OCCUPATIONAL MEDICINE SYMPOSIA

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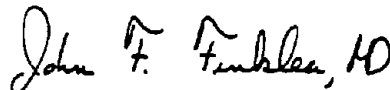
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iii

FOREWORD

A primary goal of the National Institute for Occupational Safety and
Health is to promote effective health care for American working men
and women.

Since the passage of the Occupational Safety and Health Act of 1970,
a growing number of family practitioners and internists have entered
the part-time practice of occupational medicine. Helping these
physicians to incorporate the concepts and skills of occupational
medical practice into the total delivery of health services is a
major consideration of the Institute's Division of Occupational
Health Programs. Toward this end, NIOSH co-sponsored the AMA's
34th Annual Congress on Occupational Health and cooperated in
developing the scientific program for the Congress.

The seven symposia of the Congress were presented by prominent and
eminently qualified speakers from a variety of medical specialties,
industrial hygiene, and engineering. The program was a unique and
comprehensive review of selected areas of occupational medical
practice. NIOSH is publishing the major papers presented at the
Congress in this text in order to bring the benefits of this unusual
program to as many individuals in the field as possible.

John F. Finklea, M.D.
Director, National Institute for
  Occupational Safety and Health

PREFACE

The enthusiastic response of participants to the program presented at the AMA 34th Annual Congress on Occupational Health, co-sponsored by the National Institute for Occupational Safety and Health, led to NIOSH's publication of these symposia. It is hoped that this reference text will stimulate the interest of physicians in the particular opportunities and problems of occupational medical practice, as well as meet the needs of the growing number of physicians who find occupational health considerations now a part of their practice.

The manuscript was prepared from verbatim transcripts of the Congress proceedings. Papers are published essentially as presented. However, they were edited by NIOSH for written presentation and titles were assigned to facilitate the use of the publication. Also, an Editor's note has been inserted to introduce the papers in Symposium II and to clarify references made by the speakers to NIOSH Programs. Except for NIOSH speakers, the papers do not necessarily represent the views of NIOSH but are presented as an introduction to the practice and controversies of occupational medicine.

The Congress also included lively and informative question and answer sessions. However, it was not possible to transcribe these sessions in this text due to their length and informality. The Division of Occupational Health Programs has a limited number of transcripts of this part of the Congress and will send individual copies on request while supplies last.

Marilyn K. Hutchison, M.D.
Acting Director, Division of
Occupational Health Programs

v

TABLE OF CONTENTS

VII

OCCUPATIONAL MEDICINE SYMPOSIA

ABSTRACT

Through a survey of AMA-member physicians, NIOSH determined the primary occupational medicine problems and information needs of private practitioners with part-time occupational medicine responsibilities. The results of the survey were used to develop the program of the 1974 AMA Congress on Occupational Health, co-sponsored by NIOSH. Papers were presented on the following topics: Occupational pulmonary diseases; chest X-rays for the detection of pneumoconioses; cardiovascular disease in occupational medicine; work evaluation and stress testing; medical monitoring for occupational disease; chemical exposures; noise, hearing, and audiometry, and environmental measurement techniques. The papers presented at the meeting, published by NIOSH, are an introductory text, highlighting the aspects of occupational medicine most significant to part-time plant physicians, to private practitioners generally, and to others with related interests.

INTRODUCTION

The Occupational Safety and Health Act of 1970 directed the attention of many elements of American society, including the health professions, to the health and safety problems of Americans on the job.

In its efforts to promote effective health care for American working men and women, the National Institute for Occupational Safety and Health (NIOSH) has developed programs to assist the assimilation of occupational medicine concepts and skills into general health care delivery.

The exact number of physicians who work in occupational medicine is not known, but there are indications that the number of physicians with part-time industrial practice is increasing. Those who seek specialized information often find that appropriate educational opportunities are limited.

Accordingly, NIOSH's Division of Occupational Health Programs surveyed 800 AMA member physicians to identify their specific interests in continuing education related to occupational medicine. The physicians surveyed were chosen on the basis of AMA professional records indicating at least some time spent in occupational medicine. Topics which were ranked highest by respondents included occupational pulmonary diseases, cardiovascular diseases, medical minotoring, and toxicology. This information has been added to the criteria used by NIOSH in evaluating the direction, impact, and content of occupational health programs.

Specifically, the interests revealed by the 800 part-time occupational health physicians surveyed have led to NIOSH programs designed to promote the exchange of pertinent and practical information for the health practitioner. In one such activity, NIOSH co-sponsored the AMA's 34th Congress on Occupational Health, September 9-10, 1974, Chicago, Illinois. This prestigious annual event is one of the few sources of continuing professional education in occupational medicine available to medical practitioners.

The results of the survey were used in developing the scientific program for the Congress in order to include subjects of high priority for prospective attendees, in particular, those with limited occupational medicine training and/or part-time industrial practice.

In addition, the Congress was designed as a workshop in order to allow optimum audience participation and to facilitate responsive, practical communication through question and answer sessions.

The two-day conference consisted of seven symposia. Papers presented at the Congress have been assembled for publication by NIOSH to serve as a reference text for physicians and other working in occupational health.

OCCUPATIONAL PULMONARY DISEASES

HYPERSENSITIVITY PNEUMONITIS
Howard A. Buechner, M.D.

Of the two big categories of lung disease due to the inhalation of dust, damage to the lungs caused by inorganic dust has been known for centuries mainly in the form of silicosis. Doctor VanOrdstrand will deal with that category of illnesses in the next presentation. I will discuss those diseases of the lungs produced by the inhalation of organic dust.

One such disease, bronchial asthma, you know well. And, since this syndrome, caused by the inhalation of the various organic dusts in the form of pollen or house dust, is so well known, I will make no further reference to it. I will confine my remarks to a type of illness caused by the inhalation of organic dusts, which is known as hypersensitivity pneumonitis.

In the English literature this type of illness is called extrinsic allergic alveolitis. This type of disease differs from bronchial asthma in that it produces infiltrative lesions in the lungs, or pneumonitis. Bronchial asthma does not do that. It is an area of pulmonary disease which may not be familiar to many physicians, largely because the pathological process was not recognized until relatively recently. However, since its recognition, the list of such disorders has grown tremendously. Many of the hypersensitivity pneumonias are associated with occupations-- some rather important occupations such as farming. The following table will give you an idea of what we are talking about.

TABLE 1--ETIOLOGIC AGENTS IN HYPERSENSITIVITY PNEUMONITIS

DISEASE	EXPOSURE	ANTIGEN
Farmer's lung	Moldy hay or grain	Micropolyspora faeni, Thermoactinomyces vulgaris, Thermoactinomyces saccharii.
Bagassosis	Stored sugar cane fiber (bagasse)	Same as above.
Mushroom picker's disease	Moldy vegetable compost.	Same as above.

J-1

TABLE 1--ETIOLOGIC AGENTS IN HYPERSENSITIVITY PNEUMONITIS--Continued

DISEASE	EXPOSURE	ANTIGEN
Humidifier or air conditioner, heating system disease.	Contaminated forced-air system.	Same as above.
Fog fever (cattle)	Moldy hay	Same as above.
Maple bark stripper's disease.	Maple tree logs or bark	Cryptostroma corticale.
Sequoiosis	Redwood sawdust.	Graphium, Pullularia, and other fungi
Suberosis	Moldy cork dust	Penicillium sp.
Paper Mill worker's disease	Moldy wood pulp	Alternaria.
Pulp wood handler's disease	Moldy wood pulp	Same as above.
Brewer's or malt worker's lung	Malt or barley dust.	Aspergillus clavatus.
Cheese washer's lung	Cheese Mold	Penicillium caseii.
Paprika slicer's disease	Moldy paprika pods	Mucor stolonifer.
Wheat thresher's lung	Wheat flour containing weevils.	Sitophilus granarius.
Pigeon breeder's disease	Pigeon serum and droppings.	Avian proteins.
Budgerigar fancier's disease	Contact with parakeets.	Parakeet proteins.
Chicken handler's or feather plucker's disease	Contact with chickens.	Chicken proteins.
Turkey handler's disease	Contact with turkeys.	Turkey proteins.
Pituitary snuff disease	Porcine, bovine. pituitary gland (Pitressin snuff)	Porcine, bovine proteins
Smallpox handler's lung	Smallpox scabs	Unknown.
Thatched roof disease	Dried grass and leaves.	Unknown.
Tobacco grower's disease	Tobacco plants	Unknown.
Joiner's disease	Sawdust	Unknown.
Tea grower's disease	Tea plants	Unknown.

Bible printer's disease	Moldy type . . . . Unknown.
	setting water.
Coptic or mummy disease	Cloth wrappings of . . . . . Unknown.
	mummies
Detergent disease (asthmatic-	Enzyme detergents . . . . . Bacillus subtilis.
like symptoms; true pneumonitis	
not identified).	

We are talking about diseases produced by antigens caused by microorganisms known as actino-mycetes, fungi, and various types of foreign protein. Here are some of the names of the diseases we will be talking about: farmer's lung; suberosis, which is due to the inhalation of cork dust; mushroom worker's lung; and bagassosis, which I will talk about in detail. (This disease is caused by the inhalation of dust from dried sugar cane fiber.); cheese washer's lung; sequoisis, due to the inhalation of dust from redwood trees; maple bark stripper's lung; malt worker's lung; wheat weevil disease; pigeon breeder's lung; and pituitary snuff disease.

These are some of the diseases. There are many more, and Table 1 gives you a complete listing. In the right hand column are the antigens. At the top of the column are the actinomycetes. Below are various types of fungi, some of which you may be familiar with such as aspergillus. There is even an insect, the wheat weevil, which causes some illnesses of this type. The last illness is not properly included, detergent lung, some detergents, those made out of certain enzymes from bacterial products, produce a disease more like asthma. The foreign proteins, usually of animal origin, which produce a variety of illnesses which will be covered in detail later.

From here on I will talk more or less specifically about the illness bagassosis. My reason for doing so is two-fold. First of all, it is the disease with which I personally have had the most experience. Secondly, we may use it as a model. Whatever I say about bagassosis can be applied to all of the other hypersensitivity pneumonias. They're all basically the same. So keep in mind that although I am talking specifically about one disease, the information can be applied to all these other illnesses.

The disease bagassosis derives its name from bagasse, the name given dried sugar cane fiber. It is the dust from the sugar cane fiber, which is the vehicle for the offending agent.

In the early days of the sugar cane industry in Louisiana, bagasse was considered worthless and was discarded. The very name bagasse is a French word meaning something worthless. The material was then used for fuel to stoke the boilers of the sugar cane mills. After a while its immense industrial value was recognized, and now it's used in the manufacture of paper, wallboard, or celotex, and building materials like window frames and doors. It goes into the production of heavy mud, used in oil fields. Various synthetic materials similar to plastics are made from it; also insulating material resembling tar paper. It's used for garden mulch, in cattle feed, and even in the manufacture of explosives.

The many industries based on handling this material are important and widespread throughout the world. So people do have to work with this material. However, if a worker is exposed to the dust, he developes an illness which is very similar to ordinary bacterial pneumonia. Some of the typical symptoms are shortness of breath, fever, chills, coughing, chest pains, and weakness--the typical symptoms of bacterial pneumonia. The other two symptoms of the disease, poor appetite and very rapid weight loss, differ from a pneumococcal pneumonia.

X-rays of bagassosis show a variety of roentgenographic patterns. In the acute phase of the illness, there is pulmonary edema plus an inflammatory reaction in the lung, and X-rays show widespread bilateral symmetrical lesions in the lungs which have the general appearance and distribution of pulmonary edema. In other patients there is a butterfly pattern of lesions in the lung. In some X-rays you can see a bit of an air bronchogram, and this implies consolidation

within the lung or filling of the alveoli with edema fluid. There are many patterns. Some X-rays show tiny nodular deposits throughout the lung which have somewhat the appearance of miliary tuberculosis, with which bagassosis has been confused.

The first recognized case of bagassosis occurred in a young black male brought into Charity Hospital in New Orleans in 1937. He had fever, chills, and weight loss, and was thought to have miliary tuberculosis. That was before the days of chemotherapy, so he was placed in the back ward and more or less expected to die. He fooled his doctors, however, and gradually recovered. They began to wonder what disease he did have, and when it was found that he worked in handling bagasse, further studies identified the disease. The physician who discovered the disease was Dr. Oscar Blitz. Although he collected the original cases, he did not report them in the literature, and his name has not been associated with the disease.

The disease is usually bilateral and symmetrical but, it may be unilateral, and it may involve any portion of the lung. This is true of all the other hypersensitivity pneumonias. If the patient has had recurrent illnesses due to exposure to the dust for many years, bagassosis will become chronic; however, it may also become chronic in patients who have never been ill, having been exposed to rather low doses of antigen over a long time. Chronic bagassosis, which can be lethal, results in diffuse pulmonary fibrosis with fibrotic streaks running out through the lung field with interspersed areas of emphysema. X-rays reveal the big heart shadow, cardiomegaly; the heart is shaggy due to the interstitial fibrosis. All of the hypersensitivity pneumonias, if the patient is reexposed repeatedly over a long period of time, will result in extensive pulmonary fibrosis.

It took real detective work to discover the true etiology of bagassosis. We knew it was due to exposure to bagasse, but what characteristic of the dust caused the disease? There were sugar cane producers from which the disease has been reported. And, there were countries which do not produce sugar cane, but from which the disease has been reported.

In the beginning the disease was discovered in Louisiana. Then cases appeared in Texas and Missouri, and in England. In every case we found that Louisiana bagasse had been shipped to these areas and used for manufacturing purposes. We thought there was something peculiar to Louisiana sugar cane that caused the disease, but then cases were reported from Italy, India, Peru, and Puerto Rico. It became obvious that the problem was not limited to Louisiana bagasse but could occur wherever sugar cane was grown. But the absence of bagassosis in the other great sugar cane regions of the world was unexplained.

Then, we realized that almost invariably the disease did not occur simply in areas where sugar cane was grown but more or less exclusively in areas where the bagasse was used for manufacturing. So there was something about the manufacturing process accounting for the etiology of the disease. The situation was exemplified in Louisiana. All sugar cane mills produce bagasse. But most had never had a case of Bagassosis. Nearly all of our cases were coming out of two manufacturing plants, a wallboard plant, and a paper plant. However, there were two sugar cane mills that did have cases of the disease. What we found is that the plants that produced bagasse but shipped it away while it was still fresh had no bagassosis cases. But if plants baled it and stored it and then rehandled the old material, their workers got bagassosis.

