with which they are capable of being split up into long and flexible fibers, which can be spun like cotton and woven into cloth; on their resistance to heat and acids; and on their insulating properties with respect to heat and electricity.

Varieties of asbestos possess these qualities in differing degree. Commercially, therefore, selection is made by the manufacturer of that variety and grade which is most suitable for the purpose in view, regard being paid to ordinary economic factors, such as the cost of the raw material, and the price which the finished article may be expected to command.

Consignments of asbestos of the same general variety, but with differ-

ent countries of origin, are frequently mixed in the preliminary processes of manufacture. Thus Canadian chrysotile may be mixed with Russian, or Rhodesian chrysotile, and so on. Less frequently, totally different varieties, such as amosite and chrysotile, may be mixed.

Practically speaking, all that goes under the name of asbestos, in commerce, is either fibrous serpentine or a fibrous mineral of the amphibole, or hornblende, group. The former is the most important commercially; but strictly, the mineralogists confine the

bined water, and usually more calcium, aluminium, and iron. Members of this group are resistant to acids, but are more difficult to spin, some being quite unsuitable for this purpose. The most important members of this group are crocidolite, amosite, and tremolite.

Crocidolite and amosite are mainly silicates of iron, the former having a beautiful lavender-blue color, the latter being brownish-gray. Both are spun and the yarn is woven into cloth for various purposes, such as acid filtering, and for making into insulating

TABLE 1.—COMPOSITION OF SERPENTINE AND AMPHIBOLE VARIETIES OF ASBESTOS

MINERAL GROUP	VARIETY	PERCENTAGE OF							
		SiO ₂	Al ₂ O ₃	FeO Fe ₂ O ₃	MgO	CaO	Na ₂ O	K ₂ O	Combined Water
Serpentine	Chrysotile	39-42.5	0-3.7	0.7-4.4	39-43	0-0.35	...	...	13.3-16.5
Amphibole (horn- blende)	Crocidolite	50.5-52.1	0-1	35.5-37.4	0-3	0.75	6.2-9	...	1.6-4.5
	Amosite	48-53	1.2-9.4	34-44	0.7-6.4	...	0-2.5	...	2-3.8
	Tremolite	57.2	0.9	3.2	22.8	13.4	0.6	0.3	2.4

term asbestos to fibrous forms of hornblende. These two types are sharply distinguishable, chemically and mineralogically.

Serpentine asbestos, or chrysotile, is a hydrated magnesium silicate, containing practically no calcium, a high percentage of combined water, and a low percentage of iron. This variety is very suitable for spinning, but is attacked by acids. Nearly 80 per cent. of the world's production of asbestos is derived from Canada, and is of this variety; the remainder comes mainly from South Africa and Russia.

The amphibole, or hornblende, varieties contain less magnesium and com-

mattresses, as well as for other purposes. Both are produced extensively in South Africa, from which quarter all required commercially is obtained. Amosite, a comparatively recent discovery, is found there in very large deposits; its use is steadily increasing, the initial difficulties associated with the manufacture of textiles from this variety having been overcome.

Tremolite, found in various quarters of the world, has been mined in northern Italy since the time of the Romans. Its chief use is in the manufacture of asbestos millboard and for filtering purposes.

Table 1, compiled from various

sources, shows the main differences in the composition of these four varieties.

Only a very small proportion of the world's production of asbestos, which is between 300,000 and 400,000 short tons per annum, is suitable for spinning, and the most desirable grades of spinning fiber, consequently, command a high price—now over £100 a ton. The shortage of this grade has led to improvements in manufacturing processes which have enabled less expensive grades of fiber to be utilized for spinning, some of which give rise to an increased amount of dust.

About four-fifths of the world's production of asbestos is fiber unsuitable for spinning, and this is used in the manufacture of asbestos millboard, tiles, sheeting, paper, and many other articles. It is the discovery of industrial uses for these very short fibers, and the dust-like waste, which has been responsible for the phenomenal expansion of the industry as a whole. The spinning and textile section has also shared, because of the extensive use of the yarn and cloth in the manufacture of steam packings, insulating mattresses, brake linings for motor cars, fireproof curtains, and the like.

In 1880, three years after the discovery of the large Canadian deposits, the world production of asbestos was little over 500 short tons; by 1900 it had risen to about 35,000 short tons, by 1920 to over 230,000 short tons, and by 1925 to over 330,000 short tons (15).

The imports of asbestos (all grades) into the United Kingdom rose from 18,591 tons in 1922 to 33,520 tons in 1927 (16). The figures for 1927 refer to Great Britain and northern Ireland only. Of these quantities 8,844 tons and 3,794 tons, respectively, were

reexported. Thus the consumption of asbestos in this country trebled within five years.

The Industry

On considering the uses of asbestos one is astonished, not only at the wide range of articles manufactured from this mineral, in greater or less proportion, but also at the diversity of industries which nowadays find its use necessary, either in the form of the raw material, or as manufactured articles.

Evidently, therefore, with the multiplicity of processes and dusts encountered in the ramifications of the industry, discrimination would have to be exercised, and some limit set to the processes included in the inquiry.

The processes selected may be divided, roughly, into

1. Processes involving the manipulation of asbestos, either pure, or admixed with a small proportion of cotton, or other vegetable fiber.
2. Processes involving the manipulation of asbestos together with other dusty materials.
3. Processes involving the making up of asbestos cloth into other articles.

Group 1 entails exposure to asbestos dust mainly, and to cotton, or other vegetable dust, very slightly; group 2 entails exposure to asbestos dust in very varying amounts, and also exposure to divers other dusts, such as brick dust, magnesia, kieselguhr, fossil meal, and cement; in group 3 the exposure to asbestos dust—provided no other asbestos processes are being carried on in the vicinity—is, in the majority of cases, negligible. Processes belonging to all three groups may be carried on in the same factory, and workers may transfer from one department to another (30).

Investigation into the intricate question of the effects of mixed dusts, while possibly productive of some general corollary, would lose much of its value in the absence of knowledge of the effects of the several component dusts, and moreover would introduce an incalculable variable into the final results. For these reasons, and also because the Montague Murray case, Cooke's, Grieve's, and Seiler's cases all occurred in processes included in group 1, it was considered advisable to exclude, as far as possible, from the inquiry all workers exposed to the influence of mixed dusts, and all those not employed in processes included in groups 1 and 3.

The processes included, therefore, are the crushing, preparing, sieving, opening, mixing, carding, spinning, doubling, plaiting, braiding, and weaving of asbestos, together with the operations incidental thereto. Also included is mattress making, where the filling is asbestos, and the manufacture of some insulating materials, where the dust produced is asbestos.

The carding and spinning processes have many points of resemblance to the corresponding processes in the cotton industry, but with essential modifications and restrictions caused by the different physical characters of the asbestos fiber. These processes are aided by an admixture of cotton, or other vegetable fiber, and usually from 2 to 10 per cent. by weight of cotton is added. More rarely, and for special purposes, long-fibered asbestos is carded and spun with no admixture of vegetable fiber, but usually asbestos yarns contain a core of either cotton or metal wire.

Asbestos yarns are not only used for

the weaving of fabrics, which, themselves are used for a multitude of purposes, but, braided together, are made into ropes for use as steam packings and other purposes. The interstices and center of the rope may be filled with other materials such as talc, oil, or graphite, depending on the precise use to which the rope is to be put.

Asbestos mattresses, used for blanketing steam engines, and for other insulating purposes, are made of asbestos cloth stuffed with asbestos fiber. The stuffing material may be, however, slag wool, magnesia (containing approximately 15 per cent. of asbestos fiber), or kieselguhr with a small percentage of asbestos fiber. The manufacture of mattresses filled with mixtures of asbestos fiber and other materials has not been included in this portion of the inquiry.

The shortness, slipperiness, and lack of strength of the individual asbestos fiber, as compared with cotton, flax, wool, silk, and other textile fibers, have been the cause of much technical difficulty in manufacture. The efforts of manufacturers to cope with these difficulties, together with those due to wide variations in the physical properties of the raw material, are reflected in the methods employed by each. For these reasons, and because of the great number of patented and special products manufactured, one finds considerable differences in detail in the processes in use.

The majority of the processes mentioned result in the evolution of dust, although by no means to the same extent. Differences in plant, quality of asbestos used, methods of manufacture, type of finished article, and extent of application of exhaust ventilation,

all result in variations in the evolution of dust in similar processes in different factories.

Variations in the evolution of dust in different processes being of outstanding importance, a series of samples of dust from the air of workrooms was collected, by means of the Owens jet apparatus.

Population at Risk.—A calculation of the total number of workers employed in the processes already enumerated as

TABLE 2.—DISTRIBUTION, ACCORDING TO LENGTH OF EMPLOYMENT, OF (A) 775 WORKERS ENGAGED IN ASBESTOS PROCESSES AND (B) SELECTED SAMPLE OF 363 WORKERS

YRS. EMPLOYED	(A)		(B)	
	NO.	PER CENT.	NO.	PER CENT.
0-4.....	483	62.3	89	24.5
5-9.....	200	25.8	141	38.8
10-14.....	51	6.6	84	23.2
15-19.....	24	3.1	28	7.7
20 and over.....	17	2.2	21	5.8
Total.....	775	100.0	363	100.0

included in this inquiry, but excluding those engaged in processes included in group 3 in which the exposure to asbestos dust is negligible, gives a figure of approximately 1,600; if we could add to this the number of workers engaged in handling pure asbestos fiber only, in the preliminary stages of the processes included in group 2, we should obtain the total population at risk from the effects of asbestos dust itself. Unfortunately, this latter figure is unobtainable; but a rough estimate,

and most probably an overestimate, is 600. Thus, about 2,200 appears to be the total population at risk in this country, for the purposes of this inquiry. This figure, however, does not include the large number of workers engaged in the processes in group 2 which involved exposure to the influence of mixed dusts, of which asbestos is but one, and commonly not more than 20 per cent. of the mixture.

This estimate of the population at risk, although it may be excessive, is useful, since it enables us to judge of the adequacy of the sample of workers examined, and to apply more correctly the incidence rates of any pulmonary affections disclosed by the examination of the sample (30).

The sample examined (after eleven cases are excluded of fibrosis and pre-fibrotic conditions due to causes other than the inhalation of asbestos dust) numbered 363, representing 16.5 per cent. of the population at risk, estimated as above. The manner of selection of the sample must be referred to, since just interpretation of the results depends upon a due appreciation of the relationship of the sample to the whole population at risk.

The principle of selecting primarily those longest employed was adopted; but regard was also paid to the other end of the scale, so as to obtain information as to the length of exposure to dust necessary before effects are manifested, and also as to the particular process in which the worker was engaged. It was felt that, in this way, the maximum information would be obtained in the shortest time.

Table 2 shows the distribution, according to length of employment (not necessarily in one factory), of

775 workers engaged in the processes under review, and of the sample of 363 workers, distributed in the same way. The enormous preponderance numerically of workers employed under five years in these processes is striking, as is also the very low percentage of workers employed ten years or longer.

Comparison of the two parts of the table shows that the effect of this method of selection is that the number examined in each succeeding five-year employment group is a progressively greater proportion of the total number which *could* have been examined in each particular group. With a solitary exception, all those examined were at work on the day of examination.

Not only the value, but the necessity, of radiographic examinations of the chest in investigations into the effects of dust upon the lungs, has been emphasized repeatedly by Watkins-Pitchford, and reaffirmed by the Departmental Committee on Compensation for Silicosis (17). A high standard of radiography is essential; as Watkins-Pitchford phrases it, the radiograms must be "technically satisfactory." Indifferent films are useless, since it is the fine detail of the lung which is being studied.

In a general inquiry, such as this, which involves the examination of workers in factories, large and small, scattered over the country, it is not practicable, nor is it necessary, considering the special purposes of the inquiry, for radiograms to be taken of all the workers examined. The dislocation of work in a factory, caused by the absence of a number of hands for this purpose, cannot be viewed with unconcern, especially when, as is not infrequent, the factory is remote from

a center which is equipped with the necessary X-ray plant, and where the services of a consultant versed in the science of radiography of the lungs are available. Furthermore, it must not be overlooked that such examinations are voluntary, and not, as in South Africa, compulsory. Moreover, repeated and protracted examinations only result in the exhaustion of all concerned, and much depends on the willing co-operation of employers and employees, since the only incentive is a desire to further the common welfare.

Radiography, therefore, has a different function in these inquiries, than when it is applied to individual cases for compensation or other legal purposes. This function is not that of replacing careful clinical examinations, as has been recently foreshadowed (18, p. 40), but primarily that of being an indispensable aid in diagnosis, especially of doubtful cases, in the determination of complicating lesions, in measuring the extent and progress of the disease, in locating the point at which the earliest radiographic signs appear, and finally as a check upon the human factor presented by the examiner himself.

The examinations (except one) were carried out at each factory, in a room set apart for the purpose, suitably warmed, and with the necessary appointments. On occasions the noise of traffic or from the adjoining factory was a hindrance, but in one way or another these difficulties were overcome or minimized.

All the selected workers were examined by the writer. At three factories, however, a number of workers were examined jointly with Dr. E. L. Middleton. His far-reaching experience of the

pathologic changes in the lungs, produced by the inhalation of various dusts, was of great value, and his assistance was much appreciated.

Every effort was made to complete the clinical examination of the workers before the onset of winter introduced difficulties due to ephemeral bronchitis, colds, and influenza, which would tend to obscure the main issues. Thus the clinical, and two-thirds of the radiographic examinations were completed by the middle of November, 1928, prior to the commencement of the influenza epidemic early in 1929.

CLINICAL EXAMINATION

Percussion

The inhalation of asbestos dust originates changes in the lungs, which may be looked upon as a measure of the efforts of the living tissues to repel, or incarcerate, the irritant particles of dust. These changes modify the percussion note. It is true that the note elicited may be similar on both sides, but the note is not normal. It is thinner and higher pitched than normal, and there is a sense of resistance imparted to the plexor finger. In other words there is a diffuse, but slight, impairment of resonance.

This alteration in the percussion note, however, is more difficult to recognize because it is bilateral, and extends over a wide area; consequently the aid of contrast percussion is denied. It is best elicited by rapidly, and very lightly, percussing the back of the chest from apex to base on each side. It will be found that the extreme apexes remain clear, but below the apexes the impairment is general. It increases over the root areas; below

these, it diminishes in intensity, but still persists. In other words, we find tacked on to the paravertebral dulness an area, above and below, of impaired resonance, which is much more extensive than that usually associated with old inactive hilar tuberculosis.

Impairment of the percussion note was found to be constantly more marked in the right side; in fact, at first it was thought that in the earliest degrees of fibrosis it was confined to that side, but later, and more extended observations lead to the conclusion that the earliest detectable cases are bilateral, although the signs on the left side are tenuous.

This change is so constant that it has been adopted as the most reliable single clinical sign presented by this type of pulmonary fibrosis, and no case has been classified as fibrotic in its absence.

Considering the frequency with which signs indicating what may be termed "enlarged roots" were found in asbestos workers, it may be that paravertebral dulness is one of the earliest signs produced by the inhalation of asbestos dust, indicating congestive changes in the root areas and a choking of the lymphatics with dust. Reflex impairment of note, due to irritation of the lung tissue by dust, is, however, an attractive explanation. It does not follow, of course, that workers presenting these signs will ever develop a definite asbestos fibrosis.

Clearly, the physical signs presented by any case of diffuse pulmonary fibrosis not only may be modified by changes produced by some intercurrent lung disease, but, *ab initio*, will vary according to the state of the lungs before the onset of the fibrosis. Thus,

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the fibrosis may be implanted upon a perfectly normal chest, in which case the problem of diagnosis is straightforward. In other cases, however, preexisting root changes, so common in an industrial community, may be present, or the lungs may be already the seat of emphysema and chronic bronchitis, or of definite old tuberculous lesions, or there may be a massive pleural thickening, the result of an old pleural effusion. All these examples have been noted in the present investigation, and others will readily come to mind.

Although in most of these cases the dust fibrosis, if moderate in degree, can be confidently diagnosed, especially with the aid of radiography, it must be admitted that the problem becomes very difficult when the dust fibrosis is comparatively slight, and the changes produced by other conditions are pronounced and diffuse. Three types of these cases have caused most difficulty among those examined; fortunately, numerically they were few.

