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OCCUPATIONAL DISEASE DIAGNOSIS

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I AM happy to have a few minutes to discuss the occupational diseases with a group of practicing physicians. Curiously enough, such opportunities are relatively infrequent since these diseases tend to be reserved, almost exclusively, for industrial health meetings. And few medical practitioners attend such meetings because the emphasis there tends to be on the industrial environment and its control rather than upon clinical medicine. The fact remains, however, that when a worker gets sick, he first consults his own doctor, so that this doctor is really in the front line when it comes to the need to recognize an occupational disease clinically and to make a correct diagnosis.

To the medical profession the occupational diseases are always somewhat baffling because they have their origins in environmental conditions which are unfamiliar. Unlike the community environment where the medical problems are always largely epidemiologic in character, the industrial environment, being man-made, presents problems in fields somewhat further removed from the physician's conventional training and experience, fields such as industrial toxicology or radiation.

There are literally thousands of potentially toxic chemicals which are either being produced in industry or are being used in the production of other goods, with new ones being added all the time by our research chemists. Aside from skin contact workers are exposed to many of these substances in the form of dusts, gases, fumes, vapors, or mists which become disseminated into the air of the workroom. While inhalation is by far the most important source of injury to health from exposure to toxic chemicals, skin absorption, and even ingestion may play important roles under certain circumstances.

Less familiar, perhaps, are a variety of other abnormalities in the working environment which are equally serious as potential health hazards. Aside from dangerous machinery there are abnormal temperatures and humidities; infrared and ultraviolet light, compressed air in tunnel construction; continuous vibration in the use of pneumatic tools; excessive noise, even in the supersonic ranges; radiation, as in radium dial painting operations, or the use of the x-ray or fluoroscope, as industrial inspection devices; inadequate or improper illumination with glare, etc. The industrial environment is, indeed, as complex and as fluid as modern technologic research and development itself.

For the individual worker health hazards will vary from industry to industry, from plant to plant, from workroom to workroom, and often from one process to another in a single workroom. Since labor is highly mobile, workers tend to go from one industry to another and from one type of work to another in various parts of the country. Accurate evaluation of their occupational exposures, particularly on any long-time basis, may therefore present difficulties.

For some reason the occupational diseases have always held a place somewhat removed from the rest of clinical medicine. The fact of their so-called "common etiology," the occupation, has perhaps carried with it a subtle implication that they represent a cohesive group in which there is a relatively limited number of fairly clear-cut clinical entities and that these are, on the whole, characterized by distinguishing earmarks which suggest particular occupations. Actually, the occupational diseases range over much of the entire field of clinical medicine since they reflect all of the physiologic, biochemic, and metabolic changes which develop in the body in response to working conditions which are inimical to health, including many compensatory responses initiated by the body for purposes of self-preservation.

Nor does the individual clinical picture necessarily suggest the existence of an occupational etiology. Far less does it point to any specific occupation as the etiologic factor. This is in part due to the fact that many of the same toxic chemicals and the same abnormal environmental conditions are to be found in greatly diverse industries and occupations. More important, however, is the fact that many occupational diseases are indistinguishable clinically from similar diseases which are nonoccupational in origin. Only the etiology is different. However, its recognition may provide the only opportunity for saving the patient's life.

Aplastic anemia or leukemia, for example, are the same hematologic entities whether they are idiopathic in origin or have been contracted in a factory workroom as a result of exposure to benzol or excessive radiation. Epitheliomata of the

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bladder produced by exposure to betanaphthylamine in the manufacture of aniline dyes cannot be differentiated from nonoccupational epitheliomata of the bladder, either clinically, on cystoscopic examination, or on microscopic section. Even their slow clinical course is the same. Asthma and dermatitis venenata, whether of occupational origin or not, are clinically the same. Only the precise antigen responsible may be one which is characteristic of a particular occupation, as in the case of the paraphenylene diamine dyes to which fur workers are exposed.

Some of the irritant gases present similar difficulties in differential diagnosis. Ammonia gas, for example, is so highly irritating that few persons would voluntarily remain in its presence long enough to inhale a toxic dose. An accidental break in a pipeline could subject workers to sufficient absorption to produce a pneumonitis, pneumonia, or even edema of the lungs, however. These conditions are not pathognomonic for ammonia gas. Only an occupational history will reveal what happened and provide necessary guidance for the management of the case.