Bagasse is often stored in house-like stacks, left out in the fields for weeks or months. While it is out there, it is exposed to extreme heat and moisture. Something happens to it when it is out there. Later, when it is brought back to the manufacturing plants and the bales are broken open and shredded, the workers who perform this function, the bale breakers, are the ones subject to bagassosis.

A beautiful little epidemiological study was made in Puerto Rico. A sugar producing plant in Arecibo, Puerto Rico, had been producing sugar for many years. They had been discarding or burning the bagasse and never had any trouble. Then they found out it was valuable so they built a paper mill next to the plant. The first year as the bagasse was produced fresh, it was taken on a conveyor belt over to the paper mill and everything went well but the bagasse was being produced faster than the paper mill could use it. So the excess was baled and stored out in the

field. Later they ran out of fresh bagasse and began to bring the material in from the fields and break open the bales. Immediately they had an outbreak, some hundred cases of bagassosis.

So what is it about the stored material that is dangerous? We suspected that it was a microorganism that grew in the stored material, and we cultured it and found innumerable organisms, various types of saprophytic bacteria and many types of fungi, but they were always organisms which had not at that time been known to produce this type of illness. Only when we cultured at temperatures ranging up to 57 or 60 degrees centigrade did we begin to grow out previously unrecognized organisms, the so-called heat loving or thermophilic actinomycetes. These organisms are still in process of classification. There have been many changes in their names as you can see here. This organism is now known as <u>thermoactinomyces</u> <u>saccharii</u>, a name given it within the past year. It is the principal organism that produces bagassosis. It's the inhalation of spores from this organism which set up a hypersensitivity in the lung that causes the Bagassosis. Another thermophilic actinomycete is the principal offending agent in the disease known as Farmer's Lung.

What are these organisms? They are a group of branching unicellular organisms which reproduce by fission or by means of spores. They form a borderline system considered by some as bacteria, by others as fungi or lower fungi. Some put them in a group by themselves. But most taxonomists now classify them as bacteria and not fungi. They are considered high-type bacteria. There are some technical reasons why they are classified as bacteria rather than fungi. The main criterion is that they show sensitivity to antibacterial antibiotics rather than to antifungal antibiotics. They're related to the actinomyces organisms which produce actinomycosis and to the organisms which produce nocardiosis. Although you will find actinomycosis and nocardiosis listed in any book on fungus diseases, they are not fungus diseases. They are bacterial diseases which have been misconstrued through the years. Once again the outstanding thing about these organisms is that they respond to ordinary antibacterial antibiotics rather than to antibiotics like amphotericin. They are high-type bacteria.

Immunologically it has been proved that Bagassosis and disorders similar to it are indeed allergic diseases. Some kind of antigen-antibody reaction takes place, but only in relation to old bagasse. As things progressed, we analyzed the bagasse, and we began to find the specific organisms. And, extracts from the specific organism produce the precipitin bands which show that the reaction is due to the specific microorganism.

Pathologically Hypersensitivity Pneumonitis consists of interstitial granulomatous changes in the lung, interstitial granulomatous pneumonitis (slides presented) Here is a granuloma with many plasma cells in it. Here's a remnant of an alveolar wall showing the rest of the lung is destroyed by granuloma formation. This is a typical reaction. The tissue reaction is characterized by infiltration with mononuclear cells and particularly plasma cells from which the antibody is apparently produced. The alveolar walls thicken and are infiltrated with inflammatory cells but in the alveolar spaces there are characteristically collections of large macrocytes with vacuolated cytoplasm, known as foam cells. We see this in nearly every disease of this type.

What is the incidence of the disease? In the beginning there wasn't much of it or we didn't recognize it. Between 1937 and 1958, there were only 62 cases reported in the U.S. and then between 1959 and 1963 due to the outbreak in Puerto Rico, there were about 200 cases. As time went by the United States, particularly Louisiana, began to report more cases, and a few years ago there were about 500 cases. Spain and the Philippines had joined the list of places from which the illness was reported. For every case reported, there must be 10 or 20 other cases that never come to the attention of doctors who write papers.

The known hypersensitivity pneumonitides are listed in Table 1. At the top is Farmer's Lung; the first one to be discovered, it is caused by exposure to moldy hay or grain. Fog fever in cattle is the same disease as Farmer's Lung. It occurs in cattle that eat and breathe the spores from moldy hay. Bagassosis, as I have said, is due to exposure to moldy sugar cane fibers containing the <u>thermophyllic</u> <u>actinomycetes</u>. Mushroom picker's disease occurs in workers who grow mushrooms commercially and is due to the same <u>thermophyllic</u> <u>actinomycetes</u>. Maple bark

strippers disease occurs in loggers who cut down and strip bark from maple trees which are infested by a true fungus called corticale. Sequoiosis occurs in workers who cut redwood and are exposed to moldy sawdust which contains various fungi.

Wheat thresher's lung is due to inhaling the products of the wheat weevil, which infests the grain. (There are probably other fungi that infest wheat. Just the other day, I read that a shipment of wheat going to Russia was turned back because it was infested with some kind of fungus. Maybe that fungus too can cause diseases of this type.)

Pigeon breeder's disease occurs in pigeon fanciers. It's due to the pigeon serum or some product in the pigeon feathers. Budgerigar fancier's disease is due to contact with parakeets. Parakeets are very popular in England, and the disease is due to the parakeet protein. Pituitary snuff disease occurs in people with diabetes insipidus who use pitressin in the form of pituitary powder; this disease is due to protein. Smallpox handler's lung due to exposure to smallpox scabs--I don't know this disease, but it is listed. Thatched roof disease, which occurs in New Guinea, is due to organisms which live in the dried grass and leaves from which the roofs of the houses are made. The specific antigen is unknown.

All these diseases sound exotic, except perhaps for Farmer's Lung. However, the disease hits closer to home in air conditioner's disease. Illness of this type has been found in housewives and office workers. It's been discovered that the thermophilic actinomycetes may grow in air conditioning systems and in heating systems, particularly where humidifiers are employed. So your own air conditioner or your own heating system may be the source. You can contract the disease in this room or in your office or your home. More and more cases are being discovered from contact with heating or air conditioning systems.

Then there is detergent disease to which I referred earlier. It occurs in employees of detergent industries which use bacterial products to produce enzyme detergents. The workers develop a respiratory illness which is asthma-like. Probably, Detergent disease does not belong in this category, but it is an occupational illness.

Malt worker's lung or brewer's lung caused by exposure to moldy barley, is really due to an aspergillus. Paper mill workers, those who work in mills that process pulp have recently been added to the list of afflicted workers. Any worker who deals with pulp wood may be exposed if the logs and the bark are moldy. There are many different molds other than that which causes Maple bark disease, so anybody who works with pulp wood is subject to hypersensitivity pneumonitis.

Suberosis is due to cork dust; cheese washer's lung due, to a penicillium, paprika slicer's disease occurs in middle Europe where workers slice open moldy paprika pods; the organism has now been identified as a mucor species. People who work with tobacco leaves are subject to tobacco growers disease. Bible printers disease which occurs in Europe, where apparently the water printers use to wash type becomes moldy. Now here is a way out one--mummy disease, which occurs in people who unwrap the linen from mummies and the linen is moldy. Joiner's disease, joiners are simply English carpenters, and these men are exposed to moldy sawdust. Feather plucker's disease is also known as duck fever. It's assuming importance now because poultry workers who deal with chickens, ducks, and turkeys have all been now found to be subject to hypersensitivity pneumonia. In the July 1974 issue of The American Review of Respiratory Diseases are two articles, one on chicken workers who developed the disease, the other on turkey workers. Sixty percent of the workers who are involved with raising turkeys are said to develop respiratory illnesses, usually like asthma. But about 6 percent of the workers develop hypersensitivity pneumonias, and some go on to pulmonary fibrosis.

That's the list of all the known diseases caused by inhalation of organic dust. But the important thing is that inhalation of any type of organic allergen has a potential for producing disease. This list of illnesses has grown tremendously in the last 5 years, but there are probably many other disorders of this type that are still unidentified. And I think the list will continue to grow and grow. The whole problem is emerging as a very important area in pulmonary medicine.

I'll emphasize one last thing. All chest physicians from time to time see people with diffuse pulmonary fibrosis. We call it diffuse idiopathic pulmonary fibrosis, the Hamman-Rich syndrome, and nobody knows what causes it. I feel that many of these people are suffering from the chronic form of hypersensitivity pneumonia. They are allergic to something. They have been exposed to an unidentified antigen for many years. They have never been acutely ill. We see them late in the course of the disease, and we take an X-ray. Their lungs are full of fibrosis and we don't know why. I think as the years go by, we will begin to identify the etiology for more and more of these patients. We know that many of the hypersensitivity pneumonias do result in interstitial pulmonary fibrosis, particularly in those people who have parakeets. I think we will gradually discover that these illnesses may be the basis for many patients with so-called idiopathic pulmonary fibrosis.

OCCUPATIONAL PULMONARY DISEASES

SILICOSIS
Howard S. VanOrstrand, M.D.

Howard S. VanOrdstrand, M.D.: My own classification of the pneumoconioses related to inorganic dust is broken down according to the dusts: 1) those that are non-disabling or benign, or inert and non-fibrogenic, for example, iron oxide, tin oxide, and barium sulfate (barite) and 2) those which may be disabling or even fatal, the fibrogenic dusts.

TABLE 1--OCCUPATIONAL LUNG DISEASES DUE TO INORGANIC DUSTS

I. Pneumoconioses that are non-disabling (so-called "Benign," inert non-fibrogenic dusts):

    1. Siderosis . . . . iron oxide
    2. Stannosis . . . . tin oxide
    3. Baritosis . . . . barium sulfate (barite)

II. Pneumoconioses which may be disabling and even fatal:

    1. Silicosis . . . . crystalline silica
    2. Asbestosis . . . . asbestos
    3. Berylliosis . . . . beryllium
    4. Coal Worker's Pneumoconiosis
    5. Shaver's Disease. . . . . bauxite
    6. Talcosis . . . . talc
    7. Cristobalite Silicosis
    8. Diatomaceous Earth Pneumoconiosis

The fibrogenic dusts include silica, which produces silicosis, one of the oldest diseases known to man. Yet, amazingly, the pathogeneses of success is still not completely understood. There have been at least a hundred theories about how crystalline silica reacts with lung tissue, whether it is chemical, mechanical, or electrical. It may eventually turn out to be an

immunological disorder, because we have known for years that people working side-by-side with the same exposure react considerably differently to that exposure. Also included in this second group are dusts from asbestos, beryllium (if you will agree that berylliosis is a pneumoconiosis), coal, bauxite (associated with Shaver's disease), talc, cristobalite (another type of silica), and diatomaceous earth.

I would like to emphasize that you must include in your medical history not only the type of industry but also the specific job of the worker in that industry. Needless to say, the general industrial category may reveal minimal exposure, while a detailed history from the worker will often reveal multiple occupations within the general category as well as the length of time spent at each occupation.

TABLE 2--THE VARIETY OF OCCUPATIONS ASSOCIATED WITH THREE PNEUMOCONIOSES DUE TO INORGANIC DUSTS

ASBESTOSIS	BARITOSIS	BERYLLIOSIS
Asbestos weaver	Barite miner	Alloy maker
Auto mechanic	Barite miller	Bronze maker
Brake manufacturer	Ceramics worker	Ceramics worker
Carpenter	Glassmaker	Electronic tube maker
Clutch manufacturer	Paint maker	Extraction worker
Filter maker	Rubber worker	Fluorescent lamp maker
Floor tilemaker	Well driller	Metallurgist
Insulator		Missile worker
Lagger		Neon sign maker
Millworker		Nuclear energy worker
Miner		Phosphor maker
Roofer		Propellant maker
Shingle maker		Toxicologist
Shipbuilder		X-ray tube maker
Steamfitter		

For example, asbestosis is known to occur in textile weavers, auto mechanics, brake manufacturers, carpenters, (See Table 2). Likewise, berylliosis affects more than the miner of the ore and the founder of the metal. (The fluorescent lamp industry no longer uses beryllium; it was removed as a phosphor in 1949, not because it was uncontrollable during the manufacture of the lamps but because disposal of the burned-out tubes presented a hazard in the home and other industries.)

We certainly need to consider the specific occupational coal miners, there are many jobs, both underground and above ground, where the risk of pneumoconiosis is present (See Table 3).

Table 3-- COAL MINING OCCUPATIONS

Underground Occupations

Face:
- Face Cutting Machine Operators
- Hand Loaders
- Timber Men
- Roof Bolters
- Loading Machine Operators
- Drillers Blasters
- Shot Firers

Maintenance:
- Timber Repairmen
- Bonders
- Track Men
- Mechanics
- Rock Dusters
- Ventilation Men

Transportation:
- Motormen
- Brakemen
- Belt Men
- Boom Boys
- Rope Riders
- Couplers
- Shuttle Car Operators

Miscellaneous:
- Powder Men
- Fire Bosses
- Engineers
- Safety Directors
- Dispatchers

Surface Occupations

Strip Mining:
- Bulldozer Operators
- Power Shovel Operators
- Truck Drivers
- Drillers
- Scraper Operators
- Shot Firers

Tipple:
- Mine Car Dumpers
- Car Couplers
- Switchmen
- Tipple Operators
- Crushers

Transportation:
- Hoist Men
- Aerial Tram Operators
- Motormen
- Brakemen
- Coal Truck Drivers

Maintenance:
- Electricians
- Mechanics
- Carpenters
- Welders
- Blacksmiths
- Plumbers

Miscellaneous:
- Office Workers
- Samplers
- Engineers
- Outside Foremen

Some of these jobs have much more exposure to silica than others; face workers (e.g. cutters, loaders, timber men, and so forth) in particular operate in a very dusty atmosphere. While transportation men encounter a considerable exposure to silica in the process of sanding the track to improve traction.

So too with the surface mining operations, the extent of exposure to silica is greatest with the rock breakers and progressively less through the miscellaneous workers.

Other occupations associated with pneumoconioses are listed in Table 4. I don't think Shaver's disease occurs any more, but it was an interesting model of a well studied occupational disease. Unfortunately, we still don't know whether it is related to aluminum or silica.