The first of these is where the dust fibrosis has been implanted upon lungs already emphysematous. It seems that, at any rate in the case of the asbestos fibrosis, until the fibrotic changes get the upper hand, clinical diagnosis of the fibrosis in these cases is impossible. Nevertheless, although the problem of the diagnosis of a fibrosis implanted upon an emphysematous chest has caused some difficulty in this inquiry, it seems likely that it can arise only under exceptional circumstances.

The second type of case which has been a source of difficulty in clinical diagnosis is that in which there are extensive bilateral fibrotic changes due

to healed tuberculosis. That there is an extensive fibrosis is clear enough, and that most of it is not due to asbestos is strongly suggested when examination of the lower portions of the lungs shows that they are comparatively slightly affected. Further help in these cases, of course, may be obtained from the history and symptoms.

The third type, also rare, is that in which changes following an old massive pleural effusion on one side so obscure the physical signs of any dust fibrosis as to render that side useless clinically for diagnostic purposes. If the side affected by the pleurisy happens to be the right, the radiographic picture is also curtailed by the normal partial obscuration of the left lung base by the heart shadow.

These three types were, with the possible exception of the first, uncommon, and are mentioned merely to call to mind some of the ways in which an asbestos fibrosis may be masked, in greater or less degree, by changes due to other disease.

Only passing reference need be made to the other physical signs found in the asbestos fibrosis, since they do not differ materially from those presented by silicosis. Chest expansion is diminished and may be reduced to one-half inch or even less in advanced cases. Retraction of the apexes is common, and sometimes shows a peculiar feature differentiating it from that found in fibroid phthisis. Instead of the immobile and sunken apexes seen in the latter disease, the apexes are seen to descend during inspiration, and to rise again during expiration. This seems to indicate the anchoring of normal apexes by fibrous tissue in the lower portions of the lungs. Some confir-

mation of this was obtained radiologically.

Auscultation

In the majority of the cases of fibrosis, the respiratory murmur is weakened, much or little, generally, more on the right side, and often still more at the base; the expiratory sound is weaker than the inspiratory, and often becomes less and less audible as one approaches the bases.

Transitional phases between this and harshened breath sounds and prolonged expiration are not uncommon, even in the same chest. The latter may be noticeable in the upper portions of the lungs, but progressively diminish toward the bases. Other combinations were also noted, however.

The dry character of this type of fibrosis during most of its course is rather striking. Scattered fine râles and clicks in the root areas, axillae, and bases were not infrequent; slight edema of the lower halves of the lungs was noted in one of the more advanced cases; but in a number, no adventitious sounds at all were heard.

Pleural crepitations, and rarely a slight pleural rub, were noted—these attacks seem to cause little pain—and in one case a little fluid at the right base.

No doubt this variability in the sounds heard on auscultation reflects underlying changes, temporary or permanent, in the lung, and is dependent, *inter alia*, on the extent of the fibrosis with its associated pleural thickening—changes due to past disease, catarrh, or other intercurrent affection, and to the degree of compensatory emphysema present.

Symptoms

The symptoms exhibited by these cases of fibrosis, as might be expected, closely resemble those of silicosis. The distribution of the main symptoms, and of one sign, cyanosis, which is included with the symptoms for convenience, is given in Table 3. A few cases have been excluded on the grounds that one or other of the symptoms complained of might be assigned to causes other than the fibrosis, such

TABLE 3.—DISTRIBUTION OF FOUR COMMON SYMPTOMS, AND OF CYANOSIS, AMONG CASES OF FIBROSIS

SYMPTOM	NO. EXAMINED ¹	CASES IN WHICH SYMPTOM WAS PRESENT	
		No.	Per Cent.
Cough.....	91	54	59.3
Cyanosis.....	93	52	55.9
Dyspnea.....	91	47	51.6
Expectoration.....	91	31	34.1
Pain.....	94	10	10.6

¹ Excluding those in which the presence of the symptom is referred to other causes.

as a complaint of shortness of breath in a case with a past history of mild thyroid intoxication, or of dyspnea associated with obesity.

Between 50 and 60 per cent. of the cases complained of cough, or of shortness of breath on slight exertion, or showed some degree of cyanosis, whereas only about one-third complained of expectoration, and one-tenth of pain, or discomfort, in the chest. Also, while 14.6 per cent. of the cases had no complaints and showed no cyanosis, and only 3.4 per

cent. presented all four complaints and were cyanosed, 60.8 per cent. presented two, three, or four of the five items.

Clearly, none of the four complaints, or the presence of cyanosis, can be an infallible indication of the existence of fibrosis; but the presence of two of them, shortness of breath on slight exertion, and cyanosis in some degree, in an asbestos worker, is highly suggestive, in the absence of other evident cause.

Nevertheless an advanced degree of fibrosis may be present, and little complaint made. Symptoms, unless distressing, are so often a function of the introspectiveness of the patient. Slight degrees of fibrosis, too, give rise to no symptoms, in the absence of an intercurrent bronchitis, since the remaining sound lung tissue is still amply sufficient for all purposes.

Cough was the most frequent symptom, and was of two types. For a few weeks after commencing work in a dusty process, asbestos workers often find the dust irritating, and cough while in the dusty atmosphere; this lasts for a few weeks, and then usually disappears. The writer has noticed this effect on himself, but only while in a very heavy cloud of asbestos dust. The dust seems to be only slightly irritant in this way. One or two men who had previously worked in cotton card rooms, or blow rooms, and were affected by that dust, stated that they were unaffected by asbestos dust. Certainly asbestos dust does not cause asthmatic attacks like those seen in cotton card room workers. One man, who migrated from a cotton card room ("hard waste") nine years ago to an asbestos card room, on account of the irritating effect of the dust, does not

find asbestos dust irritating, but to this day cannot work with a cotton scutcher (as he occasionally does) without precipitating a coughing attack within an hour or two. Although he suffers from winter cough and some shortness of breath, he stated that his health has been much better since the change.

Asbestos dust, therefore, has only very mild powers as a reflex irritant of the upper respiratory tract, and is, in this respect, comparable to free silica. This is an unfortunate attribute, since it leads to the assumption that the dust is more or less innocuous.

The second type of cough is more intimately associated with the development of fibrosis, and occurs, or perhaps is noticed, after a varying number of years' work. Usually it is present only in the morning on getting up, when after rather a sharp attack of coughing, a little viscid sputum "like an oyster" is brought up. A similar bout may occur at night after ceasing work; at times it is sufficiently sharp to cause retching. It is generally rather worse in winter, and may be noticed only then. Although it closely resembles smokers' cough, its features are precisely the same in nonsmokers.

Persons giving a history of cough dating from an attack of pneumonia or other illness in childhood, almost invariably state that the cough has not become worse since working with asbestos. One worker succinctly described the cough as "first in the throat; later catches the chest."

Generally, this cough causes very little inconvenience and may pass unnoticed until the development of some other symptom directs attention to the general state of health. Thus, out of

sixty-six cases of fibrosis, in thirty-three (50 per cent.) the cough was stated to have preceded the onset of shortness of breath, in twelve (18.2 per cent.) to have developed contemporaneously, and in twenty-one (31.8 per cent.) was either not noticed until after the onset of shortness of breath, or, although the latter was complained of, cough was not admitted.

Complaints such as "colds go to the chest," and "frequent colds on the chest," were not uncommon. Complaint of spitting of blood was exceptional; and in all cases in which this complaint was mentioned, the exciting cause was, primarily, some disease other than the fibrosis—*e.g.*, in one, it was due to pulmonary tuberculosis; in another, it occurred only during a prolonged attack of pleurisy; and in a third, the hemorrhage was gastric in origin. Cyanosis, a valuable sign when present, rarely amounts to more than a duskiess, or slight blueness, of the lips; it contrasts, however, with the general pallor of the face with which it is often associated in these cases. Cyanosis in some degree was noted in 56 per cent. of the cases. It may be absent even when there is a considerable degree of fibrosis, or it may come and go.

Pain in the chest is rarely complained of, and then it is usually described as "tightness of the chest," "soreness," or "aching." It was noted in 10.6 per cent. of the cases.

Well-marked clubbing of the fingers was noted in a few cases. The general nutrition is hardly affected, except in the latest stages. Only ten (10.5 per cent.) were noted as being thin. In one, the nutrition was very poor; in twenty-one (22.1 per cent.), fair;

and in the remainder of the cases, good.

Radiography

Radiograms of the chest were obtained of 133 workers, or 35.5 per cent. of the number examined clinically. Of these, over 100 were taken by Dr. E. W. Twining of Manchester, and Dr. N. Tattersall of Leeds, and the remainder (except one) by Dr. F. L. Henderson of Glasgow. To all three, I am much indebted. Dr. Twining and Dr. Tattersall have devoted many hours to the joint study with the writer of the radiograms, and have drawn freely on their wide experience of radiography of the chest in furthering the purpose of the investigation.

Dr. R. S. Paterson of Manchester, too, added his experience to a final review of the films, and Dr. E. Barclay, now of Cambridge, lent his assistance in interpreting some difficult films; to both, grateful acknowledgment is made.

The standard of radiography was very high; indeed, a high standard is indispensable, if it is desired to trace the cause of such a fine and diffuse fibrosis as that produced by asbestos.

The films are superficially, but only superficially, comparable to silicosis. In the earliest negatives studied with Dr. Tattersall, a characteristic slight obscuration of the lung fields, a general lack of translucency, was noted. This appearance, for want of a better term, was denominated as "veiling." Dr. Burton Wood has independently noted (19) and confirmed this, referring to it as the "ground glass appearance." Dr. Tattersall also drew attention to the more or less rounded "whorls" of

varying size seen in the striation in the mid-zones in some of the films.

There was general agreement as to the fine and delicate nature of the characteristic mottling. Dr. Twining (in a personal communication dated Aug. 2, 1929)—while lamenting that there has been, as yet, neither time nor opportunity to study the changes from every aspect, and therefore his present views may be modified on consideration of all the evidence—states:

In general my opinion is that the earliest stages are not characteristic radiologically, but that the stage of fine dusty stippling is almost certain to be eventually provable as an early asbestos lesion. We saw it constantly in a large series of films, and after a little experience it is quite easy to detect it.

Some of the other cases showed a few grouped lesions, like rosettes or leopard markings, each component being about the size of a primary lobule. These are similar to lesions sometimes seen in tuberculosis. In the asbestos cases I regard them as being groups of primary lobules making up a lobule, the walls of which are infiltrated. They certainly seem to correspond in size with the macroscopic lesions seen in the pathologic specimen.

The advanced cases show heavy basal and mid-field mottlings, common in pneumoconiosis, with a tendency to avoid the apices.

On the whole the lesions are distinctly less dense than those of silicosis, and are less easy to group into well-defined stages, but I think we can recognise:

1. A very doubtful stage of increased linear striations.
2. Fairly definite fine dusty stippled appearance.
3. Coarser mottling with increased linear striations.
4. Gross lesions with pleural changes and displacements due to the pull of the fibrosing lesions.

The few tuberculous lesions we have come across have been easily distinguishable.

The radiographic appearances in the earlier stages are most marked on the right side at the base or in the central zone. This corresponds with the clinical findings. This preference for the right side has been noted also in silicosis, by the South African observers. The cases showing radiographic signs of a dust fibrosis have been classified in three broad groups. This grouping, although unscientific, is convenient practically, considering the main purpose of this investigation. Indeed more precise classification based on the particular radiographic changes noted might well be misleading at the present time.

Much combined clinical, radiologic, and pathologic study is required into the whole subject of these fine types of dust fibrosis, which have been noted in workers exposed not only to silicate dusts other than asbestos, but also to other inorganic dusts containing no silica, before it will be possible to classify the radiologic changes as has been done so successfully by the South African workers in respect to silicotic fibrosis.

The hypothesis that, because asbestos dust produces a pulmonary fibrosis with consequent deviations from the normal in the lung skiagram, the degree and potentialities of this fibrosis can be assessed by comparison of its radiologic picture with those of standard silicotic films, is untenable.

At least two general types of pulmonary fibrosis caused by inorganic dusts can be recognized. A third, representing the purely peribronchial variety of fibrosis, might be added; or it may be that all dust fibrosis will be found to approximate more or less closely one or the other of these two

types. These types are (1) that produced by combined silica dust, an example of which is seen in asbestos fibrosis, and (2) that produced by free silica dust, represented by silicotic fibrosis.

These two types, while resembling one another in clinical signs and symptoms, differ materially both in the nature of the lesion produced in the lung, and in the character of the associated radiographic picture. Thus, attempts to weigh, consciously or unconsciously, asbestos fibrosis, or any dust fibrosis other than silicosis, by means of the standard radiographic changes found in silicosis, is unsound and is likely to lead to a misconception of the potentialities of the dust in question.

Badham (8) gives an example of this source of error in reporting the unexpectedly early death of a man affected with a fine fibrosis caused by an orthoclase basalt containing no free silica. Referring to some of the radiologic differences between the fine fibrosis of dusts other than silica and the nodular fibrosis of quartz dust (*i.e.*, true silicosis), he states:

Moreover, the coarse fibrosis of silica gave clear interspaces of normal lung, while the fine fibrosis presented a uniform granular mottling leading to the conclusion which is probably erroneous that the actual development of fibrous tissue was greater in a nodular fibrosis as silicosis than in a generalised fine fibrosis caused by silicates. To me it appears that the mechanical damage to the lung is greater in a fine fibrosis than in a coarse fibrosis when both are well developed.

The evidence obtained in the present inquiry amply confirms this general statement.

On studying the development of asbestos fibrosis as displayed in a series of radiograms, and especially in the few available taken a year or more before a fatal termination, one cannot help asking the question, "What is there here which could have this effect?" The answer is that the damage to the lung is much greater than it appears to be when judged by the silicotic standard. Asbestos fibrosis is much more diffuse; it spins its fine web, as it were, crisscross throughout the lung, enveloping and eventually strangling the ultimate lobular structure, rather than depositing itself in numerous more or less isolated foci, as in silicosis. Thus, at any rate in the less advanced stages, the radiologic picture of the lesions does not impress the eye, unconsciously viewing it from the standpoint of silicosis.

The radiographic picture may show soft and fairly coarse nodulation, but it is never so impressive as the nodulation in a silicotic film.

Paradoxically, the distinctive feature, both clinically and radiologically, of the asbestos fibrosis is its uniformity. The modesty of the symptoms; the unobtrusive, but diffuse, impairment of the percussion note; the homogeneous stippling of the skiagram; all are fragments of an entity, unmistakable when assembled, but enigmatic when divorced.

Only two references to the radiographic appearances of the chest in asbestos workers have been traced: one in a report by Pancoast and Pendergrass (10), and the other in an article by Burton Wood (19).

Pancoast and Pendergrass together with Miller and Landis examined "17 asbestos workers, 2 of whom showed

first stage changes and the other 15 definite second stage appearances. Of the men longest at work, one after seventeen years' occupation showed very definite diffuse, 'soft' spots throughout both lungs, and another showed about the same appearance after fourteen years' occupation. Very slight nodular shadows were found in one man after only two years' occupation. Most of these second stage cases showed also well-marked first stage appearances still present, indicating a persistence of free drainage hilumward. In all instances the nodular shadows were characteristically 'soft' and varied considerably in size." Their first stage appears to correspond with stage 1, and their second stage with stages 2 and 3 mentioned above.

It appears from the context that these observers regard asbestos fibrosis as being really a silicosis due to admixture of free silica derived from the original rock—a view difficult to substantiate.

Burton Wood (19) in a series of fifteen skiagrams of asbestos workers notes: "The most noticeable feature of skiagrams of workers exposed longest to asbestos dust is the presence of shadows suggesting a diffuse fibrosis affecting chiefly the lower two-thirds of the lungs. The fine quality of the shadows is worthy of note. Some of the cases exhibit a 'ground glass' appearance, though on close inspection fine mottling is evident. . . . when more definite mottling is present it lacks the coarse quality described in the skiagrams of chests showing pneumoconiosis, e.g. South African gold miners. . . ."

Clearly, the maturation of asbestos fibrosis is spread over a period of years,

and by the time the stage is reached when the features discussed above are, in varying degree, positive, and the radiologic picture is recognizable, the fibrosis is not in its inception, nor even in its earliest stages, but is developed and fairly widespread.