The nitrous fumes from welding operations, especially in enclosed spaces as in the holds of ships, are more insidious because they are less obviously irritating. As a result, relatively large amounts may be inhaled without warning. However, after an interval of eight, ten, or even twelve hours their caustic action in the lungs may result in death from pulmonary edema. The long latent period tends to obscure causal relation unless one is familiar with this type of situation and inquires into the occupational exposure. Standard procedure for saving a worker's life is to put him to bed immediately following a known exposure to nitrous fumes, even in the complete absence of any clinical manifestations of injury to health.

In carbon tetrachloride poisoning one is usually dealing with a toxic hepatitis, a nephrosis, or a combination of both. There is nothing in the clinical picture, however, which specifically points to carbon tetrachloride as the etiologic agent or, indeed, suggests that one is dealing with some occupational disease. If the patient is jaundiced when he first consults his physician, obstructive, catarrhal, or infectious jaundice immediately come to mind. The possibility of a toxic hepatitis is usually the last to be considered and is often not considered at all. In the early stages, however, there may be no jaundice, and the patient comes to his physician complaining only of vague gastrointestinal symptoms. In the absence of an occupational history of exposure to a hepatotoxic chemical the patient may be given a cathartic and dismissed. By the time jaundice develops it may be too late to save his life. This has occurred.

In exposure to carbon tetrachloride one is particularly impressed by the great differences in individual susceptibility. A drink of alcohol may make the difference between life and death. These marked differences in individual susceptibility are not well understood. They are by no means confined to the occupational diseases but pervade the whole of clinical medicine. In a poliomyelitis epidemic, for example, almost everyone is exposed, but relatively few contract the disease. Nevertheless, physicians are prone to assume that if no one else in a plant is sick, his patient's illness cannot be due to his occupation. This is contrary to all experience. Of two workers having an identical exposure to benzol at a workbench one may die of an aplastic anemia and the other be unaffected.

Among the pneumoconioses silicosis is perhaps the most familiar. Physicians are becoming increasingly adept at recognizing the x-ray shadows of first, second, and third stage silicosis. Less familiar, perhaps, are the pneumoconioses produced under appropriate conditions of exposure by other types of dust such as asbestos, talc, carborundum, or mica. The extent to which there may be fundamental differences in pathologic response to dust inhalation is also not sufficiently appreciated. For example, asbestosis and the inhalation of certain of the chromate dusts appear to play etiologic roles in the production of lung cancer whereas silica dust does not. Iron dust will produce characteristic x-ray shadows of "siderosis" without apparently injuring the lungs at all. Pulmonary granulomatosis produced by the inhalation of beryllium indicates a totally different mechanism at work. Whatever the pulmonary pathology, however, and no matter how characteristic the x-ray shadows may be, it is pretty well accepted at the present time that the x-ray pictures are never pathognomonic. The resemblance between third stage silicosis and miliary tuberculosis may be truly striking; beryllium granulomatosis may be readily mistaken for Boeck's sarcoid. "More fibrosis than usual" or pleural calcifications are particularly difficult to interpret without a history of exposure.

Here again, then, an investigation of the occupational history of the patient is indispensable. To be of real value, however, this occupational history must be carried beyond the patient's present job to include the whole of his working life in so far as possible, for the pneumoconioses are chronic diseases which may take many years to develop. Occupational cancer or lead poisoning are in the same category. In addition, it is important to appreciate the fact that a particular

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job classification does not necessarily provide precise information as to the nature of the occupational exposure. To learn that one's patient is a "foundryman," for example, means little unless one knows whether he is a "moulder" who handles sand, whether he pours molten metals which are toxic, whether he is a sandblaster, or whether he operates an x-ray machine to detect flaws in finished castings. Accurate and complete occupational histories of patients are often difficult to obtain because of the technical complexities inherent in the subject and the failure of patients to present the whole story. Even a well-informed chemist who contracts an occupational disease in his laboratory will not necessarily give a complete list of all of the substances which he handles, although unlike most workers, he knows exactly what they are. He tends rather to be selective, listing only those of which he is suspicious. Memory failure for occupations years back obviously further complicates the problem, but if one persists, a useful occupational history is usually forthcoming.