TABLE 4--OCCUPATIONS ASSOCIATED WITH OTHER INORGANIC PNEUMOCONIOSES

DIATOMACEOUS EARTH PNEUMOCONIOSIS:
Workers exposed to calcining of
    diatomaceous earth

KAOLINOSIS:
Brick maker
Ceramics worker
China maker
Miner
Paper maker
Potter

SHAVER'S DISEASE:
Abrasives makers exposed to the
    heat treatment of the ore bauxite

SIDEROSIS :
Demolition man
Fettler
Flame Cutter
Foundryman
Grinder
Metal worker
Metalizer
Polisher
Scarfer
Shipbreaker
Welder

SILICOSIS:
Abrasives worker
Bricklayer
Brickmaker
Ceramics worker
Coal miner
Diatomite worker
Enameller
Fettler
Filter maker
Foundryman
Glassmaker
Insulation worker
Miner
Motorman
Polisher
Sandblaster
Shot blaster
Stonecutter
Stonedresser
Stone driller
Tunnel driver
Quarry man

STANNOSIS:
Workers involved in the
    salvage of tin

TALCOSIS :
Cable maker
Ceramics worker
Cosmetic worker
Miner
Paper maker
Plastics worker
Rubber worker

Dr. Cecil Shaver, of St. Catherine's, Ontario, first described this disease in workers on the Canadian side of the Niagara Peninsula who were making crude abrasives from bauxite. The workers developed spontaneous pneumothorax. The proper hooding and ventilation of the operation has brought the situation under control, as it should in most industrial health problems.

Siderosis also arises from multiple occupational exposures, not simply from welding, but also from work environments that generate iron dust, particularly metal grinders, polishers, scarfers, and shipbreakers.

Silicosis too affects a variety of workers, including abrasive workers and bricklayers. When you think of brick laying in terms of siliceous exposure, it is mainly in the repair of open hearths where the brick for many years was essentially 100 percent silica.

My main experience with talcosis has been with rubber workers; talc or soapstone was formerly used to prevent the inner tubes from sticking to the tires. It is also seen in other industries, for example, in the cosmetics industry.

The work of Doctor Gough may be familiar to all of you in pathology of inorganic dusts. Gough was for many years professor of pathology at Cardiff, Wales, Medical School; he is one of the outstanding pathologists of the world on the subject of pneumoconioses. He thought that looking through the microscope, as we normally do, was too close to the picture; that, instead,

we should stand back and look at a gross lung. He developed a technique for preparing a total thin sagittal section of the lung at postmortem.

Gough's sections reveal clearly the gross pathology of a normal lung, or of extra-pulmonary disease, or of an early, mild and simple coal worker's pneumoconiosis. The collections of dust round the little bronchial arterioles with early, small focal emphysematous changes is clearly revealed. In other mild degrees of coal worker's pneumoconiosis, except where early fibrosis is shown by Gough's technique, you see an increasing amount of focal emphysema. Complicating tuberculosis with cavitation superimposed on the coal worker's pneumoconiosis is also found.

The classic picture of silicosis in a rock worker as shown by the Gough technique reveals hyalinized whorls quite different from the picture of coal worker's pneumoconiosis. Pneumoconiosis often involves an infarct, for examples the massive fibrosis of coal workers described originally by the British or Welsh--massive changes in the X-ray superimposed on diffuse fine or medium size nodulation.

Let's go on now to review some examples of inorganic dust reactions. This X-ray reveals a diffuse, fine nodulation that was called silicosis. (slide presented). So often the radiologist is handicapped by not having the work history.

This patient had a lung biopsy done at the same time as a pericardial biopsy; the pericardial biopsy was the major reason for checking on him, and he did have tuberculous pericarditis, according to the biopsy. However, he was a welder who never had any exposure of consequence to silica or to any mixed dust. The dust inhaled was pure iron oxide, and it is revealed in the biopsy by a stain for the iron oxide. Iron oxide has been shown to be one of the inert dusts. It does not cause disturbance to the pulmonary physiology.

A few other examples of iron oxide: A very extensive and rather large nodulation occurred in a man working for 50 or 60 years making wires for fencing material. He's now 80 years of age and completely asymptomatic; while he has a terrible looking X-ray, his pulmonary function is normal. The special biopsy stain reacts with tin oxide and iron oxide which are not engulfed in phagocytes. Aggregates of phagocytes clumped together cause the nodule in the X-ray Stannic oxide pneumoconiosis produced this terrible looking X-ray. (Slide presented) He died at 80 years of age from a carcinoma of the prostate; he never had any pulmonary symptoms or lung disability. A microscopic view of the same patient reveals tin oxide which caused no fibrosis, just massive shadows on the X-ray due to clumps of phagocytes.

The characteristic asbestos body is shown under high power in the biopsy of a man who developed asbestosis after working in the wallboard insulating industry for many years. (Slide presented) This is a picture of Doctor Shaver's disease in the Niagara peninsula's crude abrasive industry; (slide presented) you can see the multiple areas of pneumothorax that had occurred. Here is the X-ray of a coal worker who had generalized fine nodulation plus superimposed tuberculosis which was active. (Slide presented) A coal macule specimen was obtained from this patient by a simple trephine drill needle biopsy in the office.

I want to emphasize that the drill biopsy is sometimes most helpful. Another needle biopsy showed crystobalite silicosis in a man who worked making dental material; part of the ingredients to which he was exposed was crystobalite.

The last example of an occupational disease is not due to an inorganic dust. It is, however, the only case I know of chronic mineral oil pneumonia of occupational origin. The man had for many years reconditioned old cash registers; his particular job was one of spraying a fine oil on these registers after they had been cleaned. The first physician to make the diagnosis told his technician to get a sputum specimen on the patient. She came back after awhile and said, "I can't get it to stay on the slide."

VIEWBOX CHEST X-RAY DEMONSTRATION, OCCUPATIONAL PULMONARY DISEASES

BY AMERICAN COLLEGE OF RADIOLOGY

Eugene P. Pendergrass, M.D.: This morning you covered the clinical aspects and the importance of medical history in the diagnosis of pneumoconiosis. This afternoon we will take up some of the radiological problems. I'd like to bridge that gap by describing a letter I read in a July issue of JABA.

The letter likewise called attention to the importance of a careful and complete history in the evaluation of patients with possible environmental lung disease. The letter, from two Army medical officers in Tacoma, Washington reported on a 56 year old retired Army sergeant seen at the Army medical center in 1969 with right pleuritic chest pain and a low grade fever of three day's duration. He was found to have a right sided pleural effusion. Complete evaluation including thoracentesis and pleural biopsy did not show a cause for the effusion. He was followed as an out patient, and developed a bilateral pleural thickening compatible with asbestos exposure. When initially queried about occupations considered pertinent to asbestosis, like asbestos weaving and steam pipe lagging, he denied any such occupation. However, when asked later for a very detailed occupational history, he recalled working for 9 months at age 16 as a fire eater in a carnival. This entailed using an asbestos torch, inhaling and exhaling fire from the torch and frequently extinguishing the torch by placing it within his mouth. Undoubtedly that was sufficient exposure to produce pleural changes from asbestosis years later. That's as good an example of the need for searching inquiry as I can present to you.

This afternoon we'll be concerned in part with the program inaugurated by the Federal government to study and define control measures for coal worker's pneumoconiosis. The American College of Radiology was invited to assist in an educational program for physicians concerned with identifying and clarifying the type and extent of the disease process and to assist in the consideration of problems with film quality and reproducibility of roentgen interpretation. These problems involve basic considerations which are subject to continuing research.

Editor's Note: A portion of the Federal Coal Mine Health & Safety Act of 1969 requires the Secretary of Health, Education, and Welfare to conduct a periodic medical examination program

for coal miners working in or at underground coal mines. The examination must include a chest roentgenogram and any additional tests specified by the Secretary. It is specifically required by the Act that the chest roentgenograms be interpreted and categorized by a system to be specified by the Secretary.

In the cases of miners already employed in the industry on the effective date of the Act, December 30, 1969, the examinations were voluntary and were to be offered to the miners at no charge during the first 18 months after enactment, 3 years thereafter, and from then on at intervals not to exceed 5 years. Persons employed in underground coal mining for the first time subsequent to enactment were required to be examined at about the time of employment and 5 years thereafter. Moreover, if any new miner showed evidence of the development of pneumoconiosis, a third examination, 2 years later was required.

The first medical examination regulation under the Act were issued in August 1970. They specified that the chest roentgenograms were to be classified for pneumoconiosis in the UICC/Cincinnati (1968) system. (Bohlig, H., etae., "UICC/Cincinnati Classification of the Radiographic Appearances of Pneumoconiosis" Chest, July 1970, 58:1, pp. 57-67). This new classification system had some similarity to the ICD system which physicians were generally aquainted but expanded it to provide, among other things, more finely divided categories and to include small irregular opacities which were previously frequently ignored. In order to aquaint physicians who would interpret chest roentgenograms made under this program and/or receive reports in the course of treating coal miners, NIOSH entered into an arrangement with the American College of Radiology (ACR) to develop and present 2-day seminars to train physicians in the system. These physicians then became eligible to participate upon in the program as first or "A" readers. A total of _____ seminars were held with a total attendance of

_____.

The first round of voluntary examinations ended December 30, 1971, after having been extended in time because of a work stoppage in the industry. The regulations, however remained in effect, especially with respect to new miners until July 1973 when revised regulations applying to both the second round of voluntary examinations and to the mandatory examinations of new miners were published.

These revised regulations, like the initial ones, include roentgenographic quality specifications which were recommended by a special committee of the ACR. The quality requirement of the new regulations included revisions which were based on the first round experiences.

There were also three important changes in interpretation requirements for the second round, although physicians who were previously approved as first or "A" readers continued to hold that position.

(1) A new internationally recognized system of roentgenogram classification for pneumoconiosis known as the ILO u/c (1971) system was adopted. (Jacobson, G., and W.S. Lainhart, Eds. "ILO u/c International Classification of Radiographs of the Pneumoconiosis". Med. Radiog. and Photo. 1972, 48:3 pp. 65-110)

(2) All interpreters were required to have available and refer to the standard films of this new system when interpreting roentgenograms.

(3) A proficiency test in interpreting chest roentgenograms for pneumoconiosis had been devised. Only physicians who passed this examination, about 75 in number, were acceptable to make final interpretations.

Most of the presentations made in Symposium II by the American College of Radiology are condensations of the training program previously presented by the ACR for NIOSH. Many of the forms, procedures, and regulations to which reference is made are part of the NIOSH miner examination program. At the time of this Symposium, most of the data which was available to the speakers came from the first round. Thus, it should be noted that the classification system and NIOSH

interpretation form discussed by Dr. Bristol were those used in the first round of examinations. Dr. Trout, however, discusses the quality control procedures incorporated in the revised regulations for the second round.

VIEWBOX CHEST X-RAY DEMONSTRATION, OCCUPATIONAL PULMONARY DISEASES

BY AMERICAN COLLEGE OF RADIOLOGY

RADIOLOGICAL CHANGES IN PNEUMOCONIOSIS, INCLUDING COAL WORKERS' PNEUMOCONIOSIS

George Jacobson, M.D.

George Jacobson, M.D.: I'll review with you the radiologic changes that one sees with some of the pneumoconiosis, not all of them. First, I would like to present to you the most recent International Labor Organization (ILO) 1971 definition of the pneumoconiosis--that is the accumulation of dust in the lungs and the tissue reactions to its presence. For our purpose here, dust is meant to be an aerosol composed of solid inorganic particles. This morning the clinical manifestations and pathological changes of the various pneumoconiosis were reviewed. One of the important things to consider is that individuals now are rarely exposed to a single dust, and most workers who are exposed to dust are exposed to at least several varieties.

In general, the pneumoconiosis can be divided into those which produce collagenous changes and those which do not. Of those which do not produce collagenous changes, the four most common are those related to coal, tin, barium, and iron. The pneumoconiosis that produce collagenous changes are silicosis from silicon dioxide, the fibrous silicates as represented by asbestos, and again coal. We don't really understand why under one circumstance coal does not produce any collagenous changes and under other circumstances it does. The pathogenesis of coal and its production of either massive fibrosis or collagenous changes is really unknown.

I would first like to discuss and demonstrate coal worker's pneumoconiosis and silicosis. When it is inhaled, coal is phagocytized, and the coal dust is deposited in foci located in the interstitial tissues of the lung, that is in the alveoli or surrounding the local small branches of blood vessels, lymphatics, and terminal bronchioles. For radiological purposes, the pneumoconiosis may be considered as either focal disease or as diffuse disease. Both silica and coal produce a focal type of lesion, and this affects the radiographic appearance of these diseases. Here you see a coal macule located in the alveolar space among the alveoli. (slide presented) This patient died of a non-specific pneumonia. As you see the alveolar walls themselves are not involved, but they are dilated, and coal does produce focal emphysema. In a whole lung cross section of an individual who has a minimal deposition of coal with scattered coal macules, there are scattered small opacities of the so-called p type. These are very fine, averaging no more than a millimeter and a half in size. Whether or not the size of these small opacities has any pathological significance is not really well understood.

There has not been really good radiologic, pathologic, and pulmonary function correlation done in relation to coal worker's pneumoconiosis.

Some epidemiologists seem to feel that perhaps the degree of emphysema or disability which one finds with coal worker's pneumoconiosis is inversely related to the size of the small opacity. The most severe cases of emphysema often seem to occur with these pea sized opacities.

The disease may even become more profuse with very large opacities. the r size. Notice up here the appearance of very early confluence of these densities (slide presented). If you follow individuals from the early stage of the disease, the opacities don't appear to increase in size in a given individual. The type of small opacity that develops at the very beginning apparently remains constant. Then as the disease advances, this confluence occurs. There is no good explanation. At first it was thought to be related to tuberculosis and infection, but that apparently is not the case. Some immunologic reaction may very well be the explanation; there may be an altered sensitivity in some individuals. In present day medicine, any differences we really don't understand are often blamed on alteration in the immunologic system of the individual. We can't prove it, of course.

Not infrequently we see individuals who rather than this usual rounded opacity, have instead a diffuse mixture. Here are not only rounded opacities but also irregular opacities (slide presented). They have a different shape. Going back to the diagrams of Heppleston (see reference 1), it's not hard to understand the linear part of it. But opacities of other shapes may also be due to the inhalation of other dusts. As we look for these, we find they are more and more common. There are two recent papers, one from Great Britain, expressing the opinion that coal workers who have a significant number of these irregular opacities, probably have more emphysema or more disability than those that do not.