We must, therefore, recognize an earlier state when the fibrosis is present in slight degree, and also when there is evidence of choking of the lymphatics, and of a measure of pulmonary catarrh. This stage may be referred to conveniently as the prefibrotic stage.

The indications of this stage are indefinite, and some, at any rate, not specific. If, however, they can be determined and applied with only a moderate degree of accuracy, information of practical value will be available. Efforts have been made, therefore, to distinguish workers in this stage. The following tabulation shows that twenty-one workers out of 363 examined (5.8 per cent.) were so classified:

<i>Clinical Examinations</i>			<i>Per</i>
(363):		<i>No.</i>	<i>Cent.</i>
Fibrosis.....	95	28.2	
Prefibrotic conditions.....	21	5.8	
<i>Radiologic Examinations</i>			
(153):			
Fibrosis.....	52	39.1	
Suggestive changes not definitely diffuse fi- brosis.....	22	16.5	

Many of these cases present a slight diffuse impairment of percussion note—perhaps better described as a slightly increased sense of resistance felt on percussion, mostly of the right lung, at least as contrasted with the left, and associated with some weakening of the respiratory murmur. Probably this early stage could be detected radiographically, but only by means of

comparison with a radiogram taken prior to commencing work with asbestos. By such means the earliest stages of silicosis have been worked out by the South African observers. There, the radiograms taken at periodic six-monthly medical examinations can always be compared with initial radiograms taken before the employees are permitted to work underground.

group is dismissed from any further consideration.

A general summary of the findings in the 374 workers who were examined clinically, classified under the most important lesion, is presented in Table 4.

ILLUSTRATIVE CASES

The salient points of a few cases are set out below to illustrate clinical and

TABLE 4.—GENERAL SUMMARY OF FINDINGS CLASSIFIED UNDER THE MOST IMPORTANT LESION

YRS. EMPLOYED	NO. EXAMINED	CASES OF FIBROSIS		PRE-FIBROTIC CONDITIONS		PULMONARY TUBERCULOSIS			CASES OF OLD HILAR TUBERCULOSIS, OR SUGGESTING ENLARGEMENT OR CONGESTION OF LUNG ROOTS	PULMONARY AND BRONCHIAL CATARRH, AND BRONCHITIS	OTHER PULMONARY LESIONS			WITHIN NORMAL LIMITS
		Due to Asbestos	Due to Other Causes	Due to Asbestos	Due to Other Causes	With Evidence of a Dust Fibrosis	Other Active Lesions	Other Inactive Lesions			Pleurisy ¹	Emphysema ²	Other Lesions	
0-4.....	92 0	3	5	0	0	0	0	0	13	7	1	0	1	62
5-9.....	142 36	1	12	0	1	1	1	4	3	3	5	2	2	72
10-14.....	89 27	5	3	0	3	0	3	3	5	0	3	2	0	38
15-19.....	30 15	1	1	1	0	0	1	1	0	2	1	1	0	7
20 and over....	21 17	0	0	0	1	0	0	0	0	1	0	1	0	1
Total.....	374 95	10	21	1	5	1	8	21	21	13	10	6	3	180
Percentage...	100 25.4	2.7	5.6	0.3	1.3	0.3	2.1	5.6	5.6	3.5	2.7	1.6	0.8	48.1

¹ One case of thickened pleura due to old gunshot wound.

² One case of emphysema due to gassing.

In other cases included in this group, diffuse weakening of the respiratory murmur was noted, with fine sticky râles in the root areas. At present no stress can be laid upon this group. The clinical changes are so slightly marked that until comparative radiograms are available it would be unsafe to draw any deductions. For the purposes of this inquiry, therefore, this

other features. Five radiograms are reproduced to illustrate the radiologic appearances of some of the cases. There are insuperable difficulties, however, in reproducing the finer changes depicted in the original negatives.

CASE 1.—This worker, a female, aged 24, has been employed in asbestos for ten and one-half years—as a card tenter one year,

and as a doubler nine and one-half years. Her family and personal medical history contain nothing of note. She has no complaints and feels quite well. Her nutrition is good. She is pale, but shows no cyanosis.

Chest.—She has a chest expansion of 1½ inches. No impairment of the percussion note is detected. The breath sounds in the right lung are weaker, generally, than those in the left. Expiration is prolonged at the right root; no added sounds are heard. A skiagram showed some very slight and doubtful changes in the direction of increased striation.

CASE 2.—This man, aged 24, has been employed in asbestos for five years, carding and mixing. Previously he worked for four years in the cotton trade, yarn weighing. His family and personal medical history contain nothing of note. He has had a slight morning cough and expectoration for a year. His nutrition is good. His color is fresh, and there is no cyanosis.

Chest.—There is some impairment of the percussion note over the middle third of the right lung, behind, and slightly at the right base. The breath sounds are harsh, and expiration is prolonged in the upper half of the right lung, behind; expiration is also prolonged over the left upper lobe, behind. The respiratory murmur is weakened at the right base, and the breath sounds are of a "whiffing" character. There are no added sounds. A skiagram showed enlargement of the right root, and slight haziness and striation in the central zones in both lungs, suggesting early fibrosis.

CASE 3.—This man, aged 32, has been employed in asbestos for six years in the card room, and as a stripper and grinder. Previously he was employed in a cotton card room for nine years. He had army service for five years, but was not gassed. He gives a family history of asthma; otherwise there is nothing of note in his family or personal medical history. He has no complaints and feels quite well. His musculature is very good. His color is pale, but he shows no cyanosis.

Chest.—There is slight impairment of the percussion note generally, particularly at the right base. The respiratory murmur is a little weakened generally. No added sounds are detected. A skiagram showed

old tuberculosis of the lung roots and general increased striation, especially in the lower half of the right lung, suggesting early fibrosis.

CASE 4.—This worker, a male, aged 23, has been employed in asbestos for nine years, mostly mattress making. His family and personal medical history contain nothing of note. He complains of occasional pain in both sides of the chest, and perhaps a little undue shortness of breath on exer-



FIG. 1.—Case 4: Moderate fibrosis. Undue degree of striation and fine mottling in central zones of both lungs; calcified glands in roots, with rather coarse striation in upper lobes; ? interlobar pleurisy on right side, between the lower and middle lobes; emphysema at bases.

tion. His color is normal. His musculature and nutrition are good.

Chest.—There is no retraction of the apexes, but there is a slight flattening below the right clavicle. The percussion note is slightly impaired generally, behind, particularly on the right side. The breath sounds are weak, generally, and expiration is prolonged. No added sounds are detected. The skiagram (Fig. 1) shows an undue degree of striation and fine mottling in the central zones of both lungs; calcified glands in the roots, with rather coarse striation in

the upper lobes; ? interlobar pleurisy on the right side, between the lower and middle lobes; and emphysema at the bases. The case was diagnosed as moderate fibrosis.

CASE 5.—This woman, aged 40, has been employed for eleven years in asbestos, for most of the period mattress making. Previously she was employed in a laundry. She has recently been ill for three months with pleurisy (no history of tapping); otherwise her family and personal medical history

moderate fibrosis, most marked toward the bases.

CASE 6.—This worker, a female, aged 31, has been employed in asbestos for seven years, opening, and has been exposed to much dust. Her previous employment was non-dusty. Her family and personal history disclose nothing of note. She complains of having had a cough for five years, and of shortness of breath on hurrying. She is thin and undernourished, and pale.

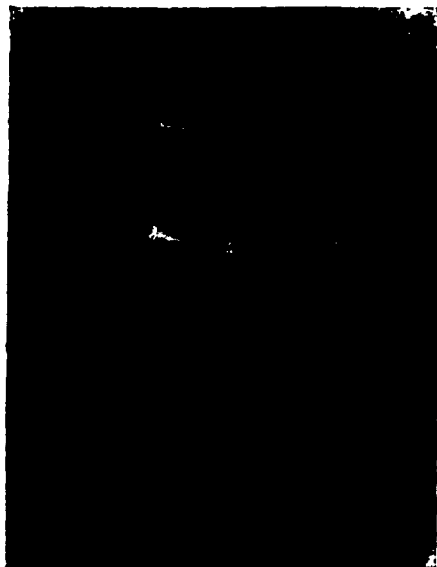


FIG. 2.—Case 6: Moderate, but fully developed, fibrosis. Enlarged glands in both roots; diffuse fine mottling in both lungs, especially the right.

are negative. She complains of having had a winter cough for three or four years, and of shortness of breath on exertion since her attack of pleurisy. Her nutrition is good; her weight is stationary. She has normal color, and no cyanosis.

Chest.—There is some general impairment of the percussion note over both lungs, behind, more noticeable at the bases. The respiratory murmur is weakened generally. Persistent crepitations and medium râles are noted low down in the right axilla. A skiagram showed diffuse fine striation and fine nodular mottling, with light opacity at the bases. The case was diagnosed as



FIG. 3.—Case 7: General fibrosis, mostly linear, but with little mottling of right lung. Old puerile tuberculous scars at apexes.

Dust is present on her hair and in her nostrils.

Chest.—She has a chest expansion of one-fourth inch. There is slight general impairment of the percussion note, definite at the apexes and the bases. The breath sounds are weak; expiration is slightly prolonged. No added sounds are heard. Her heart is not enlarged, and no murmurs are detected. The skiagram (Fig. 2) shows a number of enlarged glands in both roots, and a diffuse fine mottling in both lungs, especially the right. This is a case of moderate, but fully developed, fibrosis.

CASE 7.—This woman, aged 48, has been

employed in asbestos for thirty-two years—about three years on cards, and the remainder as a spinner. She has done no other factory work. Her family medical history discloses nothing of note. She has a personal history of nephritis seven or eight years ago, causing three months' illness. Her complaints are: shortness of breath on hills, and winter cough for about six years. Her nutrition is fair. She is rather pale.

Chest.—She has a chest expansion of one-half inch. No retraction of the apexes is noted. There is general slight impairment of the percussion note, behind. Breath sounds are harsh and expiration is prolonged. No added sounds are heard. No gross enlargement of the heart is noted, but the second sound is found to be accentuated over the aortic area. The skiagram (Fig. 3) shows a general fibrosis, mostly linear, but with a little mottling of the right lung. There are also very old puerile tuberculous scars at the apexes. The case was diagnosed as general fibrosis.

This case should be compared with Case 6. In Case 6 there was a heavy exposure to dust extending over a few years; in the present case there was exposure to a very much less concentration of dust, except possibly in the first three years, but extending over many years.

CASE 8.—This worker, a male, aged 62, has been employed in asbestos for twenty years as a weaver. He was previously a cotton and silk weaver. His family and personal medical history show nothing of note, except that he was regarded as a delicate child. His complaints are: shortness of breath on exertion, and on going upstairs, for three years; morning cough and a little expectoration for the last three winters. He is thin and pale, with some cyanosis.

Chest.—Chest expansion is 1 inch; there is retraction of the apexes. The percussion note is impaired over both upper lobes, in front, and over the upper two-thirds of the right lung, behind. Expiration is prolonged at the bases. Pleural rub and inconstant râles are noted at the left base, and a few râles at the right base. A skiagram showed a definite diffuse fibrosis with nodulation. This is a case of fibrosis in the early advanced stage.

CASE 9.—This man, aged 41, has been

employed for twenty years in asbestos in many capacities. There is nothing of note in his family history. He gives a history of pneumonia a number of years ago. His complaints are: a little shortness of breath on exertion for the last two or three years; otherwise none. His nutrition is fair only. His color is fresh, but there is slight cyanosis of the lips, at times.

Chest.—The skin is poorly elastic; there is some retraction of the apexes. There is an impaired, rather "boxy," note on percussion, generally, especially over the upper half of the left lung, and over the upper two-thirds of the right lung, behind, and in front. Respiratory murmur is generally weak. Expiration is slightly prolonged, but diminishing toward the bases. Pleural crepitations are noted low down in the right axilla. A skiagram showed a definite fine diffuse fibrosis. Neither lung lights up very well, and throughout both there is a very fine diffuse reticulation and mottling—moderate, but fully developed, fibrosis. The fine diffuse character of the radiologic appearances is particularly noticeable here.

CASE 10.—This man, aged 46, has been employed for twenty-five years in asbestos, during nine years of which he was not exposed to dust. For eleven years of the remainder he was employed in the card room, and mattress making. His family and personal medical history disclose nothing of note. His complaints are: shortness of breath on exertion for three months; cough after a few hours' work in dust during the last two or three years, with little or no expectoration; and pain in the left side of the chest for the last three months. His nutrition is good. He is pale, with slight duskeness of the lips.

Chest.—There is some retraction of the apexes. The percussion note generally is impaired, and of a "boxy" character. Breath sounds are harsh and expiration is prolonged over the right upper lobe; elsewhere the respiratory murmur is a little weakened and expiration is not materially prolonged. No added sounds are heard. The heart is normal. The skiagram (Fig. 4) shows diffuse striation and fine speckled mottling throughout both lungs—fibrosis, fully developed.

CASE 11.—This worker, a male, aged 40,

has been employed for twenty-two years in asbestos—during the first eleven years on cards and as a weaver, during which he was exposed to a considerable concentration of dust. During the remainder of the time he was employed on a non-dusty process, but was exposed to a less concentration of dust derived from other processes. His family and personal medical history give nothing of note. His complaints are: shortness of breath on climbing stairs for the last five years; slight morning cough and expectora-



FIG. 4.—Case 10: Fully developed fibrosis. Diffuse striation and fine speckled mottling throughout both lungs.

tion for about the same time; and slight aching of the chest in front, below the left clavicle. His nutrition is good. There is slight cyanosis of the face.

Chest.—Chest expansion is 1 inch; there is retraction of the apexes. There is impairment of the percussion note generally, most evident over the whole of the right lung, behind, and over the right upper lobe in front. The note is "boxy" over the remainder of the chest. The breath sounds are slightly weakened generally, except at the right apex, but there is no gross change. Expiration is prolonged generally. Fine crepitations are heard in both axillae, and

over the right upper lobe, in front. The heart is normal. Sputum is mucoid, but is negative for *Bacillus tuberculosis*. Clubbing of the fingers has been noted for about two years. A skiagram showed an extensive diffuse fibrosis—fibrosis in a fairly advanced stage.

CASE 12.—This worker, a male, aged 60, has been employed in asbestos for twenty-six years in the card room. Previously he was a sawyer's laborer. There is nothing of note in his family and personal medical history. He complains of slight shortness of breath on exertion of recent years, and of winter cough for the last three or four years; expectoration is stated to be nil. His musculature and nutrition are fair. There is slight cyanosis of the face.

Chest.—He has a chest expansion of three-fourths inch. The percussion note is impaired generally, especially over the left upper lobe in front. The respiratory murmur is weakened over the whole of the chest except the left upper lobe, where there are whistling breath sounds, and medium râles. The heart is normal. A skiagram showed very extensive changes throughout the chest of a coarse type without fine stippling. The roots showed a choked appearance. There was heavy mottling at the base. One old tuberculosis focus was noted at the left base, and some calcified glands in the left hilum. The diagnosis was fibrosis in the advanced stage.

CASE 13.—This man, aged 66, has been employed in asbestos for twenty-one years as a weaver. For about sixteen years previously he was a cotton and silk weaver. His father died of "chest trouble" at the age of 45; his sister died aged 15 of pulmonary tuberculosis. He has only been away sick a week or two in twenty-one years, and has never had any serious illness. His complaints are: undue shortness of breath on exertion during the last two years; cough for the last two or three winters. Expectoration is creamy, and occurs a fair amount of times. He has lost weight, and is thin. He is pale, but with some cyanosis of the face, and with a malar flush.

Chest.—Chest expansion is $1\frac{1}{2}$ inches; there is retraction of both apexes. Dulness is detected on percussion over the upper two-thirds of the right lung and over the left

upper lobe, behind, and over both upper lobes in front. Breath sounds are rather weak, with prolonged expiration. Scattered variable rhonchi and râles are heard over the right lung, behind, over the left upper lobe in front, and over the left root. The skiagram showed a definite diffuse fibrosis plus tuberculosis. Sputum was negative (one examination). The diagnosis was dust fibrosis in the advanced stage, with tuberculosis, probably active.