When it is discovered that a patient has been working in a dusty atmosphere, there is a tendency to concentrate attention solely upon x-ray evidences of a pneumoconiosis, forgetting that many dusts never reach the lungs at all since the particles are larger than 5 microns in size; forgetting that such dusts may, nevertheless, be highly irritating to the nose and throat or to the upper respiratory passages; and forgetting further that it is the toxicologic properties of a substance rather than the portal of entry which tend to determine the site of injury in the body. Thus, inhalation of betanaphthylamine dust can cause cancer of the bladder but never cancer of the lungs. Similarly, lead, whether inhaled as dust or fumes, reaches the alveoli of the lungs, but it does not produce lung pathology. Instead, it is carried away in the blood stream and causes lead poisoning, a systemic disease. All of the industrial solvents enter the body by inhalation, but their toxicologic effects are elsewhere. Carbon tetrachloride has a predilection for the liver and kidneys, benzol for the hematopoietic system, carbon disulfide for the nervous system, etc. Zinc fumes cause a deposition of fine particles of zinc oxide on the mucous membranes of the upper respiratory passages to which there is an anaphylactic reaction, commonly referred to as "zinc chills."

Skin contact with toxic chemicals may, similarly, produce systemic disease with or without a local dermatitis when there is skin absorption. Unlike the inorganic lead compounds tetraethyl lead is readily absorbed through the skin and has a predilection for the central nervous system. Nitrobenzene on the skin can produce a dangerous cyanosis with methemoglobinemia. Where there is exposure to a combination of chemical substances, regardless of precise portals of entry, the principle of synergism enters to complicate the clinical picture further.

Among the gases carbon monoxide is always good for a full day's session as soon as someone raises the question of "chronic carbon monoxide poisoning." Actually, whether or not there is a clinical entity such as "chronic" carbon monoxide poisoning is entirely a matter of definition. That varying degrees of anoxia may be present in persons exposed to concentrations of this gas which are insufficient to produce acute asphyxia is well known to all students of the subject. In industry cases of acute asphyxia are relatively rare. More often, workers are exposed to relatively low concentrations for long periods of time. The typical picture, as one sees it in garage workers, for example, is that of an extremely intense and throbbing occipital headache, usually associated with dizziness, nausea, nervous instability, psychologic susceptibility, insomnia, and gastrointestinal symptoms. There are two striking points about the headache: (1) Clinically, it immediately suggests a brain tumor, and (2) it continues for a great many hours after the carbon monoxide has, obviously, been eliminated from the body. It is an important cause of absenteeism among garage workers. Yet it is this continuation of the headache, even into the following day, which always causes doubt to be thrown upon carbon monoxide as a possible cause. When one considers the pathology, this seemingly puzzling situation becomes perfectly clear. Trephine operations on animals exposed to carbon monoxide gas have demonstrated the presence of edema of the brain associated with increased intracranial pressure. This immediately explains the clinical similarity between the carbon monoxide headache and brain tumor. On the other hand, while carbon monoxide is rapidly eliminated from the body, usually within the first hour or two, edema of the brain takes many more hours to subside. The headache continues during all this long period. Incidentally, quantitative determinations of carbon monoxide in blood are often carried out hours after exposure when the patient finally reaches a hospital. Obviously, these can be of little value since practically all of the gas has long since disappeared.

Another point not sufficiently appreciated is that a worker with a 40 per cent oxygen displacement due to carbon monoxide is suffering from a far greater degree of anoxia for practical purposes than is an individual with a 60 per cent hemoglobia and a corresponding oxygen lack due to

anemia, for example. This is because the oxygen dissociation curve with carbon monoxide is such that what oxygen is present is far less readily available. A garage worker may collapse on sudden exertion under such circumstances, while the patient with the corresponding anemia still has a considerable ready reserve of oxygen at his immediate disposal.

I will not attempt to discuss lead poisoning except to point out that most of the difficulties in diagnosis arise from failure to differentiate between lead absorption and lead poisoning and failure to visualize the medical findings in terms of a constantly shifting metabolic balance between absorption, storage, and excretion. These difficulties are peculiarly characteristic of a whole group of occupational diseases which develop as a result of exposure to cumulative poisons. In an effort to be brief, one might say that diagnosis rests upon three pillars: (1) evidence of *significant* exposure, (2) a characteristic clinical picture, and (3) laboratory data, particularly quantitative data as to the lead content of the urine and the blood and the hematologic picture. Sound clinical medicine requires that the findings in all three categories must be interpreted together. The laboratory data cannot be interpreted without the others.