Individuals with very far advanced disease, begin to develop large conglomerate opacities, whose etiology we do not understand. They have very severe emphysema. Such an individual might not have many visible small opacities. On occasion you will see individuals who have large opacities, but no small opacities, because they have been made invisible by the extensive surrounding emphysema. A typical X-ray shows defined large opacities and small opacities scattered throughout both lungs.

Those of you who have not read Heppleston's article, I urge you to do so. Looking at coal macules in Gough section, they have in overall geometry a rounded shape. They occupy an area which is in general, rounded. If you take the same macule and look at it in a longitudinal section, you see that coal is deposited along the walls of the terminal and respiratory bronchioles as well as around blood vessels, or lymphatics. In a longitudinal projection, they no longer have that rounded appearance but are more or less linear or at least they have a linear distribution. When one of these is seen in a chest radiograph, while the predominant pattern or appearance of the foci is in general rounded, it is not surprising that some of them have a linear shape. This fact contributes to our X-ray classification.

In silicosis the nodule has a little different appearance. It's again a focal disease, but there is a collagenous reaction. Some of the collagenous fibers extend out into adjacent alveolar walls. There is also associated emphysema, although this time the pathologist likes to call it scar emphysema as contrasted to focal emphysema in the coal macule. In a Gough section, the appearance is somewhat different; the macule is solid, dense with scarring and retraction. There is emphysema surrounding it. The radiograph reveals small opacities distributed primarily in the upper halves of both lungs. These small densities are termed rounded opacities in an attempt to use a non-pathological, descriptive term. They are focal in nature and distributed as you see. (slide presented)

In a lung that has more involvement, much more extensive coal worker's pneumoconiosis, the X-ray reveals numerous foci throughout both lungs, again associated with focal emphysema. This is coal worker's pneumoconiosis of considerable more profusion and extent of disease. The various small rounded opacities have been classified as to size p, q, and r depending upon their average size as seen on the chest radiograph. In severe coal workers' pneumoconiosis with severe focal emphysema, while there are actually more coal macules at the bases of the lungs,the number of recognizable macules actually become less. There are fewer per square inch because of the involved emphysema. As emphysema becomes more marked, the number of visible foci may diminish. Another example (slide presented) a silicotic rather than coal worker's disease but the

distribution and size of the large opacities is not significantly different. Notice how they are scattered throughout both lungs. This was a lady who was a pottery worker and was exposed to silica. This is not an unusual distribution for these large opacities. While they are predominantly in the upper zones, they may also occur in the lower zones. They may be bilateral, patchy in appearance and can simulate many other diseases. Even on this original film, it is very difficult to find small opacities.

This rather interesting film is of a coal worker in 1944 who had huge masses in both lungs. (slide presented) I have not seen any much larger than this. Two years later you can see how they've shrunken. This is not healing or regression; this is progression of the disease. These large masses may shrink, contract, move in toward the hyla, the central areas of the lung, leaving behind markedly over-expanded emphysematous lungs. This man's pulmonary function would decrease accordingly. This is not healing; it is progression.

Now I want to tell you of one of the very interesting pneumoconiosis that occurs in diatomaceous earth workers. In the type of silicosis that one sees with diatomaceous earth, the opacities are smaller than you see with coal and other such diseases. There are more of the small linear opacities, and they are very diffuse. This is a diatomaceous earth worker in 1953, and here he is 10 years later. (slide presented) He was removed from exposure in 1953. He's been an office worker since, and you can see that, in spite of the removal from dust exposure, his disease has progressed markedly. He now has large opacities in both upper zones and diffuse emphysema throughout the lower zones and his small opacities have virtually disappeared. However, here is a different individual in 1969. (slide presented) This man is still working with diatomaceous earth, and I saw some of his chest X-rays a few weeks ago. It hasn't changed much now, but you can see the marked distortion of his mediastinum, and the marked over expansion of his remaining lungs. He can out hike any person in this room, regardless of your age! This man is now in his late 60's. He's still working. He puts on a backpack and climbs in the Sierra. It is just unbelievable with lungs that look like this.

This is a coal worker (slide presented) with huge masses, proven at autopsy. We didn't know what it was when we saw them. His emphysema is just astonishing and the big masses at autopsy were almost pure coal. Even on the original film, there were no visible small opacities. One can get these very large masses with no visible small opacities. They are not common, but they do occur. The problem is, one then reasons from this, that an individual can have advanced coal worker's pneumoconiosis and silicosis and no large opacities and no visible small opacities. While that may occur, it must be extremely rare without the presence of large opacities to hide the extensive emphysema and the small opacities.

One other feature of silicosis and coal workers' pneumoconiosis: On rare occasions we will see an area of massive fibrosis which has excavated. In all the thousands that we have done in our studies, I don't think we have over half a dozen. This can occur with just vascular impairment shutting off of blood supply and producing aseptic necrosis. Or, it may be a carcinoma which has excavated. Or, it can be tuberculosis or some other infection. There is no way to tell from the radiograph itself. Of course, you have to investigate clinically.

We are frequently asked the question, "Can you distinguish coal workers' pneumoconiosis from silicosis radiologically?" And, the answer very simply is "No." Certainly not in any individual case. I would just call your attention to the difference in the prevalence of small opacities in coal mine workers and metal mine workers or silicotics. (slide presented) The percentages don't differ significantly in the size of these small opacities. If you take the large opacities between the two groups, the only real difference possibly is the fact that the very large conglomerate masses, the c type, are somewhat more prevalent in the metal miners than in the coal miners. But the overall prevalence is not different and certainly in any individual case, you can't differentiate.

There is one possible differentiation between these two pneumoconiosis and again, it is only a statistical one, and that is the egg shell calcifications. They are found in coal workers, in silicotics, and on very rare occasions in other diseases. There are certainly less than a dozen authenticated instances of egg shell calcification in individuals who have not been occupationally exposed to either coal dust or to silica dust. These cases include one that I just saw

recently in histoplasmosis, some in primary tuberculosis, and a few in sarcoid, but they are extremely rare. The other thing that may occur in these individuals is some calcification in their small opacities. Both these calcifications, in the small opacities and the egg shell calcifications, the British in particular believe is related to the amount of silica rather than just to coal. The prevalence in Great Britain is much less than in our country, and we have more silica dust in our coal dust than the British do. This may be an indication of silica rather than just coal. We do have very definitely some individuals who, while having been exposed to dust and having no visible small opacities, do have egg shell calcifications in both coal workers and in metal mine workers. The significance is that, given an individual who has been exposed to dust and does have egg shell calcifications, the odds are overwhelming that the calcification is a manifestation of pneumoconiosis rather than of some other disease. It has no clinical significance except one of diagnosis.

Another feature of both coal worker's pneumoconiosis and silicosis is the so-called rheumatoid lung or Caplan's syndrome. I mention it without going into detail. This is one of Caplan's original cases, a man who has since died and has been autopsied. (slide presented) He did have coal dust in his lung although there are no small opacities visible in the X-ray. There are large rheumatoid lesions, and can occur in people who have not been exposed to either coal dust or silica. It is probably a manisfestation of the rheumatoid state rather than pneumoconiosis. Whether it's potentiated by the pneumoconiosis or not is moot.

The prevalence of coal worker's pneumoconiosis in the United States determined in the first round of examinations under the Coal Mine Health and Safety Act of 1969, 66,000 miners in the United States, almost 65,000 bituminous workers and 1,000 anthracite workers were examined. The prevalence of the simple pneumoconiosis (categories 1, 2, and 3) is a little over 10 percent and of complicated pneumoconiosis, 1 percent. In the anthracite workers the prevalence is considerably higher than in the bituminous mine workers. Category 1 in anthracite workers is 18 percent compared to 7 percent in bituminous; category 2 is 7 perfent compared to 2 percent and category 3 is 8 percent compared to 0.2 percent. Complicated pneumoconiosis or massive fibrosis is a little more than 5.5 percent in anthracite workers compared to 1 percent.

Doctor Pendergrass knows a great deal more about this than I do. I presume, am I right, Gene, that you think that might be related to the amount of silica present in the dust?

Eugene P. Pendergrass, M.D.: Probably.

VIEWBOX CHEST X-RAY DEMONSTRATION, OCCUPATIONAL PULMONARY DISEASES

BY AMERICAN COLLEGE OF RADIOLOGY

ASBESTOSIS
George Jacobson, M.D.

There are focal non-pneumoconiotic interstitial diseases which one must always keep in mind, given an individual coming into a radiology department. Just because one has small reounded opacities doesn't necessarily mean that it is pneumoconiosis. For example, a patient with miliary tuberculosis indistinguishable radiologically from some examples of coal worker's pneumoconiosis. Or an individual with sarcoidosis which can look identical to some of the others. So radiologically we can't really tell one focal disease from another diffuse focal disease; it could even be metastases. It could be pneumoconiosis. It could be tuberculosis. It could be a whole host of things. So you must have an adequate history and adequate clinical findings to make your diagnosis in a given case.

Three examples of the benign non-collagenous penumoconioses, the most common ones, are iron, tin, and barium. Siderosis shows small rounded opacities diffusely scattered through both lungs, indistinguishable radiologically from coal worker's pneumoconiosis or from silicosis. But these are benign and have no clinical reactions. The patients do not become emphysematous, at least not related to their pneumoconiosis. It is essential that we keep these other diseases in mind for the purposes of differential diagnosis. Here is an example of stannosis or tin. (slide presented) You'll notice that while this is overall a contrasting film, in addition the density of the small opacity is related to its molecular weight, and you can see that these tin densities are much denser than either coal or silica. This is also a benign pneumoconiosis in spite of the intense involvement. Next (slide presented) barium having a very high atomic number produces lesions which are extremely dense. This man had absolutely no symptoms.

Asbestos is rapidly becoming one of the most serious occupational health problems. It has received enormous amounts of publicity in recent years. The amount of asbestos used today is anywhere from 10 to 100 times as much as 25 to 50 years ago. It is an extremely important industrial product and modern technology really couldn't exist without it.

There are four general types of asbestos: Chrysotile, crocidolite, amosite, and anthrophyllite. Ninety percent of that used in the United States today is chrysotile, a large part of which is mined in the Thetford area of Canada, also some of it is mined in the northeastern United States. There are also some small asbestos mines in California. The crocidolite and amosite are primarily mined in South Africa as is some of the anthophyllites. Other anthophyllite mines exist in Finland and Russia, and scattered throughout the world elsewhere. Chrysotile is also mined in Russia. These asbestoses are all fibrogenic; some more than others. There is some disagreement to whether all of them are carcinogenic. The characteristic thing clinically is that following inhalation of asbestos, there appear so-called asbestos bodies. We would better call them ferruginous bodies, because similar bodies can be produced by inhalation of ordinary organic fibers. One can determine whether the fiber which you see inside of this body is asbestos at the present time only by electron probes chemical analysis. So this type of body can be produced by other causes. But it is very characteristic in asbestos exposure. These bodies also occur in the general population, which compounds our problem of diagnosis. Even in the general population these bodies occur in non-working females 30 percent as compared to blue collar workers around 50 percent and in construction workers 70 percent.

Now let us go through the same type of exercise that we did with the coal workers, and try to imagine what one would see with increasing progression of disease. Here's a very early lesion in an individual who died, had asbestos exposure, and you can see a fibrogenic area (slide presented) While it looks focal in nature, the involvement extends to the alveolar walls. Adjacent to this focus there is thickening of the alveolar wall, quite different from either silicosis or coal worker's pneumoconiosis. The chest radiograph of someone with that extent of involvement, one might see linear or irregular shadows in the lower zones of both lungs, also an enlarged heart and pulmonary artery. This man came into the hospital because he was having symptoms of respiratory distress and pulmonary hypertension.

Now let us look at an individual who has more advanced disease (slide presented). Now the collagenous reaction has extended to involve large areas of the lung. There is exudate within alveoli. There are numerous areas of inflammation. He has asbestos bodies imbedded in the collagenous tissue. Here is the same individual I showed in the previous film, four years later. (slide presented) In spite of being removed from dust, his disease has progressed. Notice the distribution of opacities in asbestosis is more in the lower than in the upper zones of the lung. Here is a miner with more extensive involvement (slide presented). These small irregular opacities are larger than in the first one where they were what we call the s type. Here they are a mixture of t's, they're bigger, thicker, juicier looking. His whole lung is now involved. His heart and main pulmonary artery are enlarged. Undoubtedly he has pulmonary hypertension.

In the most advanced cases, ill defined large conglomerate masses have appeared with very dense fibrosis. In contrast to a large massive fibrosis in coal workers' pneumoconiosis, there is no margin to the asbestosis lesion, while in coal workers you see the lesion can be very clearly demarcated among normal alveolar structures. In Great Britain, commercial talc is used as insulating material; this man had worked in that industry many years and had advanced asbestosis due to talc. (slide presented) This is another miner with very advanced disease. (slide presented) See this large ill defined density; the entire lung is involved, the heart large, and the heart margins and diaphragms obscured.

Unfortunately for diagnosis, these changes are non-specific. There are a host of other interstitial diffuse diseases of the lung which can mimic asbestosis. This case went through our hospital July 31, 1974. (slide presented) His radiograph is absolutely typical of asbestosis, large mass, large heart, cor pulmonale, the diffuse irregular opacities. But, this man has rheumatoid lung. He has advanced rheumatoid arthritis. We have films going back ten years showing the gradual progression and appearance of this lesion. He even had bilateral pleural thickening in these masses. Absolutely typical of asbestosis. You can find the same thing in sarcoid. You can find it in patients who have scleroderma and other rheumatoid states.

The other very interesting manifestations of asbestosis are the pleural changes. Very characteristic of asbestosis are large pleural plaques on the diaphragm, and thickening along both chest walls. They are more common actually than the interstitial changes. Very characteristic also are diffuse serpigenous calcifications of the diaphragm and of the chest wall.

The last manifestation which is very characteristic of asbestos is the mesothelioma. This is an example. (slide presented) He has interstitial changes, he has a large mesothelioma in his right chest. The relationship of asbestos to carcinogenesis has been well shown in Philip Enterline's 1973 publication. (see reference 2) Compared to the standard mortality rates, in asbestos workers the carcinomas in general are about 50 percent increased. There is a significant increase in the number of carcinomas of the digestive tract, particularly the stomach, and colon; the relationship to bronchogenic carcinoma is almost overwhelming, two and a half times normal. Incidence of all other cancers is relatively normal. Respiratory disease is very significantly increased. About 65 percent of the deaths in individuals who were occupational exposed to asbestos are from respiratory failure.