CASE 14.—This worker, a male, aged 36, commenced work in an asbestos plant in 1910, but was employed on other work until January, 1914. During the next five years, until early in 1919, about three years and nine months were spent in army service, for nine months of which he was a prisoner of war, but he had two short periods of work in asbestos—eleven months in the weaving, and six months in the mattress, departments. From early in 1919 to November, 1927, he worked in asbestos processes, but for only six years and ten months in a dusty one (mattress making). He then ceased work. He was referred to a tuberculosis dispensary on Oct. 19, 1927, when he gave a history of bronchitis in 1916 and 1917 during the War, and of never being very well since; complaining of dyspnea, pain in the right side, and a slight cough of only about six months' duration, with a small amount of grayish sputum. Dr. N. Tattersall states that at that time:

"Dyspnoea was very marked, even on talking, and there was definite cyanosis. He had marked hollowing and respiratory retraction at both apices, especially the right; loss of note over most of the right lung and the upper half of the left; bronchial breath sounds at both apices; fairly numerous crepitations, mostly on the right side, and a pleural rub at the right base. He had a systolic bruit at the apex and pleuro-pericardial friction.

"I examined him a number of times, the last occasion being April 20, 1928. In that time he had lost 9 pounds, felt very weak, and the dyspnoea was increasing. The signs remained much the same, though occasionally, if he got a cold, the moist sounds were more numerous, especially at the bases. Seven sputum examinations were negative, and up to the time I last saw him

the sputum had remained small in amount, and its appearance did not suggest tubercle.

"My diagnosis from the beginning was one of pulmonary asbestosis with possibly added tubercle.

"I took two X-rays of him, neither of which showed appearances suggestive of tubercle, everything pointing to a very extreme degree of fibrosis."

He died on Oct. 13, 1928. No postmortem examination was obtained.



FIG. 5.—Case 14: Massive fibrosis, with pleural thickening, retracted apices, and pneumothorax on left side with incomplete collapse of lung.

It will be noted that the length of exposure to asbestos dust is unlikely to have been more than about ten years and two months, and of this time he was employed only about eight and one-half years in a dusty process. War service, of course, may have reduced his resistance. The skiagram (Fig. 5) shows a massive fibrosis with pleural thickening, retracted apices, and pneumothorax on the left side with incomplete collapse of the lung.

No doubt fibrosis of the type produced by asbestos dust can of itself lead to complete disablement, and

finally to a fatal termination, even in the absence of a superadded tuberculous infection.

The primary effect of the fibrosis would appear to be that of causing defective aeration of the blood, resulting in an added strain on the heart. For many years, and even with an advanced degree of fibrosis this may be of little inconvenience, provided physical exertion is limited, and acute

illnesses are avoided. Ultimately, however, the margin of safety, already diminished, is lost and the circulation slowly fails, with the usual signs of edema of the lungs. More often, however, an attack of bronchopneumonia, influenza, or other acute infection adds too much to the strain, impairs the cardiac musculature, and results in a fatal termination.

(To be concluded)

BOOK REVIEWS

BLEIVERGIFTUNG. By Prof. Dr. Paul Schmidt, Direktor des Hygienischen Instituts der Universität Halle a. d. S.; Priv.-Doz. Dr. Adolf Seiser, Oberassistent am Hygienischen Institut Halle a. d. S.; und Priv.-Doz. Dr. Stillfried Litzner, Assistent an der Medizinischen Klinik Halle a. d. S. Paper. Pp. 79 with index, illustrations, and bibliography. Berlin and Vienna: Urban & Schwarzenberg, 1930.

This monograph is divided into a short introductory or general section and a special section. The first part is devoted principally to a discussion of the outstanding diagnostic symptoms of lead poisoning and of the work done by Dr. Schmidt and his collaborators in the microchemical determination of lead in blood, urine, and feces. The second part discusses in detail the usual sources of lead poisoning; the absorption, distribution, and excretion of lead; the pathology and clinical findings associated with lead poisoning; and, finally, the diagnosis, prophylaxis, and therapeutic treatment of lead poisoning.

Dr. Schmidt's monograph is particu-

larly apposite. It brings the literature of the subject down to the present date and represents the mature thought of an investigator who has long made this field particularly his own. The discussion of the diagnostic features of lead poisoning is especially clear and to the point. In this connection it is of interest to note that emphasis is placed on the importance of the determination of lead in the blood.

No attempt is made to discuss the details of technic. The monograph presents instead a critical review of the more recent experimental work relating to lead poisoning and will, in consequence, doubtless have a wide appeal to all investigators in this field. The bibliography, which contains 298 references to the recent work and brings the literature of the subject down to 1930, is particularly valuable. "Bleivergiftung" may well be recommended as a valuable addition to the library of students of public health.—
L. T. Fairhall.

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PERSONAL QUALITIES IN ACCIDENT CAUSATION*

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THE main purpose of this article is to give some account of the practical and theoretical difficulties encountered in a research into the personal qualities involved in accident causation. This problem is exceedingly important and is, in a sense, one of the main problems of psychology since its solution demands a stringent research into mental integration. The occurrence of an accident, as I shall show later, is not necessarily fortuitous but is often the result of the complicated interaction of various psychologic and physiologic functions; and a research which has as its object the disentanglement of such complex mental situations (using mental in its widest sense) is bound to throw light on the nature of mental life as a whole. The accident situation is one in which everybody finds himself sooner or later, and to which he reacts either by incurring an accident

or by avoiding one. If we can find out why some people react in the one way and some in the other, we shall have learned a good deal about the nature of the reactions of mind to its environment.

About the practical importance of this problem to industry there can be little doubt. The cost of accidents is enormous not only in actual money but also in loss of time and efficiency. It is difficult to gauge the full extent of this latter loss, but the financial cost of accidents, as represented by the annual expenditure in insurance premiums, runs into hundreds of thousands of pounds. This insurance, of course, only covers cases where compensation has to be paid, and compensated accidents are actually only 1 or 2 per cent. of the total number of industrial accidents.

An accident is commonly regarded as being due to chance: that is, as an event the causes of which are external to the sufferer and not affected in any

* Received for publication March 26, 1930.

way by the nature of the sufferer himself. If this were so, one person would be just as likely to sustain an accident as would another in circumstances of equal risk; and if records were taken for a large enough group of workers over a sufficient period of equal exposure to risk, it would be found that the accidents sustained during that period were equally distributed among the people concerned. In 1919 Greenwood and Woods (1) showed that the curve of the frequency distribution of accidents is not a straight line or a normal, saddleback curve, but is a curve shaped rather like the letter J. This was confirmed by Newbold (3) in 1926. Broadly interpreted, this fact means that some people have more and some fewer accidents than can be accounted for by the operation of chance alone—a fact of great importance from a psychologic standpoint since it definitely postulates the existence of a liability to have accidents which is more or less constant for each person but varies from one person to another. In other words, accidents are due not solely to chance but also to some personal quality which we may call accident proneness. This fact alone gives a *prima facie* warrant for a study of individual psychologic differences; it was the basis of the subsequent practical work done by the Industrial Health Research Board¹ on the causation of accidents.

Another fact of practical importance which may be deduced from the J-shaped distribution curve of accidents is that approximately 70 per cent. of the accidents sustained by a group of

workers are actually sustained by only 25 per cent. of the workers; therefore, by weeding out a quarter of the workers a very considerable reduction in accident incidence would be effected.

All the accident records which have yet been examined yield the J-shaped distribution curve, and all agree with the theory that the individuals in the groups concerned differ in their proneness to accidents. Newbold (3) has shown that the frequency distribution curve for foreign bodies in the eye—a particular type of accident—is likewise J-shaped, so that even in this apparently unlikely instance the theory of differing personal proneness is confirmed.

TYPES OF ACCIDENTS

Accidents may be divided roughly into three categories. First there are those which are purely accidental in the popular sense of the word; in the second class are accidents which depend on the instrumentality of other people and not on the sufferer himself; and third there are the accidents which depend directly on one's individual proneness. The first of these divisions comprises what insurance policies call "Acts of God," which consist essentially in such injuries to the person as are the inevitable results of nonhuman environmental changes. The second class of accidents is similar to the first from the point of view of the sufferer. Again environmental change results in inevitable injury to him, but in this case the change is produced by human agency. Finally, there is the class which embraces accidents that are the result of environmental changes either produced by the sufferer himself or of such

¹ Throughout the text the Board is referred to uniformly under its new name; in the bibliography the name used is that given on the cover of the report.

a nature as to be avoidable by certain reactions on his part.

The point of this classification is that the first and second categories are marked by the inevitability of the accident: the sufferer cannot escape the accident whether the causes are human or nonhuman—he may be struck by lightning or he may be shot by a careless sportsman. Any accidents which are directly avoidable properly fall into the third category, and it is only accidents in this class which can be regarded as due to personal proneness. As yet there is no evidence to show what proportion of accidents fall into each of these classes, but the universality of the J-shaped distribution curve shows definitely that the majority of industrial accidents belong to the third category: that is, that they are due to personal proneness. There is, of course, a certain amount of overlap between these classes. For example, the activity of an accident-prone worker may produce an accident which is sustained not by himself but by another worker near him. Really such an accident should count as a third-category accident to the first worker; actually in the records it would figure as a second-category accident to the worker who sustained it.

It will be seen at the outset that any research into accident causation is handicapped by uncertainty as to the type of accidents sustained by any group of workers. It is also obvious that no investigation can lead to changes in practice which would completely eliminate accidents, since accidents in the first and second categories are not due to personal differences.

The foregoing remarks suggest a definition for the accidents which are due to personal proneness. Such an accident may be defined as a physical injury due to a faulty reaction on the part of the sufferer to some environmental change. The causes of this faulty reaction lie in the sufferer himself, and it is the attempt to discover the nature of these causes which constitutes the research which Mr. Eric Farmer and I are making in this field (4) (5).

DIFFICULTIES IN PRACTICAL RESEARCH

So far, for the sake of convenience in argument, accident proneness has been alluded to as a personal *quality*, but it is by no means necessarily the case that such proneness is a *single* quality. A study of the nature of accidents themselves suggests, on the contrary, that this proneness is complex in nature and the resultant of a number of psychologic and physiologic functions. For example, speed of movement, muscular control, and general intelligence, all seem to play a part in the avoidance of such accidents as cut or crushed fingers and burns. Further, there is nothing to indicate that these functions are combined in certain definite proportions to produce a new, integral function which we have called accident proneness; rather it would seem that in some cases one function predominates, and in other cases another. Hence, proneness may be regarded as a group of co-operating functions and not as a definite unitary function, and this in its turn forms a further handicap to practical research in this field. Not only the type of

accident may vary, but also the causes of the same type of accident may vary.

Another difficulty arises immediately we begin to consider the measurement of accident proneness, for it is evident that proneness and number of accidents sustained are not synonymous. We cannot say that a person with a high degree of accident proneness will have many accidents and that another person with a low degree of proneness will have few accidents. Such a statement would leave entirely out of account all accidents falling into the first two categories and also inequalities of exposure to risk. All we can definitely say is that in a group of people equally exposed to risk but of varying accident proneness, those with the greater proneness to accidents will tend to sustain a larger number of accidents than those with a lesser proneness. Of course, the longer the period over which accidents are recorded, the nearer does the actual accident rate approximate a true measure of accident proneness; therefore, the accident rate of the whole working life would be the closest measure of accident proneness that is possible.

Some of the practical difficulties connected with the collection of accident records may be mentioned here. These records are not always kept in a way which makes analysis easy. This is to be expected since the records are usually kept to serve practical purposes and not as a basis for statistical inquiry. In most cases a bare statement of the occurrence of an accident to a particular person on a particular date is all that is recorded. The nature of the accident is sometimes given, but its gravity is rarely recorded. Moreover it is sometimes difficult to

distinguish between redressings of the same wound and further accidents. Apart from this there is rarely any guarantee that *all* accidents are reported to the surgical stations. Some factories have first aid boxes in the workshops. Unless there is very strict supervision and a high standard of reporting, workers are likely to bandage small cuts and the like for themselves without troubling to report them, unless, as frequently happens, they are compelled to report with a septic wound a few days later. Furthermore, considerable difficulty is often experienced in getting details regarding the length of time that workers have been exposed to risk. Short time and holidays, as a rule, affect all the members of a homogeneous group equally; but records of lost time through sickness and the dates when workers are taken on and discharged are quite often not available. The discovery of the age and experience of the workers is sometimes a matter of considerable trouble; and finally, even if all accidents have been reported, it is not always an easy matter to find out exactly how many workers have had no accidents at all.

Many of these difficulties can be obviated, however, by a careful choice of the groups of workers to be examined, and statistical analysis has shown that some of the other difficulties are unimportant. For example, major and minor accidents have been found to correlate fairly highly in some cases (though this is a point which needs confirmation), so that if a record of either alone is available it will serve as an indication of the varying accident proneness of the workers.

At this point the foregoing remarks

may be summarized into a statement of our position at the beginning of a practical research into the nature of accident proneness. We have, as the sole criterion of our success in discovering the functions involved in proneness, a set of accident records which may have been inaccurately kept, which will include an unknown percentage of accidents not due to personal proneness, and which will have an unknown correlation with accident proneness itself. We have then to devise a series of psychologic tests, the ability to perform which must be related as closely as possible to accident incidence. When this has been done, if we succeed in finding a test for every psychologic and physiologic function that plays a part in accident proneness, and if further we combine these test scores, each correctly weighted, to form a single combined score which shall have the closest possible relationship to accident proneness, even then we can never hope to get anything approaching perfect correlation between the combined score and accident incidence. The reasons for this have already been pointed out: The accidents recorded do not themselves correlate perfectly with proneness, the records include an unknown proportion of accidents of the inevitable type, and the functions tested probably are not involved to the same degree in the individual's reactions to different accident situations.

RELATIONSHIP BETWEEN TESTS, ACCIDENT INCIDENCE, AND INDUSTRIAL PROFICIENCY

We now come to a consideration of the actual tests to be applied in an

attempt to elucidate this problem. Psychologic tests may be grouped under the two heads of general and specific. Now it has not yet been shown that there is a specific test or tests for accident proneness. The sustaining of an accident may be due to so many different causes that it is impossible to devise an analogous test for accidents in general in the same way that a test may be devised for proficiency in a particular repetitive task. In other words, although the individual has a certain proneness to accidents, it may be that he has no special ability for their avoidance. Hence, until the existence of a specific factor or factors in accident proneness has been established, we can only apply tests of a general nature. These tests may be for functions at any mental level from pure reflex to ideation; and if we consider the nature of the various types of accidents which occur, we shall find no reason for assuming that the functions involved in accident proneness all lie at some definite mental level. Rather, the reverse is true. In some cases the accident may be due to failure or slowness in making the simplest muscular reaction, in some to poor coordination of neuromuscular coordination of a more complex type, and in others to sheer lack of intelligence. Hence it is necessary to use a number of tests which together cover as large a mental field as possible.

The ideal battery of tests would include one for each function involved in accident proneness. The actual relation of each test to accident incidence could then be found and all the tests weighted together in their correct proportions to yield a single score

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which would have the highest possible correlation with accident incidence. Theoretically, as we have already seen, the correlation between this ideal group of tests and accident proneness would still fall short of unity to a degree which we have no means of measuring. For practical purposes it is of course desirable to limit the actual number of tests used to as few as possible. Such tests should not correlate with one another, since high intercorrelation in this case would mean unnecessary overlapping.

It should perhaps be said here that the foregoing depends on the assumption that all the functions involved in accident proneness are directly measurable. However, there is no reason to assume that psychologic functions which cannot be measured at our present stage of knowledge do not also play their part in accident causation. Temperamental differences may be cited as an example of such nonmeasurable functions. Here we have yet another factor militating against success in measuring accident proneness, since we have no means of scoring nonmeasurable functions and combining such scores with those of functions which are directly measurable.