"Evidence of *significant* exposure" stresses the need for accurate information as to dosage. Dosage in industry is quite as important as in a doctor's prescription. The fact that a person works with a toxic chemical or that he has been exposed to radiation, for example, does not per se indicate that his health has been injured thereby. The question is always, "How much?" Frequently the only way in which this can be answered is by a technical evaluation of the industrial environment. This may require dust counts, chemical air analyses, radiation or noise measurements, or the application of a variety of other measuring sticks to the particular problem at hand. These findings can then be compared with accepted safe standards, the so-called Maximum Allowable Concentrations (M.A.C.), always taking into account individual differences in susceptibility among people. The practicing physician is obviously not equipped to make such tests. In New York State he can always call upon the Division of Industrial Hygiene in the Department of Labor for assistance. The services of its professional staff of physicians, chemists, and engineers are always at his disposal without charge.

In these days when everyone is psychiatrically minded, there is an increasing tendency to refer workers showing personality changes to a psychiatrist without stopping to consider the possibility of a toxic etiology, possibly occupational. There are a number of industrial chemicals which are capable of producing psychoneuroses, psychoses, and even neurologic conditions which cannot always be distinguished from those which are nonoccupational in origin. Least serious, perhaps, are some of the less toxic solvents which are, nevertheless, narcotic in their effects. Workers exposed to trichlorethylene, for example, may under certain circumstances become drowsy, irritable, and restless, and they may complain of vague gastrointestinal disturbances which are not easy to verify on physical examination. In the manufacture of rayon, workers exposed to toxic concentrations of carbon disulfide have developed mild to serious psychoneuroses and psychoses which for a long time were not recognized as being due to this exposure, and many were confined to psychiatric hospitals. A characteristic Parkinsonian syndrome may develop in manganese poisoning. In the manufacture of fur-felt hats workers are exposed to low concentrations of mercury for long periods of time. Here, instead of the severe renal damage with possible uremia which one sees in a patient who has swallowed some bichloride of mercury tablets, we find an intention tremor and remarkably complicated personality changes. The Mad Hatter in *Alice in Wonderland* is a true case of industrial mercury poisoning! And so, it is highly important at this time to caution against jumping to conclusions that what the patient needs is a psychiatrist.

Summary

In conclusion I would say that most patients are workers. Few workers nowadays succeed in escaping the hazards to health which modern technology has put in their path. Toxic chemicals not only are to be found in industry, but they are used in the home and in many hobbies as well. Carbon tetrachloride is a popular cleaning fluid with the housewife; benzol is a common constituent of rubber cements, and potentially toxic moth proofing materials and insecticides are in common use by almost everyone. For that reason, a history of exposure is of the essence. One must never fail to consider the possibility of an occupational etiology in every differential diagnosis.

I wish to stress again the fact that ascertaining the occupational etiology in a given case, where it exists, may be the only means of saving the patient's life, since no amount of treatment will be effective unless he can be immediately removed from further exposure. Beyond that I would like to direct your attention to the fact that recognition of the occupational etiology contributes to proper reporting of occupational diseases, thereby making more accurate the

occupational disease statistics available. Very few people realize that we have no reliable data at the present time as to the statistical incidence of any of the occupational diseases, principally because physicians fail to recognize them. With the best intentions in the world a physician will not report a case of "aplastic anemia" if it does not occur to him that it is occupational in origin. Practicing physicians, therefore, have a great contribution to make, as individuals, in the matter of prevention in that they can provide public health agencies with accurate morbidity and mortality statistics which are the building stones of all public health programs.

80 CENTRE STREET

ANNOUNCEMENT

PRIZE ESSAYS

The Lucien Howe Prize and the Merrit H. Cash Prize will be open for competition at the next Annual Meeting of the Medical Society of the State of New York, May 4 to 8, 1953, in Buffalo.

The *Lucien Howe Prize* of $100 will be presented for the best original contribution on some branch of surgery, preferably ophthalmology. The author need not be a member of the Medical Society of the State of New York.

The *Merrit H. Cash Prize* of $100 will be given to the author of the best original essay on some medical or surgical subject. Competition is limited to the members of the Medical Society of the State of New York who at the time of the competition are residents of New York State.

The following conditions must be observed:

Essays shall be typewritten or printed with the name of the prize for which the essay is submitted, and the only means of identification of the author shall be a motto or other device. The essay shall be accompanied by a sealed envelope having on the outside the same motto or device and containing the name and address of the writer.

If the Committee considers that no essay or contribution is worthy of a prize, it will not be awarded.

Any essay that may win a prize automatically becomes the property of the Medical Society of the State of New York, "to be published as it may direct."

All essays must be presented not later than *February 2, 1953*, and sent to the Chairman of the Committee on Prize Essays of the Medical Society of the State of New York, 386 Fourth Avenue, New York 16, New York.

MATHER CLEVELAND, M.D., *Chairman*
Committee on Prize Essays