Doctor Selikoff relates some of the carcinogenesis, particularly bronchogenic carcinoma, to smoking. In his figures, cigarettes potentiate the exposure to asbestos. He thinks maybe that without smoking carcinogenesis, or at least the prevalence of bronchogenic carcinoma, may not be significantly increased, but you put the two together, and it is dynamite.

One other issue is the asbestos fiber count in drinking water, and in various beverages. This has gotten into the newspapers recently in connection with the Reserve Mining Suit in Minnesota. I would like to ask Len Bristol to describe his experience. Recently there was a paper tending to show that perhaps the recent rather extraordinary increase in pancreatic carcinoma might be related to asbestos exposure. That has not been corroborated yet, only one study was reported.

Leonard J. Bristol, M.D.: The Reserve Mining case in Minnesota has been through the Federal Appeals Court and is now back in the original court. It might be presumptuous of me to make many comments until the court has made its decision. But I think I can state this. I don't think it has been proven that the fibers in the waters of Lake Superior or coming out of the stack at Silver Bay, Minnesota, are in fact asbestos fiber. This has to be proven, because there is a scientific feeling that the fiber must be of a certain length before it can be described as being potentially dangerous. Sections of colon that have been removed in the city of Duluth have been studied for fibers. Doctor Selikoff and his group, and a group from Cardiff, Wales, and other scientists of the Mayo Clinic have agreed that they have not found any fibers. This is very preliminary comment. The incidence of carcinoma of the lung and the incidence of carcinoma of the intestinal tract in Duluth and surrounding areas is no higher than it is in the rest of the State of Minnesota. As a matter of fact, it is lower than it is in New York City. I studied X-rays of employees of this company from its inception about 1951-52 through 1973. I found no evidence of silicosis and no evidence of asbestosis. Of the entire group, there were 1,500 to 1,700 who had been employed anywhere from 18 to 24 years not only in Reserve Mining but in and around that area, the taconite area. We have no X-ray evidence at this time of asbestosis, and the clinicians do not have evidence of clinical asbestosis. I want to caution all of you that these findings are preliminary. We have to wait for the final results.

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VIEWBOX CHEST X-RAY DEMONSTRATION, OCCUPATIONAL PULMONARY DISEASES

BY AMERICAN COLLEGE OF RADIOLOGY

A PLASTIC STEP-BLOCK FOR TESTING FILM QUALITY
E. Dale Trout, Sc.D.

I wish I could tell you that if you go home and use so many kilovolts and so many milliamperes per second, you'll have a good chest film, but it doesn't work that way. Twenty years ago that might have been true. But now we have too many different kinds of X-ray machines, each different in its characteristics. We've had to do something else to try to arrive at a common denominator.

Without over-exposing patients, and considering the impossibility of calling them back if you want to take a second look, we developed the following procedure which NIOSH has incorporated into the miner examination program. We made a polystyrene step block 17 inches long and 7 inches wide. If it is exposed with an average chest technique, it will give you the range of densities that you would produce on a chest film.

NIOSH's Appalachian Laboratory for Occupational Respiratory Diseases (ALFORD) at Morgantown, West Virginia, has 50 of these test blocks. They are in boxes so that they can be shipped. If you want a facility certified to take X-rays under the Federal Coal Mine Health and Safety Act 1969, write to ALFORD's X-ray Receiving Center. They will send you copies of the regulations, the necessary application forms and one of the test blocks.

The step-block device can be exposed as many times as you want, because you're not exposing people. And, it provides a lot of information. It tells us about the centering of your X-ray tube. Some of you are using grids, and the centering of the X-ray tube is critical with these 100 line biratial grids. We put a pin on the device, and a ring on the back, and when things are correctly lined up at 6 feet with the tube centered to the center of the film, the pin will fall in the ring on the radiograph.

It also tells about the distance that you are using. The regulations call for 6 feet, so we have two bars on the block, and when a 6 foot distance is used, the distance between the radiographic images of those two bars is exactly 10 centimeters. If you cheat 6 inches, we can detect it. We have wire mesh on the two sides of the device, because the regulations say that you should use medium speed screens and medium speed film, and if instead you use high speed film and high speed screens the resolution on the film is poor. We detect it immediately.

To use the test block, you just put it in front of your chest holder; tube everything as if you were doing a chest film, and make an exposure that you would make for an average chest 20 or 22 centimeters in thickness. The film is submitted to ALFORD. They look like this. (slide presented) Here's the ring and the pin. This one is properly centered. You can see here is the thick end of the block down in the diaphragam area. So the densities here are very light. As you get up to where the tissue thins out up over the shoulders the densities get very high.

A densitometer is used to measure the density of these steps. The densities have been plotted that you should get for an average chest technique using this thing with all the different types of conditions: no screen, the 6 to 1 grid, the 8 to 1 grid, and the 10 to 1 grid. The density of your film is plotted against this, and you receive a sheet of paper which tells you whether your exposures are high or low. The image of the centering device and the image of the wire mesh are also examined and if you're too bad you have to do it again. The thing that occurs most often is that the tubes are not lined up to the cassette changes. This happens because people do not have good marks and good mechanical devices to put stops on these things. If we're going to get away from overlying the whole chest with a thickness of lead as you shoot into the side of these lead strips with these grids, and therefore increase the exposure to patients, we have to get these things lined up. They are out of line almost as often on new installations as on old ones. The service men just don't take enough care in centering them.

That is about the story. NIOSH is using this technique in order to solve the film quality problems experienced in the Federal program to X-ray coal miners. ALFORD sends you the test block; under Federal contract, we measure the film. If we think we need additional information, we call you up and talk with you about it. If you have questions about this testing device, write us a letter.

There is a good reason for every requirement; they are intended to produce optimum films for coal worker's pneumoconiosis. One of the commonest things that we have to discuss is the medium speed screen and film. We still find poeple trying to use high speed screens.

VIEWBOX CHEST X-RAY DEMONSTRATION, OCCUPATIONAL PULMONARY DISEASES

BY AMERICAN COLLEGE OF RADIOLOGY

GOOD FILM QUALITY FOR DETECTING PNEUMOCONIOSIS
E. Nicholas Sargent, M.D.

I want to emphasize the importance of film quality not only in the detection and study of the pneumoconioses but also in the study of all chest diseases. By far the greatest number of radiographic examinations involve X-rays of the chest. There are actually many more chest X-rays than there are of the abdomen, extremities, and other areas of the body. It is in the chest X-rays that poor film quality will result in errors in diagnosis.

We have certain criteria for a good chest film in a normal patient. And we have certain criteria for a good film particularly for a study of the pneumoconioses.

We want to see the peripheral pulmonary structures best, because it is in between the blood vessels that we're going to pick up the earliest disease processes. While it is desirable to be able to expose the chest so that we see the mediastinum and the peripheral lung structures equally well, it is usually not possible. In a film that is great for the mediastinum, it's great for seeing behind the heart, but we're burning out the peripheral pulmonary structures. A film that may be exposed well for the mediastinum or ribs is not what we need for the study of the pneumoconioses. So we compromise.

If we see the vertebral bodies faintly behind the heart shadow and some of the major blood vessels through the cardiac shadow, that is an adequately exposed film for study of the pneumoconioses.

There are other criteria for what we consider a good chest film. We don't want to see the patient rotated. We like to see the sternal ends of the clavicle equidistant from the mid-line. We like to see the patient in full inspiration, and I'll go into this in more detail. We don't want to see the scapulae in the way. All of these are very important, particularly if we're going to pick up minimal early interstitial diseases of the lung.

Macklin many years ago showed that in exhalation the deaeration usually occurs mostly at the bases and periphery of the lung. This film taken in expiration actually shows compression of the basal portions of the lung. (slide presented) It may be adequate for the upper zones, because in this area you don't compress the lung in expiration. But if you are looking for the finer structures, you certainly will be unable to see them in an expiration film. Furthermore, you notice that the cardiac shadow appears to be large. If I were to tell you this man came in coughing and he had pneumonia, you'd say surely he had disease. But, if filmed in inspiration, this is actually a perfectly normal chest.

What criteria do we use for determining that the patient has taken a full inspiration? You should see the leaves and domes of the diaphragm, at about the level of the sixth rib anteriorly or the tenth rib posteriorly. But we don't want to see an expiration chest, and this film shows why. This patient came in coughing and was diagnosed as pneumonia. (slide presented) But if you count the ribs—1, 2, 3, 4—you see that the patient has failed to take a deep inspiration and this produced a spurious finding, because in deep inspiration there is no evidence of pneumonia. Inspiration is extremely important.

We don't want to see a film in which the patient is rotated. Here is a patient who is semi-rotated. (slide presented) You see widening of the mediastinum. This part of the chest in left anterior oblique is somewhat foreshortened; it's hazy looking. It looks as if he has pleural thickening. This could lead you to diagnose disease when the appearance is in fact only due to technical factors.

A recumbent A-P film is never acceptable, particularly for the study of the pneumoconioses. Here's a patient in a recumbent position. (slide presented) Notice the mediastinum widening, and the vascularity—the blood flow is to the upper lobes. In fact with the patient in an erect position, this is a perfectly normal film. (slide presented) Again it is very easy to diagnose disease when faulty technique is used. The patient must be erect, and the film must be taken in deep inspiration.

Another spurious finding we encounter occasionally is the Valsalva effect. Some technologists will tell the patient to take a deep breath, and then they wait too long before they make the exposure. So the patient squeezes down against the closed glottis. This increases the intrathoracic pressure, squeezes out the blood from the blood vessels, and compresses the heart. The film can look almost like an emphysematous patient, when if it is taken immediately after a deep inspiration, it is in fact a perfectly normal patient. A Valsalva maneuver can shrink the heart as much as 30 percent. And, this is important for another reason. If you had taken such a film originally and the patient came in again for a cardiac infarct, you would say, yes, his heart is getting larger. In fact the heart size on the original film is due merely to the fact that he waited too long. The patient squeezing out against the closed glottis caused the Valsalva effect.

If the scapulae are not retracted away from the chest they mask the blood vessels. And, if the arms are not abducted away, it can look like pleural thickening (which we see in asbestosis) when in fact it is merely caused by the patient's arm being at his side.

Hair also can present problems. Here is a rounded shadow. (slide presented) Notice that it goes off into soft tissue. This is actually due to braided hair draping over the patient's shoulders. When you remove the hair braid, it is a perfectly normal patient. It would be very easy for the uninitiated to call this tuberculosis or one of the large opacities that may be associated with pneumoconiosis. Of course, this is a female but we do see women who are now working in the mines, and we see women from clay factories in Mexico. They have silicosis. Male miners also have long hair today.

Another problem is clothing. Today we have many synthetic materials in sweaters or shirts. This in fact looks like p opacities throughout the entire lung. (slide presented) Really it is due only to synthetic material in a sweater. On a t-shirt there is often lead. (slide presented) We see artifacts due to the printing on t-shirts, and if I blow this up a little more, it looks like q opacities. In fact if you take the patient's clothing off (and that is the way it should be done) the patient is perfectly normal. You must pay meticulous attention to the positioning, the patient's clothing and to getting the patient in deep inspiration.

Now what about the other parts of the technique? What about the film? There are numerous types of film processing artifacts. I want to show you one in particular. This original film shows small rounded opacities. (slide presented) They look like p opacities. If you are wary of this you will see that the "p opacities" actually extend into the soft tissues. Here is a magnified view. Now you see rounded white spots with a central radial lucency. I know of no pathological process in the lung that will produce this on the X-ray. This is actually due to exhausted developer and dirty rollers, but the film itself can look just like the p opacities. We have to

pay meticulous attention to technique if we are going to avoid mistakes.

I don't know how many of you are radiologists, but those who are not may hear the word cassette, and ask: "What is a cassette?" It is merely a light-proof carrier for the film. In this cassette, the film is sandwiched between two sheets of material known as intensifying screens. The intensifying screens probably give us the most problems in technique; these can cause all sorts of artifacts that give you spurious findings on the film. Why do we use these intensifier screens? Well, first they can cut down the amount of radiation necessary to obtain a photographic effect by as much as 1/15th to 1/40th. What happens is that the primary radiation hits the crystals on the intensifying screens and they emit light. It is because of their light that the photographic effect is enhanced.

Anything that interferes with this light hitting the film will cause difficulties. Suppose you had a scratch or dirt on the intensifying screen? Then you decrease the amount of light hitting the film, causing an artifact on the film. Let me show you a few examples. (slide presented) It's easy to call this rounded opacity here tuberculosis or pneumoconiosis. In fact this is due to dirt on the intensifying screen interfering with the emission of light. When you expose the patient with a better screen you see it is a perfectly normal finding. This problem is dramatized in a series of films we just read for pneumoconiosis. (slide presented) Here's a large oval opacity exactly like tuberculosis or pneumoconiosis. Yet in this same group of films, different patient now, the same opacity. And, a third patient with the same opacity. Of course now it is extending out into the soft tissue and clearly this is due to a dirty intensifying screen or artifact, or a dent in the screen in the films of all three patients. It is very important that we have clean screens.

Poor film screen contact is another extremely important problem because it is subtle. You must know how to detect it, particularly for pneumoconiosis. The problem is, if you have a bent intensifying screen, it may interfere with the closeness or proximity of the film to the intensifying screen. This will cause a loss of light. The light will have to travel further. The light will come off at odd angles, and you will lose detail. I'm going to show you some examples of this, because it is particularly important in pneumoconiosis. (slide presented) This example was due to a cassette that had a poor screen clip preventing good contact with the film. You compare the blood vessels, the sharpness of outline, the detail on the right side to the left. You lose informational detail. Many errors occur if you have poor screen contact. And this slide shows it. Here's a patient with the small rounded opacities of pneumoconiosis with good screen contact. Here's poor screen contact. You're losing information on film with a blurry image. You cannot possibly make an accurate diagnosis. You can't count opacities to estimate profusion. Compare this side to this side. This is poor screen contact.

Fogging of the film can occur for many reasons. It can be fogged due to light striking the film. It can be due to heat, scattered radiation, or processing. This film was purposely fogged on one side. (slide presented) Compare the sharpness of outline of the blood vessels. Compare the left to the right. We've lost information. We have poor detail, which is important because we are looking for small images of 1 to 3 millimeters. A simple reason for fogging the film is failure to limit the radiation beams to the patient's chest.