So far only the attempt to measure accident proneness with the practical object of reducing industrial accident incidence has been discussed. The avoidance of a large accident rate is, however, only one of the problems arising in industry. One of the most important of the other problems is how to obtain the maximum efficiency from the workers, and it will be seen at once that, as a matter of practical politics, no employer can be expected to choose operatives with a low degree of acci-

dent proneness if by so doing he gets less efficient workers. This suggests a further aspect of the research into accident proneness—namely: What is the connection between accident incidence and industrial proficiency? To answer this question it is necessary to discover the relation between the tests used for accident proneness and such records of industrial proficiency as are available. Theoretically it would seem likely that there is a fairly high connection between a high accident proneness and a low standard of efficiency, for the accident situation is also a situation occurring in the performance of an industrial task and a reaction to this situation which results in an accident cannot at the same time yield an efficient performance of that task. However, the question can by no means be fully answered in this way since the number of accidents sustained by even the most highly accident-prone worker is very small in comparison with the number of tasks he performs unscathed. The only way to solve this problem satisfactorily is to see if the tests which correlate with accident rate also correlate with industrial proficiency.

In face of all the difficulties which beset an investigation of this sort, it is evident that the greatest care in technic and accuracy in analysis must be exercised if the results are to have any real meaning and value. It is not, as a rule, possible in the biologic sciences to keep all factors constant except the one we desire to measure. Therefore, it is necessary to examine the results obtained from several different angles in order to make sure that we are actually measuring what we set out to measure. To apply

certain tests to a group of workers and correlate the scores obtained with the accident records of the subjects is by no means sufficient to yield a reliable answer to the problem. High correlation coefficients may in isolated instances arise quite by chance, and even if they are not spurious they may be due to a relation quite different from that which they appear to measure on first sight. This can be guarded against only by repeating the same tests on as many different groups of subjects as possible and by subjecting all the results obtained to statistical analysis by such methods as that of partial correlation. Correlation coefficients which consistently repeat themselves in a number of different groups may be regarded as fairly reliable, and even if low in actual numerical value are probably definite indications of real relationships. It is necessary to examine the data with the utmost care at each stage in the investigation. The frequency distributions of the scores made in the tests, the intercorrelations of the tests themselves, the relationship between the tests and such variables as the age and experience of the workers, and the time of day at which the tests were performed—all these points have to be carefully examined for each group tested, and their influence, if any, on the final results eliminated before we can decide what meaning to attach to the results obtained.

An investigation of the nature described above, made over a large enough field, is of value quite apart from the practical solution of the immediate problem that it may afford. It will add new facts to the body of scientific knowledge and may definitely

advance psychologic theory. As was said at the beginning of this article, such a research involves a thorough investigation into the interconnection of various mental functions; and provided the work is of sufficient scope, light will be thrown not only on the nature of these functions themselves but also on their relationships with one another and their relative importance in different psychologic situations.

Our attitude toward the study of accident proneness is pragmatic: We are concerned with finding tests which will correlate with accident incidence, no matter what they actually measure. However, it is useful from the point of view of theory to name tests even though we cannot define the functions which they measure. Hence a set of intercorrelating tests which bear a definite relation to what is commonly called intelligence may conveniently be called intelligence tests. Similarly another set of tests may be called aesthetokinetic tests and be said to measure aesthetokinesis.³ Such names, of course, throw no direct light on the nature of intelligence and aesthetokinesis, but the nomenclature is useful for purposes of reference and comparison. For example, if we make the statement that both intelligence and aesthetokinesis play a part in engineering ability, what we actually mean is that there is some factor involved in both engineering proficiency and the ability to do well in certain

³ Three sensorimotor tests which were found to intercorrelate were called aesthetokinetic tests because their successful performance required accurate and rapid coordinated reaction to perceptual stimuli, although the form or perceptual setting of the tests differed considerably. They therefore form a special group of sensorimotor tests, distinguishable from sensorimotor tests in general (4) (5).

tests which we call intelligence and aesthetokinetic tests. This is a definite advance in psychologic theory; for, without knowing in the least what the function we have called aesthetokinesis really is, we can definitely say that it is a function which is measured by a certain set of sensorimotor tests and that it plays a part in engineering proficiency and in the avoidance of accidents. The relative importance of the part it plays in the two cases may be determined by correlation.

This article would not be complete without a brief account of the practical work which has already been done in this field. In the course of several years Mr. Farmer and I have given thirteen different tests to a large number of groups of workers, totaling over 1,800 subjects in all. Full details of the early part of this investigation have already been published by the Industrial Health Research Board (4) (5), and I shall just indicate here, in a few words, the scope of the experiment and the chief facts elicited.

Tests were given designed to measure intelligence—expressed through both a linguistic and a mechanical medium—sensorimotor ability of various degrees of complexity, physiologic characteristics such as physical strength and muscular control, and nervous stability. The subjects, who were for the most part engineering apprentices, were carefully chosen so that as many as possible of the factors such as age, experience, and exposure to risk were kept constant. These subjects fell into two groups, those who had suitable accident records and those who had suitable records of industrial proficiency. Unfortunately, no group possessed both sets of rec-

ords; consequently direct comparison between the relations of the tests to accident incidence and to industrial proficiency was not possible. This comparison could, however, be made indirectly.

It was found that a group of sensorimotor tests, those designated as aesthetokinetic tests, correlated with accident incidence, and that when their scores were weighted together with the entrance examination scores of the subjects, a final correlation coefficient of 0.406 with the accident rate was obtained. The correlation coefficient between the entrance examination alone and accidents was 0.294.

In the course of analyzing these results an interesting fact was revealed. It was found that where the accident records extended over several years, the relation between aesthetokinesis and proneness to accidents became more marked as the period of exposure to risk increased. Thus, while the coefficient of the correlation between aesthetokinesis and accidents for only one year was 0.099, it increased in size when the accidents were recorded for periods of two and three years, and became 0.444 when the period of exposure was four years. It is evident that the longer the period over which accident records are taken, the nearer does the actual number of accidents sustained come to being a true measure of accident proneness. Also, if a test really measures accident proneness, it is obvious that the better the measure of proneness, the higher will be the correlation between the test scores and proneness. Hence it follows that the relation between the test scores and accident incidence should become more marked as the period of exposure

increases. Since this condition is satisfied in the case of the aesthetokinetic tests, within the limits of the data available in this experiment, we have yet more evidence from a different standpoint to show that the aesthetokinetic tests really do measure a factor in accident proneness.

On examining the relation between the tests and industrial proficiency, it was found that three groups of tests correlated significantly with proficiency. First, there was the group of aesthetokinetic tests; second, a group of intercorrelating intelligence tests; and third, a dynamometer test for physical strength. Since these three groups of tests did not correlate with one another, we were able, by taking a single score for each group, to obtain three nonintercorrelating measures of industrial proficiency. When these were weighted together with the entrance examination scores of the subjects, a final coefficient of 0.472 with proficiency was yielded, the entrance examination alone giving a coefficient of 0.388 with proficiency.

Representative tests from each of these three groups had been given to four groups of subjects with suitable accident records. For these subjects, the tests were weighted into a single score, using the same weights as in the case of the correlation with industrial proficiency; and the correlation coefficient between this new weighted score and accidents was 0.446, which should be compared with the coefficient of 0.294 between the entrance examination alone and accidents.

These correlation coefficients, though statistically significant, are in no case very large; but a number of reasons have already been given to

show that the maximum coefficient of correlation that could possibly be obtained falls short of unity by an unknown, but probably considerable, amount.

Summarizing these results it will be seen that tests have been found which, when combined with the entrance examination of apprentices to a skilled trade, correlate more highly with accident proneness than does the entrance examination alone. Also, the same tests combined with the entrance examination correlate more highly with industrial proficiency than does the entrance examination alone. Therefore, if the entrance examination of these engineering apprentices were supplemented with certain of the psychologic tests, the candidates selected would on the whole be more proficient and less liable to have accidents than those selected by the present methods. The experiments are still in progress, and in the course of time it will be possible to publish a more extensive report in which the results of new tests for other psychologic functions, as well as the results of applying the same tests to further groups of subjects, will be given.

CONCLUSION

In conclusion I should like to emphasize the fact that although the study of accident causation is full of difficulties, as we have seen in the foregoing survey of the problem, yet certain definite advances have been made. That individuals differ in their proneness to accidents has been established beyond dispute, and it has further been shown that certain measurable psychologic functions play a definite part in this proneness. Moreover, imperfections in

the accident records militate against success in establishing the existence of a relation between any tests and accident incidence, so that any correlation that is found is not due to faulty data. Hence the results that have already been obtained must not be discounted by the fact that the data are imperfect; on the contrary, it may be said that if the data were free from error, the relation shown to exist between aesthetokinesis and accident proneness would be seen to be still closer than now appears.

Stress has been laid upon the extreme care and accuracy necessary to produce reliable results in an investiga-

tion of the sort described in this article. This, of course, only applies to the actual investigation itself. Once relations between certain psychologic functions and accident proneness have been definitely established, the tests measuring these functions may be used as a practical basis for selection, with the effect of lowering accident rate and increasing industrial proficiency.

The results presented in the foregoing are most encouraging and suggest that further research along similar lines may show what other functions are involved in accident proneness and may yield results that will be of great practical value.

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TOTAL HEAT DIFFERENCE AS A MEASURE OF WET KATA COOLING*

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IN 1922, O. W. Armspach and Margaret Ingels (1) proposed as the measure of wet kata cooling rate, at a given velocity, the difference between the total heat content of air saturated at the average temperature of the kata-thermometer and the total heat content of air saturated at the wet bulb temperature. This idea received little attention from other investigators until lately, when a number of correlations have been attempted with varying success. If total heat difference can be made the basis for calculating cooling rate, it should be possible to develop a formula involving total heat difference which can be shown to give the same results as the rational formula published in a recent issue of *THIS JOURNAL* (2).

The rational formula for the rate of cooling of a warm body at constant temperature, and covered with a film of moisture, was shown to be

$$n(T_k - T_w) = R(T_w - T_s) + \frac{C[(T_w - T^1) + 91(e_w - e^1)]}{1}$$

n = Combined coefficient of conduction and radiation into the water film, from the body.

T_k = Temperature of body, in degrees Fahrenheit.

T_w = Temperature of surface of water film, in degrees Fahrenheit.

T_s = Temperature of surrounding objects.

R = Coefficient of radiation.

C = Film coefficient of conduction from water to air.

T^1 = Wet bulb temperature of air.

e_w = Vapor pressure of water at temperature T_w , in inches of mercury.

e^1 = Vapor pressure of water at wet bulb temperature, in inches of mercury.

W. H. Carrier (3) has shown that the total heat above 0°F. in 1 pound of air can be calculated from the formula

$$H = C_p T^1 + L^1 W^1$$

H = Total heat in British thermal units per pound of dry air.

C_p = Specific heat of air at constant pressure (0.24 British thermal unit per pound).

T^1 = Wet bulb temperature, in degrees Fahrenheit.

L^1 = Latent heat at wet bulb temperature (British thermal units per pound).

W^1 = Weight of vapor in pounds associated with 1 pound of dry air, assuming saturated condition, at the wet bulb temperature.

Consider 1 mol of dry air saturated with moisture. If the barometric pressure is P and the pressure of the vapor is e , the pressure of the air is $P - e$. With every mol of air there will be associated $\frac{e}{P - e}$ mols of vapor, and with every pound of air there will be associated

$$\frac{e}{P - e} \times \frac{18}{28.98} = 0.621 \frac{e}{P - e} \text{ lbs. of vapor}$$

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where 18 and 28.98 are the molecular weights of water and air.

Total heat may then be expressed for a given wet bulb temperature

$$H = C_p T^1 + 0.621 \frac{L e^1}{P - e^1}$$

Consider now a warm body at constant temperature covered with a film of moisture. The difference between the total heat of air saturated at the temperature of the water surface and the total heat of air saturated at the wet bulb temperature is

$$C_p(T_w - T^1) + 0.621 \left(\frac{L_w e_w}{P - e_w} - \frac{L e^1}{P - e^1} \right)$$

In the temperature range from 60° to 95° the latent heat may be considered constant at the average value of 1,048 British thermal units.¹ Since the vapor pressures are small compared with the barometric pressure, they may be neglected in the denominators without great error, and the total heat difference may be written

$$C_p(T_w - T^1) + 0.621 \frac{L}{P} (e_w - e^1)$$

If the barometric pressure is 29.92 inches and L is 1,048, the expression becomes

$$0.24 (T_w - T^1) + 21.8 (e_w - e^1)$$

Take 0.24 outside of the bracket, and we have

$$0.24 [(T_w - T^1) + 91 (e_w - e^1)]$$

This is the total heat difference, the total heats being computed by neglecting the vapor pressures in the expression $P - e$. Comparing this

¹ From the Steam Tables of Marks and Davis.

expression with the last term of the rational formula, we see that the rational formula could be written

$$a(T_w - T_w) = R(T_w - T^1) + \frac{C}{0.24} (H_w - H^1)$$

where H_w and H^1 are the total heats at the temperature of the water and at the wet bulb temperature, computed by the approximate method.

Accordingly the cooling effect of the air can be computed either by the expression

$$C [(T_w - T^1) + 91 (e_w - e^1)]$$

or by the expression

$$4.16 C (H_w - H^1)$$

If actual values, not approximate values for total heat are used, the expression should be

$$3.94 C (H_w - H^1)$$

This factor was determined empirically by using values of total heat and vapor pressure from the psychrometric chart of Dr. E. Vernon Hill (4). The following are some values from the chart:

Wet Bulb Temperature °F.	Total Heat B.t.u./lb.	Vapor Pressure in. Hg
95	61.01	1.645
80	42.64	1.022
70	33.51	0.732
60	26.18	0.517

For example, assume that the temperature of the water is 95° and the wet bulb temperature 70°. The cooling effect of the air, by the rational expression, is, then

$$C [(95 - 70) + 91 (1.645 - 0.732)] = 108.08 C$$

By the total heat expression, it is

$$3.94 C (61.01 - 33.51) = 108.35 C$$

The agreement, when any wet bulb temperature in the range from 60° to 90° is used, is within 2 per cent.

The heat balance equation expressing the heat loss in a given environment is as follows, when the total heat difference is used.

$$n(T_a - T_w) = R(T_w - T_d) + 3.94 C(H_w - H_d)$$

SUMMARY

The effect of air at constant velocity on the rate of cooling of a warm body at constant temperature and covered with a film of moisture can be expressed closely as a function of the

difference between the total heat of air saturated at the temperature assumed by the surface of the water film and the total heat of air saturated at the wet bulb temperature.

The total heat of air saturated at the temperature of the body is in no way involved.

The rate of cooling of the warm body in a given environment cannot be expressed as a function of total heat difference, because total heat difference has to do only with the effect of the air, while the total rate of cooling is influenced by radiation exchange with surrounding objects.

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THE INFECTIVITY OF SILICOTIC TUBERCULOSIS*

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DR. COLLIS, in "The Statistical Characteristics of Dust Phthisis," published in *THIS JOURNAL* in November, 1926, referred, at the end of his paper, to "one last statistical peculiarity . . . , the certitude of which rests upon rather insecure data . . . "; viz., the supposed low infectivity of silicotic tuberculosis. He then set forth the evidence upon which this belief was based. It will, I think, be generally agreed that the evidence is mainly inferential in character, and that more positive data are needed before the fact can be regarded as established.

The examination and observation of "contacts" to cases of tuberculosis is now one of the statutory duties of British Tuberculosis Medical Officers. Accurate data regarding the infectivity of tuberculosis should, in time, emerge as a result of their labors. As far as silicotic tuberculosis is concerned, a special opportunity for settling a very interesting problem awaits the Tuberculosis Officers who work in areas where industrial silicosis prevails.

Although the examination of contacts was not so prominent a part of the work of a Tuberculosis Officer in 1926 as it is today, it seemed worth endeavoring to see whether some figures bearing on the matter referred to

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by Dr. Collis would not emerge from an investigation of the health of the families of the few cases of silicotic tuberculosis which I had under my care in the gritstone districts of Derbyshire, despite the smallness of their numbers. With that object in view, the following investigation was made in the winter months of 1927 to 1928.

Up to 1927 I had met with twenty-four cases of silicosis with pulmonary tuberculosis, mainly among the Derbyshire gritstone masons.¹ In every case the diagnosis had been verified by the presence of tubercle bacilli in the sputum, and the majority of the cases had already ended fatally. Although the number of cases was small, I thought that it might be worth while to investigate the condition of the widows, wives, and children of these men, and to compare it with the condition of the widows, wives, and children of a similar number of men with non-silicotic open tuberculosis, from the same district and of the same age at the time when tuberculosis was first diagnosed.