A cone is used to limit the beam of radiation, a process which we call coning. The nice sharp outline to the blood vessels in this film demonstrates this process. (slide presented) The cone was wide open in this film, and secondary radiation hit the abdomen or the wall and scattered to the patient's chest. This is additionally undesirable, because the patient's gonads will get over exposure to radiation, which is harmful. We like to see the cone cut 1 or 2 centimeters below the level of the costo-phrenic angle.

Fog on the film from radiation scatter happens particularly in obese patients. In order to clean up this scattering or secondary radiation, we interpose what is known as a grid between the patient and the film. This could also be done with an air gap technique, but let me show you the effects of the grid. Here's a heavy patient who requires as much as 130 kv to penetrate if you use a grid, (slide presented) and you see fine detail because this grid cleans up the secondary or scattered radiation. Here is the same patient without a grid at 80 kv, and you've lost all the detail. The rounded opacities have disappeared with failure to use a grid, particularly in

the higher kilovoltage range. If we go above 80 kv the scattering increases.

As we increase the amount of radiation going through the film, we can make the disease disappear. We talk about quantity of radiation based upon milliampere seconds. As we increase the quantity of radiation 5 to 8 to 12 milliampere seconds, we're actually causing the disease to disappear. This man has a lot of p opacities. (slide presented) Yet with an improper exposure by increasing the quantity of raidaiton, we're not going to detect the disease. This is emphasized in a recent article by Reger, et al in the American Journal of Roentgenology. They compared preferred films with good exposure to films that were underexposed and overexposed. They found that in underexposed films, we tend to read more disease that is actually present on the film and that this is particularly true of an inexperienced reader. On the other hand, we tend to read less disease than is actually present on overexposed film. The inexperienced reader usually sees much less in the over-penetrated film than is actually present. So in general in underexposed film we see more disease than is actually present; in overexposed film we tend to read less than is actually present.

So what do we do when we look at a film: We'd like to see that the film is properly identified with a permanent marking. We want to see the identification permanently imprinted on the film. We want to see a film that is properly positioned and nonrotated. We want to see a cone-cut; one that is properly collimated. We want to be sure that the sets are tight so that there are no light leaks. We are going to look for clean screens, screen artifacts, poor screen contact. We want to see that the film is properly exposed and processed. Improper processing where you don't use the proper time temperature development can never be used as a crutch for improper exposure. We're going to look for all types of fogging on a film. Look for the fact that the patient is nonrotated, that the clavicles are roughly equidistant from the mid-line. The patient has taken a deep inspiration. The film is in the erect position.

If you keep all this in mind, with meticulous attention to film quality, your percentage of errors in the reading of the pneumoconioses will fall dramatically. My message today is that film quality is one of our most important problems in the detection of pneumoconiosis.

VIEWBOX CHEST X-RAY DEMONSTRATION, OCCUPATIONAL PULMONARY DISEASES

BY AMERICAN COLLEGE OF RADIOLOGY

INTERNATIONAL CLASSIFICATION OF PNEUMOCONIOSIS
Leonard J. Bristol, M.D.

I'm not going to go into the background of the development of the UC internationally accepted classification of the pneumoconioses. (Figure 1.) But I would like to walk through the radiograph interpretation form used by NIOSH in the first round of medical examination for coal miners with you (Figure 2). You'll see that each division has a number. Number 1 is the "miner's social security number." Number 2 is the "date of X-ray," the month, the day and the year. Number 3 is most important, film quality.

The films are gradual +, exceptional quality; +, fair; +, just barely readable; and UR, unreadable. We have been preaching as we go around the country, to the radiologists that if in their opinion the film is not up to snuff that it not be coded, not classified, and that it be UR. Because if we do read a film of poor quality, three things happen. First, we are promoting poor film quality technique. Second, we are not being fair to the individual whose X-ray we are reading. And third, we are certainly reducing the credibility of the medical profession.

Moving on to number 4 and the classification of the film for pneumoconioses: "Is film completely negative?" If, in your opinion, the film is completely negative, then we ask you to put an X where it says "Yes." Either 0/0 or 0/-. I stated earlier that this is an international classification. The 0/- which is on the left was included in the classification at the insistence of the British, because they felt that some films are of the barn door normal type, meaning that they are so normal that you can't miss it. However, we in the United States very seldom use that category although there is no reason why we can't. We usually classify them as 0/0 if the film is completely negative.

If the film is negative in your opinion, then we ask you to forget about everything else on the page and to go right down to number 15 at the bottom, the last line where you sign your initials, your social security number, and the date you read the film. You have completed the work for that particular chest roentgenogram.

However, if in your opinion the film is not completely negative, you are asked to put an X in the "No" box in item number 4, and once you commit yourself there, you must fill out the rest of the form. It's not a big job. This classification has been tried for all forms of pneumoconioses and is reproducible. All pneumoconioses don't depict the same kind of opacity so as we proceed, you will see that we are characterizing different kinds.

Box number 5 is labeled "small opacities, rounded." This means that there are present on the X-ray film small rounded opacities. There are three different types of small rounded opacities according to their cross sectional diameter. There are p, q, and r types of small rounded
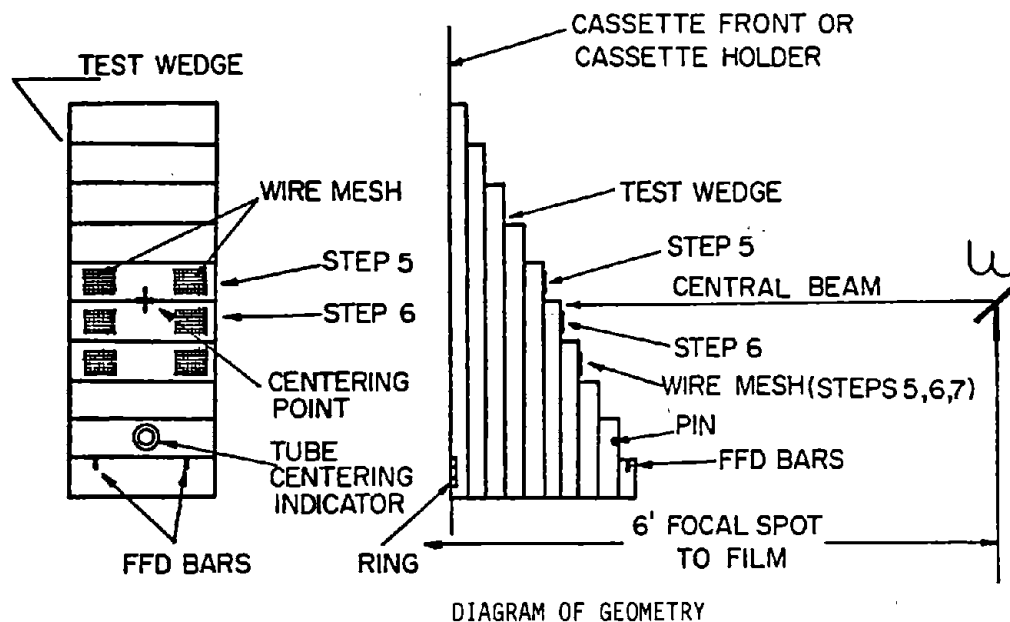
RADIOGRAPHING THE TEST WEDGE

DIAGRAM OF GEOMETRY

1. PLACE THE WEDGE ON ANY LEVEL SURFACE THAT WILL PUT THE FLAT SIDE OF THE WEDGE IN CONTACT WITH THE CASSETTE OR THE CASSETTE HOLDER. THE SUPPORT MAY BE A BOX, STOOL, ETC. DO NOT TRY TO HANG THE WEDGE FOR IT IS VERY DIFFICULT TO DO SO AND KEEP IN CONTACT WITH THE CASSETTE OR CASSETTE HOLDER.

2. CENTER THE USEFUL BEAM AT THE CROSS BETWEEN STEPS 5 AND 6.

3. USE THE TECHNICAL FACTORS THAT YOU WOULD USE FOR AN AVERAGE PATIENT, MEASURING 22 CM.

4. PROCESS AS YOU WOULD ANY OTHER CHEST RADIOGRAPH.

5. RECORD THE FACTORS USED ON THE DATA SHEET.

6. RETURN THE DATA SHEET WITH THE RADIOGRAPH.

REQUISITES FOR ACCEPTABLE RADIOGRAPHS

a) ALL TEN STEPS SHOULD BE DETECTABLE IN A RADIOGRAPH HAVING ACCEPTABLE DENSITIES.

b) ACCEPTABLE TUBE CENTERING WILL BE INDICATED BY THE IMAGE OF THE PIN FALLING WITHIN THE IMAGE OF THE RING. WHEN THE IMAGE OF THE PIN FALLS ON THE IMAGE OF THE RING OR OUTSIDE THE IMAGE OF THE RING, TUBE CENTERING IS NOT ACCEPTABLE.

FOR CONSISTENT TUBE CENTERING FROM PATIENT TO PATIENT, SOME SYSTEM MUST BE ESTABLISHED TO RELATE THE POSITION OF THE TUBE TO THE CENTER OF THE FILM. VISUAL SIGHTS RARELY ARE ADEQUATE. THE BEST SYSTEM IS A HEIGHT SCALE ON THE FILM HOLDER AND ON THE TUBE SUPPORT.

c) ON AN ACCEPTABLE FILM, BOTH THE FOCAL-FILM DISTANCE (FFD) BARS MUST BE VISIBLE.

d) TO FULFILL THE ABOVE REQUIREMENTS, THE CENTERING CROSS ON THE TEST OBJECT MUST BE CENTERED ON THE FILM.

e) ONLY ONE RADIOGRAPH MADE WITH THE LONG DIMENSION OF THE TEST WEDGE PARALLEL TO THE LONG DIMENSION OF THE FILM NEED BE SUBMITTED.

f) IN CASE OF MORE THAN ONE MACHINE BE SURE TO MARK THE RADIOGRAPH AS TO WHICH MACHINE WAS USED.

FIGURE 1 -- DIAGRAM OF PLASTIC STEP-BLOCK (TEST WEDGE) AND INSTRUCTIONS FOR RADIOGRAPHING THE DEVICE

opacities. Basically speaking the p nodule is a nodule that you can see visibly and does not exceed 1.5 millimeters in its cross sectional diameter. The q are nodules are rounded opacities exceeding 1.5 millimeters; they go up to about 3 millimeters in the widest cross section diameter. And then the r type of opacity are those that are rounded and measure anywhere from 3 millimeters up to about 10 millimeters in their widest cross sectional diameter. All zones of the chest are not equally involved. And also the profusion of opacities varies in different zones.

I will discuss zones first, before we consider profusion. Each chest roentgenogram is divided into six zones, equally divided: two upper, two middle, and two lower. It is quite possible and not unusual for an individual who has pneumoconiosis to have only two zones affected. The entire six zones may not be involved. We refer to them as the right upper, right middle, and right lower zone, left upper, left middle, and left lower zone.

Now, to consider profusion: By profusion we mean the number of densities per area of the lung or the concentration of the densities in a particular zone of the lung. There are international standard films developed to demonstrate mid-category 1, mid-category 2 and mid-category 3. Let's go back and discuss what these categories are.

Category 0 is a normal film in your opinion which means that there are no opacities present. Or if there are, they are so few in number that they do not meet at least the category of 1/1 international film. Category 1 is a small number of opacities definitely present. But they are few in number, and the normal markings of the lung, the pulmonary vascular structures, are still visibly seen. In category 2, there are small rounded opacities more numerous than in category 1, and the normal lung margins are now less easily visible. In category 3, the small rounded opacities are very numerous, and the normal lung margins are now almost completely obliterated.

You'll notice on the left of the form 1/1, 1/2 and a 1/0. What do these mean? When interpreting the chest roentgenograms, it is necessary that one refer to the standard international films. As you look at a film you put it up and you say this film is abnormal. It looks like a definite category 1, but you are not sure. It could be normal. You put up the 1 standard for comparison, and you come to the definite concluson that what you thought was a 1/1 is certainly less than a 1/1. But in comparing it with the normal, it's greater than the normal. Therefore, you classify the film 1/0. This indicates to those who will be interpreting your results that you have classified this particular inidvidual as having pneumoconiosis stage 1 but in the process of reviewing the film, you thought that it could be normal. In other words, it's not a mid-category simple coal worker's pneumoconiosis stage 1. It's less than a stage 1 but greater than a stage 0.

The same thing would happen in category 2. You looked at a film and you say this is a 2/2, but then you said "No," maybe I'm wrong, maybe it's more than a 2/2. So you would get out the category 2 standard film, compare it, and decide that it is greater than a 2/2. But, now you must make another decision. If it's greater than a 2/2, is it a 3? So you pull out the category 3, and you say "No," it is less than a 3. So, you have committed yourself, this ia a 2/3. This means to the individual who will be reviewing your work that in your opinion, this particular individual has simple coal worker's pneumoconiosis category 2, but you did consider category 3. In other words, it's greater than a mid-category 2. This process of deciding greater or less than the mid-category goes on through category 3.

The next box is number 6, "small opacities, irregular." The small irregular opacities are classified as s, t, and u. They are so variable that we cannot give them a numerical category as we did with the small rounded opacities, the p, q, or r. What we say then is more descriptive. The s irregular opacity is fine wavy densities. The t is a little broader in width, and the u more less approach a blotchy appearance. We do have a set of standard films for the small irregular opacities.

When it comes to profusion, we again deal very simply with the concentration or the number of these small irregular opacities located in one particular area of the lung. The lung is divided up, as we did for the small rounded opacities, into six equal zones--two uppers, two middles, and two lowers. The standard film is the determining factor as to whether or not the patient

has category 1/1 or greater. And, you go through the same classification process that you did for small rounded opacities. You make the determination that there are small irregular opacities of a varying size in a particular location of the lung. It might be both upper zones and both middle zones, the lower zones being relatively clear. You will then compare that film with the mid-category 1, mid-category 2 or mid-category 3 standard film, and make your determination. This is category 1, or catefory 2, or category 3; it is slightly less than category 1 or slightly greater than category 1, slightly less than category 2 or slightly greater than category 2, and so forth.

It is the standard films once again that determine whether or not you have a mid-category or above the mid-category in the particular case that you are looking at. This is the same thing we went through before. If you classify an individual 0/1, you are stating that it is a normal film or that the individual does not have definite X-ray evidence of pneumoconiosis. But in the process of reviewing that film you did take out the standard mid-category 1 chest roentgenogram, made the comparison and determined that it was less than a 1. Therefore, it was a 0/1 giving those people who will review your work an indication that it is a rather high 0 film. In category 1, the small irregular opacities are definitely present but few in number. The normal lung markings, as in the small rounded opacities, are usually visible. Category 2, the small irregular opacities are numerous. The small lung markings are usually somewhat obscure, but they can still be made out. In category 3, the lung markings have completely disappeared, and the small irregular opacities are very numerous.

The international standard films that I refer to cannot be bought in this country. You still have to send away to the ILO. I don't think it is difficult. All you have to do is go to your bank and get a bank check. I forget the exact price. It is in Swiss francs. Send that over to the ILO, and there is no problem, except possibly in the time of delivery. If anyone is interested in this work, either on an investigative basis or in actual practice, it is mandatory that the standard films be obtained. The profusion which we talked about here, the category 0, 1, 2, and 3, is based on those standard films.

Next on the form is item number 7, "large opacities." As you know, it is conceivable and not unusual for an individual with coal worker's pneumoconiosis silicosis, and the other pneumoconioses to have small irregular and small rounded opacities coalesce and become large opacities. In the definition of large opacities, we state that they are densities greater than 1 centimeter. That is above the r stage small rounded and u stage small irregular opacities.
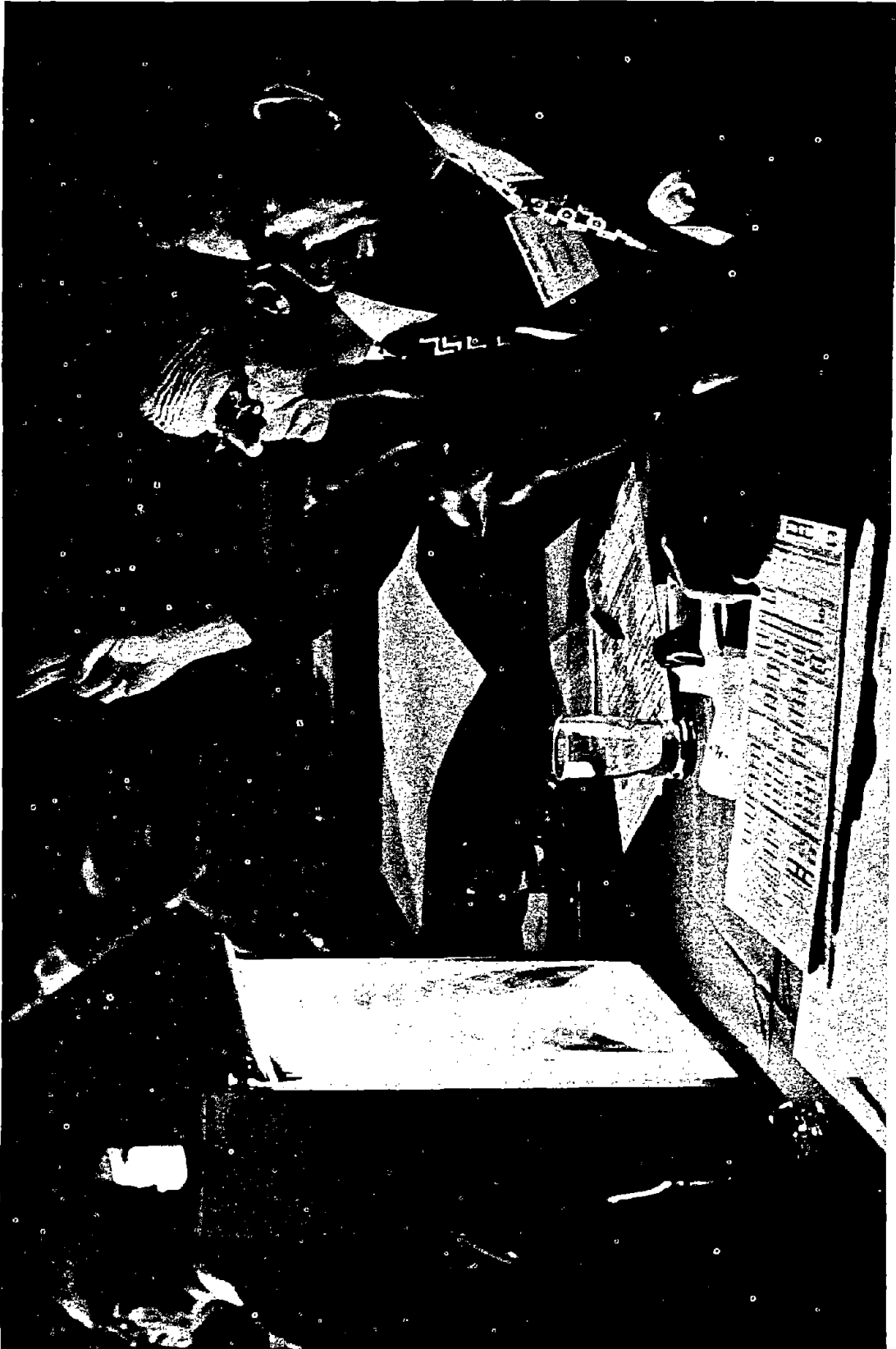
Now we have divided the large opacities into A, B, and C. An A large opacity is one that measures up to 5 centimeters, anywhere from 7 to 5 centimeters in its widest diameter. B is a larger opacity that can fit into the right upper lobe. If you think in your mind's eye, you can take this opacity and fit it into the right upper lung zone, then the opacity would be classified as a large opacity B. Anything larger than that is classified as the C large opacity.

On the form just to the right, you see types WD and ID. The WD stands for "well defined," and the ID stands for "ill defined." A well defined large opacity you can sharply demarcate from the rest of the lung. An "ill defined" large opacity is one for which you just cannot visualize its entire border.

In recording large opacities, it is very important that you believe it is the result of the pneumoconiosis and not the result of tuberculosis, another type of granuloma, or of carcinoma. You will be doing the man a grave injustice if you misclassify a large opacity, because for a large opacity as a result of pneumoconiosis there usually is no form of therapy. But, if in your opinion, it is due to tuberculosis or neoplasm, and you so classify it (on another part of the form which we'll talk about later) there might be some form of remedial therapy, and the individual certainly should be given promptly the benefit of treatment.

There is of course the question of compensation if it is a large opacity due to pneumoconiosis. But compensation is none of our concern. Compensation is left to other people. We are asked to report our findings on an X-ray; we're recording findings to the best of our professional ability, and the possibility of compensation is left for others to judge.

1. MINER'S SOCIAL SECURITY NUMBER

1 2 3 4 5 6 7 8 9 0

MONTH DAY YEAR

+ ± + U/R

FACILITY IDENTIFICATION

4. IS FILM COMPLETELY NEGATIVE?

YES: 0/- 0/0 IF YES, GO TO ITEM 15

NO: IF NO, COMPLETE ITEMS 5-14 AS APPLICABLE AND ITEM 15.

SMALL OPACITIES — ROUNDED

a. TYPE	b. PROFUSION	c. ZONES
p q r	0/- 0/0 0/1	R L
	1/0 1/1 1/2	
	2/1 2/2 2/3	
	3/2 3/3 3/4	

SMALL OPACITIES — IRREGULAR

a. TYPE	b. PROFUSION	c. ZONES
s t u	0/- 0/0 0/1	R L
	1/0 1/1 1/2	
	2/1 2/2 2/3	
	3/2 3/3 3/4	

7. LARGE OPACITIES

a. SIZE: O A B C   b. TYPE: WD ID

8. PLEURAL THICKENING

a. Costophrenic Angle _____ O R L %

b. Diffuse _____ O R L %

c. Plaques _____ O R L %

d. Grade of b/c _____ O 1 2 3

9. ILL DEFINED DIAPHRAGM

O R L %

10. ILL DEFINED CARDIAC OUTLINE

O 1 2 3

11. PLEURAL CALCIFICATION

a. DIAPHRAGM O R L %   b. WALL O R L %   c. OTHER SITES O R L %   d. GRADE OF a/b/c O 1 2 3

12. OTHER SYMBOLS

a. OBLIGATORY _____ O ca co cp es tba od   (SPECIFY od.)

b. OPTIONAL _____ O ax bu cn cv di em hi ho k px rl tbu

OTHER _____ (SPECIFY OTHER)

13. COMMENTS

SEE DOCTOR

YES

(SPECIFY REASON. HAS PRIVATE PHYSICIAN BEEN NOTIFIED?) -

14. OTHER COMMENTS

I understand that all the above information will be held STRICTLY CONFIDENTIAL and divulged only as specified in Part 37 of the Code of Federal Regulations (42CFR).

15. FILM READER'S INITIALS

PHYSICIAN'S SOCIAL SECURITY NUMBER

DATE OF READING

MONTH DAY YEAR

ECA-116 (ON) 3/72

FIGURE 2. NIOSH ROENTGENOGRAPHIC INTERPRETATION FORM USED IN FIRST ROUND COAL MINER EXAMINATION PROGRAM BASED ON UICC/CINCINNATI CLASSIFICATION SYSTEM. (EDITOR'S NOTE: THE INTERPRETATION FORM USED IN THE SECOND ROUND IS BASED ON THE ILO/UC CLASS-IFICATION SYSTEM AND IS SLIGHTLY DIFFERENT.)

Number 8 "Pleural Thickening," is new in the ILO classification; the old ILO classification did not take into consideration the disease known as asbestosis. Asbestosis is notorious for pleural involvement. We'd like you to record whether or not the right and left costophrenic angles are involved in the pleural involvement. If they are, you will put an X in "costophrenic angle," either right (R) or left (L), or if it is both, in the right hand column where it says "both," If in your opinion there is no evidence of costophrenic angle blending, you will put an X in "zero".

If there is Thickening of the pleura along the walls of the diaphragm, this must be recorded in the box identified as b, R if it is only on the right side, L if it is only on the left side, and "both" if it involves both sides. But we also record width and extent. Grade a indicates up to 5 millimeters of thickness at the widest part of the pleural involvement. Grade b is above 5 millimeters and up to 10 millimeters of thickness of the widest part of the pleural involvement, and then grade c is anything above 10 millimeters at the widest part of the pleural shadow. Qualifying this as to extent, grade zero means that there is none present or less than grade 1. Grade 1 is definite pleural thickening in one or more places, but the total length does not exceed one half the projection of one lateral wall. The standard radiograph is the lower limit of grade 1. Grade 2 pleural thickening is far greater than grade 1 and anything above that.

Now we get into the next box, number 9 "ill-defined diaphragm." There are standards for ill-defined diaphragm. The lower limit is one-third of the affected hemi-diaphragm. If the diaphragm is one-third or more affected, then you must classify it as an ill-defined diaphragm. Again the roentenogram in the UC/ILO standard films is the lower limit of grade 1.

The next box, number 10, "ill-defined cardiac outline," is also important from the point of view of asbestosis. You notice we have four grades here, 0 through 3. Zero means it is either absent or up to one-third the length of the cardiac border or the equivalent. Grade 1 means that there is irregularity to the outline of the cardiac silhouette above one-third and up to two-thirds of the length of the left cardiac border or its equivalent. Grade 2 is above two-third and up to the whole length of the left cardiac border or its equivalent, grade 3 is more than the whole length of the left cardiac border. This was referred to years ago as a "shaggy heart," but becuase of international semantics, the word shaggy was dismissed, for the classification "ill-defined cardiac outline."

Number 11, "pleural calcification," was inserted in the classification, because in asbestosis there is evidence of pleural calcification in many instances. This is again graded as to its site and extent. The site may be on the wall of the chest, along the diaphragm, or along the cardiac border. Again, we grade it from 0 to 3. Zero means that there is no evidence of pleural calcification. Grade 1 is in evidence of pleural calcification the sum of whose greatest diameters does not exceed about 2 centimeters. Grade 2, one or more areas of calcification, the sum of whose greatest diameters exceeds 2 centimeters but does not go above the 10 centimeter measurement. Grade 3 is anything above 10 centimeters.

The next box is "other symbols," number 12. At the beginning of my talk, I said, if in your opinion the film was not normal, you would mark an X in "no" at the top of the form. It is possible, in such an instance, that the individual does not have pneumoconiosis. In that case, you would still have to fill out every box down through number 12 putting an X in all spots where it is 0; in small rounded opacities, it would be 0/0, and in small irregular opacities, it would be 0/0. In large opacities, it, would be 0. Pleural thickening, ill-defined diaphragm, ill-defined cardiac outline, and pleural calcification all would be 0.

Now, supposing the individual has large bullous emphysema, but no evidence that you can see of the preceding changes. Then you would X "bu" in number 12 box, indicating that in your opinion this individual has bullae.

Or, if you felt that the individual had an abnormality of the cardiac outline due to size or shape and that's all the individual had, you would put an X in "co" (cardiac outline).

If you felt that there was a pleurisy with effusion, not the result of pneumoconiosis, and nothing else you would put an X in the box "ef".

If the individual has shadows that are compatible with a tuberculosis, in other words areas of increased density in the upper lung zone with cavity formation, and if in your opinion this is active tuberculosis, you would X "tba."

If you saw some fibro-calcific densities in one or both upper lung zones which you felt was probably the result of tuberculosis, but if you were not sure whether it is active or inactive, you would put an X in "tbu", meaning "tuberculosis activity uncertain."

If you saw furry lines, maybe from a cardiac non-pneumoconitic situation, you would put X in the box "k" which would indicate that there is pulmonary septal line involvement.

These "other" symbols are intended mostly for the individual who has a normal chest X-ray for pneumoconiosis, no evidence of pneumoconiosis in your opinion. It is possible, however, for an individual to have pneumoconiosis and other cardiac or pulmonary conditions. These other conditions should be recorded in number 12 in addition to the pneumoconiotic items. Greater detail on these "other symbols" is provided in the Home Study Syllabus available from the American College of Radiology on pages 12 and 13.

Editor's Note:

At this time, the audience turned on individual viewboxes, and an informal discussion of sample films followed. The audience was asked to record abnormalities in the films. Standard films were available.

CARDIOVASCULAR DISEASE IN OCCUPATIONAL MEDICINE

DIAGNOSIS OF CORONARY HEART DISEASE
Nicholas DePasquale, M.D.

Nicholas DePasquale, M.D.: It has become almost a cliche to introduce a paper concerned with the general problem of coronary heart disease with a series of statistical values that point up the enormous morbidity and mortality associated with the disease. To add poignancy to the issue, it is also commonplace to remind the reader that coronary heart disease often strikes down men at the most productive period of their lives and at the time when they are fulfilling the essential role of breadwinner. It is best to resist these temptations and to state simply what everyone knows—coronary heart disease is the major public health problem in the United States today.