¹ The distribution of these cases according to occupation was as follows:

Gritstone bench hand.....	13
Gritstone scappler.....	4
Gritstone quarryman.....	3
Potter.....	2
Silica and ganister brickmaker.....	1
Cutlery grinder.....	1

I found, however, that in the district under investigation married men over 45 years of age rarely developed tuberculosis except they were silicotic; consequently I had to go a little further afield and to include some cases from outside the gritstone area in order to make up the necessary twenty-four controls. Finally, when I had collected the controls, I found that their ages averaged 52.5 years as against 48.5 years in the silicotic group.

The results worked out as follows:

	<i>Tuber- culosis with Sili- cosis</i>	<i>Tuber- culosis without Sili- cosis</i>
Cases:		
Total no.....	24	24
Average age on diagnosis.....	48.5	52.5
Wives and Widows:		
Total no.....	19	20
No. developing tuberculosis.....	0	1
Percentage developing tuberculosis.....	0	5
Children Born:		
Total no.....	91	68
No. developing tuberculosis.....	13	8
Percentage developing tuberculosis.....	14.3	11.8
Children under 14 on Date of Fathers' Diagnosis:		
Total no.....	23	9
No. developing tuberculosis.....	11	3
Percentage developing tuberculosis.....	48	33.3

The general rate of incidence of conjugal tuberculosis has been the cause of much controversy, but is, I think, now generally accepted as averaging about 5.6 per cent. of noninfected persons who marry infected persons; and the investigations of the Tuberculosis Officers of Lancashire have given us

monumental figures of the percentage of children of tuberculous fathers who develop the disease—figures which show a higher incidence of the disease than in the cases of the forty-eight fathers detailed above. As far as the meager figures which I collected can be relied on, they show that for approximately the same age group, the silicotic fathers were slightly more infectious to their children than the non-silicotic. And further, the figures suggest that the apparent low infectivity of silicotic tuberculosis is attributable to the late age at which the tuberculosis develops.

The British workman, in the silica industries as in others, marries young. By the time he has reached 48 years of age, most of his children have grown up and passed the most dangerous period of susceptibility to tuberculous infection. In the exceptional cases in which young children were found in the households of the silicotic fathers, the tuberculosis incidence rate was very high.

The total absence of conjugal infection among the wives and widows of the silicotic cases is on all fours with the oft-quoted observation of George Bauer—better known as Georgius Agricola—who wrote, in 1557, that women could be found in the mining communities of the Carpathian mountains, who had been widowed six times over by miners' phthisis. The work of this German on miners' phthisis, published nearly 400 years ago, suggests to us that we might pay a little more attention today to German work on silica and its biochemistry, if for no other reason than because its priority is so unassailable.

The figure of 5.6 per cent. men-

tioned for conjugal tuberculosis is an "average" figure, and therefore subject to the usual fallacies of averages. There is, for example, little doubt that the incidence rate among young married couples is higher than 5.6 per cent., and that in middle age it is lower. There are many other factors to be considered, as well as the ages of the infectious and the noninfected spouse. Child bearing, lactation, housing, nutrition, educational and social condition, all have their influ-

ence. But when, eventually, all the factors which determine the incidence of conjugal tuberculosis come to be included in one comprehensive equation, we must not be surprised to find that the variable for "silicosis" has dropped out.

Meanwhile, facts are needed. It is in the power of the Tuberculosis Officers working in silicotic areas to pile up these facts, little by little, from the families of silicotic patients who come under their observation.

THE OCCURRENCE OF PULMONARY FIBROSIS AND OTHER PULMONARY AFFECTIONS IN ASBESTOS WORKERS

(Concluded)

E. R. A. MEREWETHER, M.D.

H. M. Medical Inspector of Factories

MORBID ANATOMY

Professor M. J. Stewart of Leeds has kindly supplied me with a detailed description (dated March 28, 1929) of postmortem appearances of several cases. From this account the following points are extracted:

The gross morbid anatomy of this lesion, as seen in persons who have been at work for many years in the dusty atmosphere of an asbestos factory, consists in a widespread pulmonary fibrosis affecting especially the basal region of both lungs. There are extensive, densely fibrous adhesions throughout the pleural sacs, which may, indeed, become completely obliterated. In particular, the bases of the lungs are firmly adherent to the diaphragm, and between these two structures there is often a thick, homogeneous layer of fibrous tissue, similar in appearance and consistence to yellow fibrocartilage. As a result of these two processes—pulmonary fibrosis and pleural sac obliteration—bronchiectasis develops in the midst of the more grossly diseased tissue and may go on to the formation of multiple abscesses with smooth walls.

The distribution of the fibrosis, apart from the extensive basal lesions, is rather irregular, the peripheral, subpleural regions of the lungs being more affected than the central areas. In one case in which the patient had been away from work for four years (following nineteen years in the mill), the areas of dense fibrosis were extraordinarily sharply circumscribed. The nodular character of the lesion which is so striking a feature of silicosis in its earlier stages is not met with in this disease.

The totally fibrosed areas of lung show grayish-black mottling, owing to immobilization of carbon. This tissue is excessively dense and completely airless; but there is no evidence of calcification. Other portions of lung show varying grades of the same process, and even the least affected parts are definitely tougher than normal. In none of the cases was there any evidence of an active tuberculous lesion.

HISTOLOGY

The essential lesion is a chronic interstitial fibrosis of the lung. In the earlier stages, there is fibroblastic proliferation in the alveolar walls, which become increasingly thickened in consequence; and there is catarrhal desquamation of the alveolar epithelium. At this stage of the disease peculiar golden-yellow "asbestosis bodies" are found in varying, usually considerable numbers, both in the alveolar spaces and in the thickness of their walls. Detached portions of bodies may often be seen engulfed by alveolar phagocytes, or a large unbroken body may be partially surrounded by these cells. In the more advanced stages of the disease, the fibrosis of the lung is complete. Alveoli can no longer be made out; but the asbestosis bodies still remain embedded in the fibrous tissue. Varying quantities of carbon are also present, chiefly in little masses as though it had been contained within phagocytes. Chronic bronchitic

changes are present and all grades of bronchiectasis may be seen, in cases of long standing, from simply bronchial and bronchiolar dilatation, with walls still more or less intact, to large spaces filled with pus in which the original bronchial walls have become converted more or less completely into fibrous and granulation tissue. Many of the smaller arteries show obliterative endarteritis.

Reversionary metamorphosis of alveolar epithelium is frequent, and such alveoli are often filled with albuminous fluid, in which clumps of "bodies" may be found.

ASBESTOS DUST IN THE LUNGS

Dr. W. E. Cooke has supplied (under date of June 25, 1929) details of his researches on asbestos dust in the lungs, and on the constitution of the curious bodies, which have been freely used in the following notes:—

Sections of lung and the results of digestion of the lung with trypsin show an enormous amount of fine black granular dust, much of which is carbonaceous. In addition, there are two striking features. The first is the almost complete absence of the fine translucent spicules of fiber which make up a great proportion of asbestos dust in factories. The second feature is the presence of large fragments varying in length from 10 to 360 microns. They are found in fibrotic and necrotic areas, singly and in groups. The particles are so large—masses of them are seen in some sections—that they must have occluded small bronchi and resulted in fibrosis of the surrounding area.

Comparing these large particles in the lung with those found in asbestos

dust, close resemblance in sizes, shapes, and colors is apparent. There are the same black, blue, brown, and translucent fragments. In fact it is easy to take such a single particle from the lung and immediately find its brother in a slide made from the dust.

Curious Bodies

In addition to the fine granular dust and larger fragments of asbestos, sections of the lungs show curious bodies. They are found in alveoli, bronchioles, fibrous and necrotic areas, and in phagocytes in sections of both lungs. If a portion of lung is teased and extracted with water, or digested with trypsin, or, as Professor Stewart pointed out (1928), if a smear is made from the cut surface of the fresh lung, these bodies are seen in myriads. The larger bodies measure from 20 to 100 microns or more in length, and are of a golden-brown color. They may have single clubbed ends or may appear as elongated dumb-bells; some are filamentous, while others suggest a series of disks.

Single coccal and spore-like forms, and aggregations of these, and streptococcal forms are not uncommon; the color varies in the smaller types from a very pale yellow to a yellowish-brown. The bodies do not stain with any of the aniline dyes, but in chrysotile workers, they give the Prussian blue reaction for iron in varying degrees of intensity. These curious bodies have been found in every autopsy in pulmonary asbestosis. It would be unprofitable to retrace all the steps which led down many by-lanes during the course of the work, but I will mention a few pertinent details of interest.

Professor Stewart suggested to me

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that a better method than simple extraction of the lungs with water or saline would be to digest portions with trypsin. During this process it was found that if the specific gravity was kept at about 1,070, the black dust and larger fragments of asbestos, as well as the partially digested lung tissue, sank to the bottom of the tube. By decanting the apparently clear supernatant liquid, and by centrifugalizing, neutralizing, and washing the deposit, the curious bodies could be obtained in a pure state. This was done, and sufficient material for X-ray examination was obtained. The bodies were attached to a hair with gum and subjected to a seven-hour exposure by Professor Bragg's method. If the bodies were mineral, the X-ray film would have shown a definite translatable atomic pattern. The films, however, did not do so, and we were then able to exclude the suggestion that the bodies were altered asbestos fiber. The result definitely pointed to the greater proportion of their composition being of nonmineral origin.

The bulk of the curious bodies is soluble in strong acids and alkalies, and if the solution is observed under dark ground illumination, their bases are seen as extremely fine spicules some of which by transmitted light would probably be invisible. Under a dissecting microscope it is possible partially to fracture the larger bodies and to show a central fine core.

The greater portion of asbestos dust consists of slender translucent fibers. In sections and extracts of the lungs there is a remarkable paucity of these fine spicules. The end-results of digestion show the fine granular dust and

the large black, blue, and brown particles and what appear to be pieces of quartz. Relatively few fine spicules are found; but curious bodies of all descriptions are present in enormous numbers.

All these facts lead us to imagine the bodies to consist of a central nucleus of asbestos spicules, upon which colloidal aggregate of blood proteins plus, possibly, soluble fractions of asbestos and, in the case of chrysotile workers, an iron salt have been adsorbed and molded by currents in the bronchi and the alveoli.

The method of formation would appear to be as follows: The fine spicules of asbestos cause, by mechanical action on the bronchioles and alveoli, either minute extravasations of whole blood, or serous exudates which envelop them. Solution of any soluble fraction of asbestos takes place. The total amount of asbestos that is soluble must be extremely small, as is proved by the X-ray pattern of the curious bodies, but in the case of chrysotile workers some solution is suggested by the free iron reaction. We must remember, however, that the Prussian blue reaction may be due to the iron of the hemoglobin. Any surface in contact with a colloidal solution may act as an adsorbent, and in the present case the fine spicules must be considered to do so. Interaction between the soluble fraction of chrysotile and plasma proteins takes place, syneresis occurs, and, with the loss of water, the adsorption is rendered irreversible. The adsorbent is permanently ensheathed with stable colloidal aggregates which become molded into the familiar

shapes by alveolar and bronchial currents.

Support for this idea is found in the fact that micro-organisms adsorb colloidal material in the presence of blood serum and colloidal asbestos. Staphylococci so treated appear as large round yellowish-brown disks; in the process their property of staining with aniline dyes is lost. The organisms coalesce and form masses; and I think it probable that some of the coccid and spore forms are similar organisms.

Finally, are the curious bodies diagnostic of pulmonary asbestosis? Asbestos is unique among minerals in being fibrous; and its dust, generated during manufacturing processes, is also unique. As can be imagined from the formation of the curious bodies, there is no reason why, given any silicosis, a fine spicule of mineral should not have colloidal matter deposited around it and become molded into a curious body. But as no other mineral dust is fibrous, this occurrence must be so rare as to be negligible from a diagnostic point of view. The conditions which, apparently, must obtain for the formation of curious bodies are the presence of plasma proteins and fine spicules soluble only with difficulty. These conditions are ideally found in asbestos workers; for this reason I believe that curious bodies, if found in any numbers, are pathognomonic of pulmonary asbestosis.—

W. E. Cooke and also Roodhouse Gloyne (20) have independently demonstrated the presence of a mineral core, evidently derived from asbestos fibers, in the curious bodies. The bodies have been found (Stewart and Haddow (21)) in the intrathoracic

lymphatic glands; in material obtained by lung puncture during life, first suggested by S. A. Henry; in the sputa; and in smear preparations from the cut surface of the lung. In smear preparations they can be readily demonstrated.

The importance of these findings lies in the fact that the bodies have never been found, as yet, in any other human affection. That similar bodies may be found to occur in the lungs of workers exposed to other dusts, is quite possible, although they have not been found in silicosis, nor were they present, according to Simson (6), in the fibrosed lungs of a hematite miner.

They appear within a comparatively short period of time after exposure to asbestos dust; at the moment, their presence in the sputa, in the absence of clinical or radiologic evidence of pulmonary fibrosis, cannot be taken as indicating anything more than previous inhalation of asbestos dust. The presence of the bodies, however, in the sputa of persons with signs of a diffuse fibrosis, or their presence in numbers on postmortem examination of a fibrosed lung, has evident implications.

Professor Stewart, who is continuing his work on this subject, has devised the following method of examining sputum for asbestosis bodies:

Half an ounce or so of sputum is added to an equal quantity of undiluted antiformin. This is gently agitated until the sputum is completely dissolved, after which it is diluted with 2 or 3 ounces of water and allowed to stand in a large test tube for three or four hours. The bulk of the supernatant fluid having then been decanted, the remainder is centrifuged at a moderate speed for ten to fifteen minutes. The whole of the supernatant fluid is now poured off, and the deposit transferred to an albumin-

ized slide, by means of either a platinum loop or a pipette. After thorough drying on a hot plate and final fixation over a bunsen burner, the film is very gently washed in water, dried, and mounted in Canada balsam. After a little experience the asbestosis bodies are readily picked up with the low power, and their true nature is then confirmed with the $\frac{1}{4}$ -inch or oil immersion lens. As a rule they are present in very small numbers, perhaps only one or two in a whole film. In the case of some of the older workers, however, numerous bodies, up to one or two per field in certain portions of the film, have been found. It is obviously necessary to cleanse thoroughly all glassware, etc., used in making these examinations; otherwise there is a risk of contamination of subsequent specimens.

The films can be treated with hydrochloric acid and ferrocyanide of potash to demonstrate the Prussian blue reaction given by the bodies.

ASBESTOSIS AN OCCUPATIONAL HAZARD

Out of a total of 374 workers examined, 105, or 28.1 per cent., were found to be affected with fibrosis of the lungs, in greater or less degree. But when these figures are corrected by the exclusion of cases in which previous work in dusty occupations (*e.g.*, quarrying, coal mining) may have been the prime, or a contributory, factor in the development of the fibrosis, ninety-five, or 26.2 per cent., of 363 workers were found to be affected with pulmonary fibrosis due to the inhalation of asbestos dust, and a further twenty-one, or 5.8 per cent., showed precursive signs of this disease.

This percentage may well be compared with the corresponding figures found by Middleton (22) in his examination of metal grinders (46.9 per cent.) and by Sutherland and Bryson (23) (24) in their examinations of potters

(39.9 per cent.) and of sandstone workers (58.1 per cent.).

The precise significance of the lower figure found in this inquiry is not easy to determine. At first sight it seems to indicate that asbestos dust is less potent as a cause of pulmonary fibrosis than are the dusts containing free silica. Other factors, however—such as the measure of exhaust ventilation, or other means of reducing the concentration of dust, in the several industries; differences in the average length of employment of the comparable groups in the samples examined in the respective inquiries; and the relative numbers employed in the more dusty and the less dusty processes—will affect the crude incidence rates expressed by these percentages.

From comparison of the amounts of dust evolved—determined visually and by means of dust counts—in dusty asbestos processes, uncontrolled by local exhaust ventilation, with obviously defective exhaust ventilation, and with comparatively good exhaust ventilation, there can be no doubt that this preventive measure, which has been applied in some degree for more than seventeen years, has been a positive factor in minimizing the production of fibrosis—probably in the direction of lengthening the period before the fibrosis becomes fully developed.

Consideration given to the distribution of the workers examined both according to length of employment, and according to age, together with the incidence rates of fibrosis in each case, indicates the outstanding importance of length of employment (and hence length of exposure to dust), and the negligible effects of age, on the production of fibrosis. Thus age groups 30 to

39 and 50 to 59 have incidence rates of 30 per cent. and 37.9 per cent., respectively—not a wide difference, since the groups include workers in various processes exposed to different concentrations of dust. Moreover, the average length of employment in these two groups, excluding the cases of fibrosis,

fibrosis in age group 20 to 29, has been shortened by increased susceptibility at ages under 20. As, however, the average age of this group of cases of fibrosis is 26.7 years, and also because the establishment of fibrosis does not necessitate immediate retirement from work, this is largely discounted.

TABLE 5.—DISTRIBUTION OF WORKERS EXAMINED WHO HAD BEEN EMPLOYED FOR FIVE YEARS OR OVER, TOGETHER WITH CASES OF FIBROSIS, ACCORDING TO PROCESS IN WHICH WORKER WAS LONGEST EMPLOYED

PROCESS	NO. EX-AMINED	CASES OF FIBROSIS		AVERAGE LENGTH OF EMPLOYMENT IN YRS.	
		No.	Per Cent. of Group	Group Less Cases of Fibrosis	Cases of Fibrosis
1. Crushing, opening, disintegrating, and mixing.....	21	9	42.9	8.9	10.9
2. Carding.....	39	16	41.0	9.1	13.2
3. Spinning, twisting, doubling, plaiting, etc.....	87	12	13.8	10.1	18.7
4. Mattress making.....	15	7	46.7	7.3	13.0
5. Weaving and associated processes.....	88	40	45.5	10.0	12.7
a. Cloth weaving.....	28	21	75.0	9.5	14.1
b. Band weaving.....	21	4	19.0	10.9	13.5
c. Cloth and band and unclassified weavers.....	14	4	28.6	7.2	9.5
d. Warpers, beamers, loom tuners, charge hands, and others associated with weaving.....	25	11	44.0	11.2	10.8
6. Miscellaneous processes and remaining unclassified workers.....	24	11	45.8	8.8	13.8
Total.....	274	95	34.7	9.6	13.5

is also similar, but considerably less than the average length for all the cases of fibrosis (13.5 years).

No special susceptibility to the development of fibrosis is shown by young persons, unless it is considered that the figure of 8.7 years, the average length of employment of the cases of

The effects of length of employment are such that after five years' exposure, the incidence rate of fibrosis mounts rapidly, and after ten years increases almost in geometric progression. Table 5 shows the distribution of 274 workers examined, and of those with fibrosis, according to the process

in which they were longest employed. This was a compromise necessitated by the common customs in the industry of having several different processes going on in the same room, and of workers transferring from one process to another. Since the intention was to obtain some measure of the *relative* incidence of fibrosis in workers in the various processes enumerated, and since no case of fibrosis clearly due to asbestos dust was found among eighty-nine workers examined, who had been employed for less than five years, inclusion of this group of workers would only have introduced a varying source of error.

The processes differ, some very materially, in the amount of dust which they cause, and consequently in the amount inhaled by the operators (30). The outstanding point brought out by Table 5 is the relatively low incidence rate of fibrosis in "spinners" (group 3), as compared with each of the other groups. They are corroborated by counts of the dust particles in samples of air, a number of which have been taken and are discussed under *Dust Risk*.

There is also some indication in these figures that not only are "spinners" less likely to develop fibrosis, but when they do, it takes longer to develop. Study of the radiograms suggests that in such cases (i.e., in the less dusty processes) the resulting fibrosis is, for a considerable period, much more linear in its radiologic features, and shows less mottling. The history and clinical features in these cases, and the comparative dust counts, all contribute to the view that with comparatively low concentrations of dust, the resulting fibrosis is longer in developing and

remains longer in the milder, and radiologically linear stage.

This view is evidently of considerable practical importance since it suggests that in such cases, the rate of accumulation of dust in the lung has not greatly exceeded the rate of elimination, the changes still being centered in the main lymphatic system.

If this hypothesis is accurate, it should follow that in order to prevent the full development of the disease, among asbestos workers, within an average working lifetime, it is necessary to reduce the concentration of dust in the air of the workrooms to a figure somewhat below that pertaining to spinning at the present time.

Fortunately, also, the spinning group is the largest individual group in this section of the industry—e.g., out of over 400 workers employed by one firm, this group accounts for more than one-third.

Although all workers examined who have been included as spinners have been engaged in spinning, or in similar processes for convenience termed "spinning," for a longer time than on any other process, only a few have been employed solely on spinning, and in workrooms where no other, and more dusty processes, were being carried on.

These two factors, prior work in more dusty processes and present work in proximity to more dusty processes—especially the latter—have had, it is believed, some effect in raising the incidence rate of fibrosis for this group.

Dust Risk

In an effort to obtain additional data on the influence of concentration of dust in workrooms, a number of samples of air were taken and the dust con-

TABLE 6.—SUMMARY OF DETERMINATION OF DUST CONTENT OF ATMOSPHERE OF WORKROOMS AND OTHER PLACES

SAMPLE NO.	PROCESS AND SOURCE OF SAMPLE	LOCAL EXHAUST VENTILATION TO PROCESS OR NOT	OTHER PROCESSES IN SAME ROOM	NO. OF PARTICLES PER C.C.	PERCENT-AGE 2 μ AND UNDER	PERCENT-AGE 7.5 μ AND UNDER
1. 1/2	Carding; passageway between cards	Yes	Yes	2,139	81.3	95.1
2. 1/5	Spinning	No	Yes	1,456	95.9	100.0
3. 1/3	Cloth weaving, dry	No	Yes	1,756	84.1	96.7
4. 1/7	Opening	Yes	Yes	2,384	80.9	93.7
5. 1/6	Sieving; shoveling fiber on to sieve	No	Yes	2,829	83.0	97.6
6. 1/8	Plaiting	No	No	1,687	99.0	99.0
7. 1/1	Office	...	...	901	98.8	100.0
8. 2/11	Office	...	...	1,480	98.7	100.0
9. 2/1	Cloth weaving	No	Yes	1,788	88.9	98.6
10. 2/14	Spinning	No	Yes	1,073	96.2	100.0
11. 2/10	Mattress making; filling	Yes	No	985	92.5	98.1
12. 2/8	Mattress making; beating, damp	Yes	No	970	85.9	95.8
13. 2/7	Mattress making; beating, dry	Yes	No	1,629	86.9	96.6
14. 2/9	Mattress making; after filling 2 mattresses	Yes	No	1,204	96.6	99.4
15. 3/1	Band weaving, wet	No	No	808	96.6	100.0
16. 3/4	Opening	Yes	No	2,313	77.7	96.6
17. 3/3	Carding; between cards	Yes	Yes	1,528	82.5	97.4
18. 3/5	Carding; feeding card	Yes	Yes	1,321	82.9	96.4
19. 3/2	Office	...	...	1,049	96.3	100.0
20. 4/1	Mattress making; filling	No	Yes	1,858	60.1	90.1
21. 4/1	Mattress making; filling basket	No	Yes	1,390		
22. 4/3	Emptying settling chamber	No	Yes	3,401		
23. 4/4	Same	No	Yes	4,711		
24. 4/5	Insulating sections; leveling fiber	No	Yes	1,838		
25. 4/5a	Carding; side of dirty card	Yes	No	1,402		
26. 4/6	Carding; end of clean card	Yes	No	1,092		
27. 4/7	Carding; feeding card	Yes	No	1,689		
28. 4/9	Cloth weaving, very slightly damp	No	Yes	3,033		
29. 4/10	Cloth weaving, damp	No	Yes	758		
30. 4/11	Braiding	No	Yes	506		
31. 4/12	Spinning	No	Yes	954		
32. 4/13	Office	...	...	689		
33. 5/1	Band weaving, dry	No	Yes	2,202		
34. 6/2	Cloth weaving, dry	Yes	No	885		
35. 6/3	Band weaving, dry	Yes	No	701		
36. 6/9	Opening	Yes	Yes	643		
37. 6/6	Carding; feeding card	Yes	Yes	562		

TABLE 6.—Continued

SAMPLE NO.	PROCESS AND SOURCE OF SAMPLE	LOCAL EXHAUST VENTILATION TO PROCESS OR NOT	OTHER PROCESSES IN SAME ROOM	NO. OF PARTICLES PER C.C.	PERCENT-AGE 2 μ AND UNDER	PERCENT-AGE 7.5 μ AND UNDER
38. 6/4	Carding; center of room	Yes	Yes	620		
39. 6/5	Carding; grinding rollers	Yes	Yes	540		
40. 6/7	Spinning	No	Yes	620		
41. 6/8	Office	...	...	666		
42. 7/2	Cloth weaving, dry	Yes	Yes	4,688		
43. 7/1	Opening; shoveling fiber into box	Yes	Yes	4,880		
44. 7/3	Opening	Yes	Yes	6,324		
45. 7/4	Band weaving, wet	No	Yes	5,353		
46. 7/5	Spinning	No	Yes	6,044		
47. 7/6	Carding; between cards	Yes	Yes	4,607		
48. 7/7	Office	...	...	3,413		
49. 8/2	Cloth weaving, dry	No	Yes	1,287	88.6	94.9
50. 8/3	Band weaving, dry	No	Yes	1, 43	78.2	93.7
51. 8/5	Carding	Yes	Yes	1,195	67.2	94.9

tent was determined by means of Owens' jet apparatus. Here also the undetermined effect of dust from neighboring processes must be noted. Nevertheless some information of practical value was obtained.

Reference to Table 6 shows that the counts ranged from 596 to 6,324 particles per cubic centimeter in the air of workrooms, and from 666 to 3,413 per cubic centimeter in the air of offices associated with the factories. In some cases there was a smaller count in a workroom than in an office of the same factory; this was due either to there being much fewer smoke particles in the air of the workroom, or to adherence of the smoke particles to the inorganic dust in the workroom.

The character of the dust in the two cases is, however, entirely different. In the air of the office almost all the particles are black, or brownish, and amorphous, evidently smoke; in the

air of the workroom, the majority of the particles are, just as clearly, crystalline particles of asbestos, together with, frequently, some cotton fibers and numbers of black and brownish particles, some adherent to asbestos spicules and some free. Most of the latter are iron containing and are derived from the asbestos, and there are relatively few smoke particles. Isolated golden-brown scales or plaques, also derived from the asbestos, are scattered about the field.

The asbestos fiber first fragments longitudinally, and apparently this process can go on indefinitely, since there is no ultimate fiber comparable to a vegetable fiber such as cotton. The dust collected in the Owens apparatus contains numbers of these very fine fibers—some about 0.5 micron in transverse diameter, many less—which have fragmented transversely. Thus spicules of very diverse length—up to

about 80 microns—but about 0.5 micron broad, are found in numbers on the sample, together with the black, brownish, and yellow particles already mentioned. The great majority of the spicules are found broken down into particles of the order of 2 microns and less, many being about 0.5 micron and more or less rounded. The proportion of elongated to rounded particles seems to vary in the different processes; for example, a greater proportion of elongated particles is found in the air near dry cloth weaving than near a spinning frame. Weaving damp reduces the proportion of elongated particles.

This variety of microscopic spicules, particles, and frequently some cotton fibers, found in the samples of dust collected in the Owens apparatus, results in a tendency to clumping on the cover slip, making counting, especially of the higher concentrations, a matter of extreme difficulty. Occasionally, also, part of the slot of the apparatus choked up with dust. The effect of the clumping is to reduce the counts of all except the low concentrations.

Furthermore, this apparatus is much influenced by local air eddies and currents. It takes a grab sample, which is accurate enough as an indicator of the local conditions at the moment of sampling, but does not give a picture of the average state of the atmosphere during a period of time, as does a gravimetric apparatus. Moreover, with high concentrations of dust, accurate counting becomes impossible.

The apparatus has, however, many advantages, including those of convenience, quickness in operation, and direct examination of the nature of the dust. By serial observations, too, temporary and local large increases in

the dust content of the atmosphere—such as that produced by shoveling asbestos fiber into a sack—can be detected and estimated. This is of great importance since short, but repeated, exposure to massive concentrations of dust is agreed to be very harmful. Counts Nos. 5, 22, 23, and 43 in Table 6 are examples. Much useful work can be done by means of serial dust counts in various dusty processes in the same factory, and by comparison of the results.

The counts set out in Table 6 are derived from eight different factories, and it is a matter of interest to consider whether counts from different factories can be directly compared with one another. Strictly they cannot, since the content of smoke particles varies in each group. The difficulty might be obviated by counting only the particles derived from asbestos. The method has been applied successfully to counts of crystalline silica dusts; but it is not so easily applicable to counts of asbestos dust, which contains numbers of black and brownish iron containing particles, many of which are not quickly distinguishable from smoke and tarry particles.

To classify the processes accurately on the basis of amount of dust evolved, it is necessary to take serial samples at intervals corresponding to the details and complexity of the processes. With some straightforward processes, such as spinning, few samples will be necessary; but in others, such as mattress making, mixing, grading and opening—especially with old-fashioned plant—in which the evolution of dust rises and falls rapidly in a comparatively short space of time, many more observations are necessary throughout

a shift, to determine the average concentration of dust. Repeated controls are also required.

Comparative data show that the dustiest processes are opening (with old-fashioned teasers), sieving (with no local exhaust ventilation), and shoveling, or otherwise handling asbestos fiber. The heaviest counts of all were found in this group, in fillingsacks, by hand, with disintegrated fiber in a settling chamber. The visual cloud of dust was intense, and irritating enough to provoke immediate coughing on the part of the observer. The clumping of the dust particles of the sample on the cover slip was very marked, with the result that the total dust count arrived at was undoubtedly too low. With counts of asbestos particles of this order, however, the precise count is of little practical importance, since the dust concentration is far beyond the safe limit.

Next in order is cloth weaving, dry and without the application of local exhaust. This is undoubtedly a dusty process, and although local exhaust reduces the count at the breathing level of the weaver, much dust still escapes into the workroom. Further counts are necessary to estimate this. Weaving cloth wet, not merely damp, reduces the dust count to a remarkable degree. The figure for band (i.e., narrow) weaving, wet, is not accurate since it was raised by dust from a neighboring dry cloth loom; still the improvement due to wet methods is noticeable. One asbestos weaver, who was formerly a cotton weaver, and who has to wear glasses, stated that whereas he used to clean his glasses about three times a day when cotton weaving, he has to clean them about five times a day

when asbestos tape weaving dry, and only once a day when weaving asbestos wet.

Next in order comes mattressmaking, without any precautions such as exhaust ventilation, or damping floors, cloth, and tables. Application of exhaust ventilation and damping reduce the count considerably, but it is believed that the figures are too low, since counts for some subsidiary processes such as buttoning, sewing, and cutting out are not available.

The figures for spinning, plaiting, and braiding are believed to be rather too high, owing to contamination of dust from neighboring and more dusty processes, and further investigation is required here.

A number of the samples taken with the Owens apparatus were examined to determine the size of the particles, and the percentages of particles 2 microns and under, and 7.5 microns and under, were ascertained. The 2-micron standard was retained for purposes of comparison, and because of its accepted importance in silicosis. The 7.5-micron figure was also adopted because of the pronounced acicular character of much of the asbestos dust, and because, representing the diameter of the human red blood corpuscle, it is a standard easily distinguishable, and may conceivably represent the limit size of particles which can be conveniently engulfed by phagocytic cells, which are somewhat larger. Among twenty counts from various processes (Table 6), in two, between 60 and 70 per cent. of the particles were of sizes 2 microns and under; in two, 70 to 80 per cent.; in ten, 80 to 90 per cent.; and in six, 90 per cent. and upward. When the counts were analyzed ac-

cording to the 7.5-micron standard, all twenty showed 90 per cent. or more of the particles to be of this size or less.

With regard to the effect on the lungs of different varieties of asbestos, no evidence was found to indicate that any one of its varieties is more potent in producing fibrosis than the others, other factors, such as concentration of dust, being equal. There is now no doubt, however, that both chrysotile and crocidolite will produce fibrosis; while the third, the comparatively newly discovered amosite, resembles crocidolite so closely in its chemical constitution, and in the characteristics of the dust, that there can be no reasonable doubt, also, with respect to it.

Length of Dust Exposure

No case of diffuse fibrosis clearly due to asbestos was discovered with under 5 years' employment. Three cases were found with 3, 3½, and 4½ years' work, respectively, who showed clinical signs of fibrosis. Definite confirmatory radiologic evidence was obtained in the first, definite but slighter radiologic changes in the second, and indefinite suggestive changes in the third. The previous occupation in the first case was flax weaving for 18 years; in the second, coal hewing for 16 years; and in the third, work in an iron foundry for 1 year. All these occupations are dusty; and in the last two there may be some exposure to free silica. Also increased silica content has been found in the lungs, after incineration, of flax dressers. For these reasons, these three cases could not be certainly ascribed to asbestos dust, although it was, probably, at least a contributory factor. Among

the cases of fibrosis found, thirty-six had been employed in asbestos for periods ranging from 5 to 10 years; two of these cases, one employed for 9 years and the other for 7 years, show that a considerable degree of fibrosis may occur with less than 10 years' exposure.

F. W. Simson (6) examined the lungs of a guinea-pig which had been experimentally dusted by Mavrogordato for two hours a day on each of fifty days between February and April, 1925, and which died from other causes in December, 1927. He states that histologic sections showed a slight generalized fibrosis. This observer also examined portions of the lungs of two native asbestos mill workers; one had been employed for twelve months and had died from a miliary tuberculosis, and the other, employed for two years, had apparently never recovered from an attack of lobar pneumonia a year before death.

In commenting on the amount of fibrosis found, he states that "a comparison between the human cases and the experimental animal showed that the fibrosis was more rapid and extensive in the human cases than in the experimental animal," and, again, that "the amount of fibrosis in two of the human cases . . . was quite definite, and, if due to the presence of asbestos dust, the initial rate of production was rapid when compared with present-day non-infective silicosis on the Rand."

Experiments by Professor Beattie, in 1912, demonstrated that the lungs of guinea-pigs exposed to asbestos dust for forty-three and sixty-seven hours showed "definite cellular proliferation, though not very extensive, and this is

certainly a preliminary stage in the production of fibrosis."

These pathologic and experimental findings suggest that the inhalation of asbestos dust rapidly causes some changes in the lungs. But no evidence was obtained that asbestos dust can produce an acute type of fibrosis comparable to that formerly noted in South Africa after repeated exposure to massive concentration of free silica, a few cases of which have occurred recently in Great Britain, causing death in from two to four years.

The amount of disablement produced by the development of pulmonary fibrosis in these workers is surprisingly slight for a number of years, even more so than is generally the case in silicosis. The nature of the work, however, in the majority of the processes does not involve much physical exertion—in fact, in this respect it is "light work." The affected person may, and often does, continue at work—with occasional intermissions, latterly, due to exacerbations of bronchitis—until the condition is advanced, although he suffers increasing inconvenience from shortness of breath on the slightest exertion.

Usually these cases cease work a year or more before death, but sometimes a terminal bronchopneumonia, or other acute infection, commences while they are still at work, and there is no long period of invalidism.

Fatalities

Particulars of eight deaths have been collected, and are summarized in Table 7. In six of these, advanced pulmonary fibrosis was the primary cause of death. In the remaining two cases, pulmonary tuberculosis was a

contributory factor. In six of the eight cases the diagnosis was verified by postmortem examination; in the other two cases, confirmatory radiographic evidence was obtained.

Information has recently been obtained in regard to three other cases, in which death occurred in 1929. The details of one have been published by Wood and Page (25). Postmortem examinations have been made in all three cases, and in all, the presence of pulmonary asbestosis, without tuberculosis, was verified. In one case, the pulmonary fibrosis was the primary cause of death; in one, information as to the extent of the fibrosis has not yet been obtained; and in the third case, a lobar pneumonia was superimposed on the fibrosis.

It is not suggested that these few fatalities, in which the cause of death has been verified by strict inquiry, are any criterion of the true effect of the disease on the mortality rates of asbestos workers. Others are known to have occurred in which the existence of the asbestos fibrosis has been determined in life, but no postmortem examination has been possible.

Difficulties in Diagnosis

Many factors have contributed to impede both the recognition of individual cases of this disease, and the establishment of asbestos dust as the determining cause. These factors are consequent, partly on the nature of the disease, partly on the nature of the dust, and partly on the industry itself, its rapid growth and internal conditions. Difficulties in diagnosing the disease, its points of resemblance, in its latest stages, to fibroid tuberculosis, and the liability of those affected to be

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TABLE 7.—PARTICULARS OF FATAL CASES

NO.	CASE	DATE OF DEATH ¹	SEX	AGE	YRS. EMPLOYED	MAIN PROCESS	CONDITION OF LUNGS	POST-MORTEM EXAMINATION	IN-QUEST	REMARKS
1.	Montague Murray case	6/13/1900	M	34	14	Card room 10 yrs.	Advanced pulmonary fibrosis; no tuberculois	Yes	No	
2.	Cooke's case	3/14/24	F	33	18 ²	Spinner latterly	Extensive pulmonary fibrosis and tuberculois	Yes	No	Exposed to dust from other processes.
3.	H. S.	1927	F	48	24 ²	Mattress maker	Pulmonary fibrosis and tuberculois	No	No	Two radiograms confirm diagnosis of pulmonary fibrosis.
4.	M. S.	3/7/27	F	34	20+	Mattress maker	Chronic pulmonary fibrosis; no tuberculois	Yes	No	
5.	Grieve's case (W. L.)	3/24/28	M	34	13 ²	Mattress beater	Advanced pulmonary fibrosis; terminal bronchopneumonia; no tuberculois	Yes	Yes	
6.	M. M.	8/25/28	F	34	15-16	Mattress maker	Advanced pulmonary fibrosis; no tuberculois	Yes	Yes	Worked for less than the period stated, as a mattress maker.
7.	J. C.	10/13/28	M	36	18	Weaving 1 yr.; mattresses dept. 7 yrs. and 2 mos.	Advanced pulmonary fibrosis; no tuberculois	No	No	Two radiograms confirm diagnosis.
8.	L. H.	2/-/29	F	41	19	Mattress maker	Advanced pulmonary fibrosis; no tuberculois	Yes	Yes	

¹ Arrangement of dates follows the American custom—i.e., month, day of month, year.² Estimated.

carried off by some intercurrent disease—which alone is noted on the death certificate—are all of importance.

A concrete example encountered during the inquiry will illustrate one of these points. J. C., Case No. 7 in Table 7, was known by his own doctor and by the Chief Tuberculosis Officer of the town to be affected with advanced fibrosis of the lungs. Four or five months before his death it was thought that a change of air would be of advantage to him. Through the generosity of the firm with which he had been employed, he was transferred from the town to a farm in the country, where he died. The death certificate, given locally, stated that pulmonary tuberculosis was the cause of death. In another case chronic bronchitis was given as the cause of death.

In the less advanced stages of the disease, the symptoms are so unobtrusive that the worker rarely consults his doctor. In the later stages, bronchitis, or the supervention of acute bronchopneumonia, pulmonary tuberculosis, or other acute infection, all so much more common, masks the underlying fibrosis; and the terminal condition is noted as the cause of death.

In the second place, the silicate dusts—of which asbestos dust is one—representing silica in the combined form as opposed to free silica, have naturally been overshadowed by the silica dusts, since they do not produce the picture of silicosis and have not been shown to be associated with an increased mortality from pulmonary tuberculosis.

Moreover, although the asbestos industry has very rapidly expanded, it is still comparatively small, and scattered over the country. A large proportion of the workers have been employed in

asbestos only for comparatively short periods, and of the processes considered so far, the less dusty spinning group is at the same time the largest. Thus only now the existence of a health risk in the industry is beginning to be recognized.

DOES ASBESTOS DUST PREDISPOSE TO OTHER PULMONARY DISEASES?

The important question as to whether asbestos workers show an increased liability to other diseases of the lungs, such as pneumonia, pleurisy, and pulmonary tuberculosis—especially the latter—requires further investigation.

A history of pleurisy was given by ten workers, in eight since commencing work in asbestos; in addition, one worker had a slight pleural effusion at the time of examination, and one showed signs of old pleurisy, but no history was obtainable. Of the two with attacks prior to work in asbestos, one had normal lungs, and the other showed signs of a bronchiectasis. Of the eight giving histories of pleurisy, three showed signs of fibrosis, four showed signs of the old pleurisy only (one being due to a gunshot wound during the War), and the eighth showed signs of an old inactive tuberculous lesion of the right upper lobe.

A history of pneumonia was obtained in sixteen workers—in ten prior to commencing work in asbestos, and in six since. Of these six, two showed signs of a thickened pleura only, one showed merely enlarged lung roots, two had diffuse fibrosis, and in one the lungs were normal. Of the remaining ten, one had a thickened pleura, two bronchiectasis, two diffuse fibrosis, one

commencing fibrosis, and in four the lungs were normal.

With regard to evidence of bronchitis, and bronchial and pulmonary catarrh, fifty-eight of the 363 workers (16 per cent.) showed signs of one or other of these conditions, all being quite mild. Of these, thirty-one also showed signs of diffuse fibrosis, representing 32.6 per cent. of the fibrotic group, and eight were in the prefibrotic stage (38.1 per cent. of that group). The fact that the clinical examinations were conducted during the warmer months of the year, no doubt operated to reduce the incidence of these complaints. Under the circumstances, the preponderance among the cases of fibrosis is the more noteworthy, as indicating persistent irritation resulting from the dust.

Of other diseases, eight gave histories of rheumatic fever, and of these, two had definite valvular disease of the heart. In addition, valvular disease of the heart was noted in one worker, but no history of rheumatic fever was obtained.

With regard to pulmonary tuberculosis, so definitely an added risk in silicosis, this disease group has been drawn up to include all workers presenting signs indicating a past or present pulmonary tuberculous infection, but excluding cases of old inactive hilar tuberculosis. Under this heading are included not only workers with definite clinical signs of active tuberculous lung infections, or of old inactive lesions, equally definite, but also those workers showing signs of old apical collapse, usually attributed to a past tuberculous infection.

Signs of the latter are not uncommonly found in quite healthy persons,

and are of no importance unless it is shown that the inhalation of asbestos dust is associated with pulmonary tuberculosis to an increased degree, when such cases, as indicating a group of workers with a latent infection, would assume a greater significance. No close association is apparent, however, since only thirty-seven, or 9.9 per cent. of the 374 examined, showed evidence of this disease, excluding cases of old inactive hilar tuberculosis, even when those showing the minor changes mentioned above are included. In only four was there a family history of pulmonary tuberculosis; and three out of four active cases belonged to this group. Thirty-three cases were inactive, of which twenty-one presented no evidence of dust fibrosis, and twelve presented evidence.

Out of the 374 persons examined, fourteen, or 3.7 per cent., gave a family history of tuberculosis. Evidence of active pulmonary tuberculosis was present in three; in two of these three the source of infection appears to have been a near relative.

Thus, no outstanding susceptibility to pulmonary tuberculosis was disclosed, either among asbestos workers as a class, or among those with fibrosis, considering the frequency of old healed apical lesions among the general population.

We have, however, by no means disposed of the question. The supervention of a tuberculous infection on a lung already the subject of fibrosis produces an increase in symptoms, previously unnoticed or disregarded, and causes the worker to seek his doctor's advice. He is then appropriately advised to give up his dusty employment, migrates from the industry, and

may or may not accept sanatorium treatment. Thus there tends to be a drift of such cases from the fibrosis producing industry.

During the course of the inquiry, information was obtained of a number of persons, previously employed in asbestos, who were either at home or in sanatoriums, suffering from chest complaints. Where an examination of workers is confined to those at work, a certain number of advanced cases of fibrosis, and a certain number of cases of pulmonary tuberculosis, with or without an asbestos fibrosis, in persons who have either given up work, or who are off work temporarily, will be missed.

It is necessary, therefore, to leave this question of increased susceptibility to pulmonary tuberculosis in abeyance, pending further investigation.

With respect to any increased susceptibility to the supervention of other lung diseases in workers with an asbestos fibrosis, the data obtained were insufficient to lead to any conclusion, except as to bronchial catarrh. There are indications, however, that when pneumonia or bronchopneumonia supervenes on a fibrotic lung, the prognosis is grave.

ASBESTOSIS AND SILICOSIS CONTRASTED

In view of the relationship between the asbestos fibrosis and silicosis, since they are both varieties of fibrosis of the lungs caused by the inhalation of dusts, it seems desirable to summarize shortly the main points of similarity and dissimilarity between the two diseases, so far as has been ascertained.

1. Inhalation of asbestos dust, under favorable conditions of concentration of dust and length of exposure, will

produce a serious and even fatal degree of pulmonary fibrosis.

2. The type of fibrosis produced, however, exhibits a number of divergences from that caused by the inhalation of free silica dust, and the outcome of these variations is not yet apparent.

3. The divergences noted so far are exhibited in the radiologic picture, and on postmortem examination of the affected lungs, both in the gross morbid anatomy and in microscopic sections.

4. In the latter case the appearances of the asbestos fibrosis seem to be sufficiently distinctive not to permit of confusion between it and silicosis.

5. The radiologic appearances of the asbestos type of fibrosis, even in the more developed stage, are more delicate, softer, and more diffuse than the silicotic fibrosis.

6. It follows, therefore, that an opinion as to the degree and intensity of an asbestos fibrosis, based on a comparison of the radiologic changes with those shown in standard silicosis films, will be an underestimate.

7. In the absence of a full medical and industrial history, possibilities of confusion of the two types of fibrosis will arise in the interpretation of radiograms.

8. There is evidence that other inorganic dusts, containing no free silica, may be productive of this fine type of fibrosis in varying degree.

SUMMARY

1. There is a definite risk of the development of a diffuse pulmonary fibrosis in persons exposed to the inhalation of asbestos dust.

2. This risk varies directly as the length of employment (and hence

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length of exposure to dust), and is unaffected by the age of the worker.

3. There is a considerable difference between the least dusty and the most dusty processes investigated, and there is a corresponding variation in the relative risk of the development of fibrosis in the workers in these processes.

4. With continued exposure to high concentrations of dust, the disease may be fully developed in seven to nine years, and may cause death after about thirteen years' exposure, exceptionally in a shorter period.

5. Fibrosis as it occurs in asbestos workers differs considerably from the disease as it occurs in workers exposed to free crystalline silica dust; it differs in the radiologic picture, and also on postmortem examination of the affected lungs.

6. The more dusty processes are those involving the preparation and manipulation of the raw asbestos (other than crushing), dry cloth weaving, and mattress making.

7. The less dusty processes are spinning and similar processes, and processes, other than dry weaving, involving the manipulation of the spun yarn.

8. Further investigation is required to determine whether there is any increased susceptibility to the superven-

tion of pulmonary tuberculosis associated with the development of the asbestos fibrosis.

Much generous assistance has been received from many sources during the course of this inquiry, and for this acknowledgment is gratefully tendered.

In addition to those already referred to, the writer is beholden especially to Dr. S. A. Henry, who did much to facilitate the investigation, particularly by his detailed reports on various relevant matters, and by much organization and liaison work; to many others of his colleagues in the different districts, to a number of directors and officials of various firms, foremen, and employees; to Dr. W. E. Cooke of Wigan and Professor M. J. Stewart of Leeds for pathologic material and for much information as to the progress and results of their own researches into the gross anatomic and the histologic features of the asbestos fibrosis, and the constitution and location of the curious bodies; to Dr. MacGregor, Medical Officer of Health for Glasgow, for various clinical and radiologic facilities, and for his ever-present readiness to lend the aid of his Department, and especially to Dr. H. E. Seiler of his staff; to Dr. J. C. Robertson for the loan of a radiogram, together with detailed clinical notes of one patient and other valuable information; to Dr. W. H. Bateman for the loan of a radiogram, for some clinical histories, for obtaining and examining several specimens of sputum, and for other aid; and to Dr. J. Rennie, Chief Tuberculosis Officer at Sheffield, for a series of radiograms showing various stages of silicosis.

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