The problem of coronary heart disease diagnosis is really several problems. First, recognition of symptomatic coronary disease. Second, identification of patients with presymptomatic disease. Third and by far the most important, the identification of susceptible individuals before coronary artery disease develops. This paper offers a broad survey of each of these problems.

SYMPTOMATIC CORONARY HEART DISEASE

Although an oversimplification, it is clinically useful to separate the manifestations of obstructive coronary heart disease into a spectrum of increasing severity—angina pectoris, coronary insufficiency, and myocardial infarction. In general, coronary insufficiency and myocardial infarction offer few diagnostic difficulties. Regardless of the potential inaccuracy of this statement, the thrust of diagnosis should be directed toward detection of early coronary disease.

Because angina pectoris is the dominant initial manifestation of coronary heart disease, its early identification is essential. Although angina pectoris is the most important early

diagnostic clue, it must not be assumed that it reflects only minor obstructive coronary disease. Angiographic experience has amply demonstrated that patients with relatively mild angina may have severe triple vessel coronary disease.

Angina pectoris refers to recurrent brief episodes of cardiac pain induced by effort or psychic stress but sometimes occurring at rest. Angina may also be precipitated by cold, heat, meals, sexual intercourse, or defecation. The symptom occurs as a result of an imbalance between myocardial oxygen supply and demand and is usually due to obstructive coronary artery disease. The pain may occur anywhere in the chest, arms, neck, or head. It is described variously as constrictive, squeezing, oppressive, or aching. Nevertheless, angina has many disguises--a dull pain in the infrascapular region, an ache in the elbow, an odd sensation in the throat, a vague retrosternal discomfort.

The diagnosis of angina pectoris ultimately depends upon the physician's personal judgment. Because angina pectoris may be so subtle and the patient's complaints apparently so innocent, the physician may be reassured; in turn he reassures the patient who may not seek medical advice again until the signs of coronary heart disease are obvious. It should not be inferred that all chest pain should be interpreted as angina, only that if there is any doubt concerning the origin of the pain, further studies should be carried out to discover its true nature. Chest wall syndromes, pericarditis, esophagitis, and hiatus hernia may sometimes be associated with pain resembling angina pectoris.

It is disturbing that many patients admitted to the coronary care unit are surprised to learn that they have heart disease, when, on careful questioning, it is clear that they have experienced angina pectoris and many have sought previous medical advice. This means that some physicians are not sufficiently adept at history-taking.

Because of the marked propensity of coronary heart disease to shorten life, it is far better to assume that questionable pain is cardiac in origin and to proceed with further evaluation of the patient than to dismiss the symptom as noncardiac. This notion is supported by Kuller's observation that in 28 of 131 (21 percent) instances of sudden, unexpected cardiac death, the victim had no history of heart disease but had visited a physician within one month prior to death (see reference 1). One wonders in how many of those 28 people was a subtle complaint of antina pectoris misinterpreted.

The question of whether proper recognition would have changed the outcome is not the issue. In view of the large number of individuals with undetected coronary heart disease, physicians should be less concerned with making cardiac neurotics out of middle-aged men and should vigorously pursue any clue suggestive of coronary heart disease regardless of how subtle it may be. It is discouraging enough that many potential victims never seek medical advice, but it is calamitous when those who do are not adequately evaluated.

Assuming that the physician judges a patient's complaint to be angina pectoris, where should the diagnostic lead? Continuing with the history, the physician should determine the number of coronary risk factors operative in his patient. Most and sometimes all of the important risk factors can be discovered by a careful history. The more risk factors, the greater the likelihood that the patient has coronary heart disease and that the complaint is angina pectoris. The coronary risk factors are considered in more detail in another section of this paper. Although dyspnea on exertion, palpitation, weakness, fatigue, dizziness, or syncope are nonspecific, a history of these symptoms may reinforce the suspicion that the chest pain is angina pectoris.

The physical examination is of limited but definite value. Presence of a fourth heart sound, or the murmur of papillary muscle dysfunction, or both reinforces the diagnosis. If the patient is examined during an episode of angina pectoris, some very important diagnostic clues may be discovered. A rise in arterial blood pressure may be noted in association with the appearance or intensification of a fourth heart sound, and the murmur of papillary muscle dysfunction, all of which return to the preanginal state after the pain subsides. Sweating and pallor may also be observed during the attack.

Following a careful history and physical examination, the first laboratory study obtained is the electrocardiogram. There are no electrocardiographic abnormalities that are pathognonomic for coronary heart disease. Nevertheless, with some exceptions healed myocardial infarction can be diagnosed from the electrocardiogram with a high degree of confidence.

However, we are emphasizing the diagnosis of early coronary artery disease before more obvious signs such as myocardial infarction have occurred. ST-T wave depression is the most frequent abnormality associated with ischemia due to obstructive coronary disease. Unfortunately, ST-T wave abnormalities are found in conditions other than coronary heart disease. Moreover, many patients with early coronary heart disease have a normal or borderline electrocardiogram.

In order to increase the sensitivity of the electrocardiogram in the diagnosis of coronary artery disease, various stress tests have been devised. Table 1 summarizes our experience with the stress electrocardiogram in patients who had undergone coronary arteriography. The many problems related to electrocardiographic stress testing will not be discussed in detail; however, several points deserve emphasis. Neither the single-load (Master's test) nor the multistage test achieve much better than 80 percent sensitivity. The single-load two-step test is less reliable than a multistage stress test. And, more rigid diagnostic criteria do not improve the accuracy of the single-load or multistage stress test.

Redwood and Ebstein (see reference 2) have offered some interesting data that show the sensitivity and specificity of a careful history to be about the same as the electrocardiographic stress test. Regardless of bias concerning the electrocardiographic stress test, there is no doubt that normal stress responses may be found in patients with significant obstructive coronary disease.

It should be noted that we are considering symptomatic patients in this section. Despite its limitations, the stress test is the only feasible method of identifying individuals with asymptomatic (latent) coronary heart disease, a point that will be taken up later.

TABLE 1.--CORRELATION OF EXERCISE PROTOCOL AND S-T
SEGMENT CRITERIA WITH CORONARY ARTERIOGRAPHY

(Diagnosis of coronary heart disease based on angiographic demonstration of greater
than 50 percent luminal narrowing of at least one major coronary artery.)

EXERCISE PROTOCOL	S-T CRITERIA FOR POSITIVE TEST, mm depression	PATIENTS, number	SENSITIVITY (true positive), percent	SPECIFICITY (true negative), percent	MISCLASSIFIED, percent
DOUBLE MASTER'S (two-step)	0.5	95	68	70	31
	1.0	101	81	50	34
85 percent of maximal heart rate	0.5	79	82	72	21
	1.0	79	83	70	23

The diagnosis of angina pectoris can be made by thoughtful history-taking in the majority of patients. This assumes that the history is accurate and that the patient is neither exaggerating nor denying symptoms for his own purposes. In addition, a positive therapeutic response to a coronary dilator in addition to a program to eliminate or modify risk factors may substantiate the diagnosis.

In those patients under 60 years of age in whom the diagnosis of angina pectoris remains in doubt, we often forego electrocardiographic stress testing and proceed to coronary arteriography. Our reasoning is as follows: If the stress test is negative, it does not definitely rule out coronary heart disease and the question is still open; if the test is positive, it provides no information concerning the distribution of the coronary disease. Because exercise and physical conditioning are important components of a therapeutic program to manage early coronary artery disease, it is essential to know the distribution of the disease. Unless we are certain that an unjeopardized collateral source exists, we are reluctant to recommend exercise. In our opinion triple vessel disease and left main stem disease are not medical but surgical problems, neither of which can be predicted from the exercise electrocardiogram.

PRESYMPTOMATIC CORONARY HEART DISEASE

Recognition of subjects with significant coronary artery disease who have not yet developed symptoms is one of the greatest challenges in medicine today. Despite its limitations, the electrocardiographic stress test is the only means by which individuals with presymptomatic coronary artery disease can be recognized. Although some individuals with presymptomatic disease may have an abnormal resting electrocardiogram, they represent the minority. It would be ideal if all men beyond 40 years of age had a periodic stress electrocardiogram. However, at present facilities for mass screening are not widely available.

What is worse, even if coronary heart disease is suspected, many physicians do not have the capability nor the time to perform a stress test, nor do they have access to a facility that will perform adequate testing. Referral of the patient to the heart station of the hospital for a step-test performed by a technician is usually not satisfactory. Perhaps the commercial stress-testing laboratories that have recently sprung up in many areas will, at least in part, fill the need for stress testing facilities.

In the absence of mass stress-test screening, physicians must develop criteria for referring patients for stress testing in whom they suspect presymptomatic coronary heart disease. It is reasonable to propose that any man over 40 years of age with any two of the three cardinal risk factors--diastolic hypertension, cigarette smoking, hyperlipidemia--should be referred for stress testing. Furthermore, men with one cardinal risk factor and any two of the following: obesity, diabetes mellitus, lack of physical conditioning, aggressive personality, and family history of premature coronary artery disease, should be referred for testing. Although a compromise, if even this much could be accomplished, it would represent a significant improvement over current medical practice.

The accomplishment of this goal requires that physicians search for each of the major and minor risk factors including hypertriglyceridemia. Unfortunately while many physicians are keenly aware of the significance of hypercholesterolemia as a risk factor, too few physicians are aware of the importance of hypertriglyceridemia. In our experience most patients with premature-onset coronary heart disease have hypertriglyceridemia rather than hypercholesterolemia. Even in the matter of cholesterol many physicians fail to recognize that cholesterol operates as a risk factor at serum concentrations greater than 250 mg percent rather than the "upper limits of normal" of 300 mg percent.

The diagnosis of presymptomatic coronary artery disease depends entirely upon the physician's diligence in searching out risk factors, understanding the meaning of these risk factors, and then referring those individuals who are at risk for an adequately performed stress test.

IDENTIFICATION OF CORONARY-PRONE PATIENTS

As in any area of medicine, it is far better to prevent coronary disease than to treat it. A great mass of epidemiologic information clearly suggests, but has not yet proved conclusively, that it is possible to identify subjects who are at high risk of developing early-onset (premature) coronary atherosclerosis. For example, data from the Pooling Project of the Council on Epidemiology of the American Heart Association (see reference 3) have shown that individuals free of hypercholesterolemia and diastolic hypertension who do not smoke cigarettes had a much lower morbidity and mortality from coronary heart disease over a 10-year period than did individuals with any two or three of these traits. In fact, individuals free of these traits had less than one-third the 10-year mortality rate of individuals with two of these traits and about one-fifth the mortality rate of individuals with all three traits.

Consideration of other risk factors--such as diabetes mellitus, obesity, lack of physical fitness, agressive personality, and family history of early-onset coronary heart disease--although less powerful predictors of coronary heart disease is also helpful in identification of the coronary-prone individual. There are not yet sufficient epidemiological data on hypertriglyceridemia, but experience suggests that it may be one of the more powerful predictors of premature coronary heart disease.

Because each of the coronary risk factors can be eliminated or modified, it is essential to develop mass screening programs to identify coronary risk factors. However, unless complementary programs are developed to implement elimination of risk factors, mass screening programs to identify risk factors will be of little value. All of this requires patient and physician acceptance. Unfortunately, many physicians seem to be threatened by screening programs. Furthermore, motivating individuals to eliminate risk factors requires a great deal of time and patience, much more than the busy, harassed practitioner usually is willing or able to give. Until the riddle of how mass screening can be established in the community and the results translated into preventive therapeutic programs without interfering with the private practice of medicine is solved, coronary heart disease will very likely remain epidemic in the United States.

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CARDIOVASCULAR DISEASE IN OCCUPATIONAL MEDICINE

JOB PLACEMENT AND MANAGEMENT OF THE CARDIAC PATIENT AT WORK
Leon J. Warshaw, M.D.

Leon J. Warshaw, M.D.: The diagnosis and treatment of heart disease requires (1) a vast store of scientific information; (2) skill in diagnosis, an integral part of which is the mastery of evermore complicated technical procedures; (3) therapeutic acumen; and (4) the sound judgment that grows out of clinical experience.

However, they do not suffice for the successful job placement and proper management of the cardiac patient at work. This involves a multiplicity of factors, some of which are frequently far more significant in influencing the results than the nature or extent of the heart disease.

The physician addressing this problem needs to supplement his competence in cardiology with understanding of such diverse areas as psychodynamics, social psychology, business management, personnel administration, labor-management relations, industrial hygiene, ergonomics, insurance, and health education. In addition to having sufficiently keen insights from these various perspectives to be able to evaluate their interplay, he must also be able to communicate effectively with those who function within them. Finally--and this is difficult for some physicians--he must abandon rigidity and authoritarianism and continue to work just as hard toward a successful outcome for the patient when his recommendations are modified or even unheeded.

Job placement and management of the cardiac patient at work constitute a single entity, a continuum in which one merges into the other. They involve fundamentally identical considerations at different points in time. Job placement can be viewed as a still photograph, a snapshot of a particular situation at a particular point in time which forms the basis for specific recommendations with respect to a particular individual. In this analogy, management of the cardiac patient at work becomes a full-length psychodrama in which the same process is regularly repeated, and the original recommendations are modified to assure their continuing success as the saga unfolds.

Time limitations require me to make this presentation in terms of common denominators and generalities. However, it would be most unfortunate if this led to decisions based on categoric labels and stereotypes. Each cardiac patient presents his own unique constellation of significant factors with a unique pattern of reciprocal relationships. And, both the factors and their relative importance change with time and circumstance. Clearly--this cannot be over-emphasized--to be effective and equitable, the evaluation must be individualized and objective.

Now, let us focus on some of the factors that most often command consideration.

ATTITUDE

Attitude is probably the most critical factor in job placement of the cardiac patient; often it is far more influential than the nature or extent of his impairment. Anxiety is an inevitable component of every cardiac disorder. When appropriately handled, it reinforces the acceptance of any necessary constraints and promotes adherence to the prescribed regimen. When excessive, however, it can be paralyzing. And, when it surfaces in a pattern of denial, it leads to a lack of caution that can not only threaten the individual's well-being but also can, under certain circumstances, make him a menace to his fellow workers.

The attitude of the patient to the acquisition of heart disease usually stems from his basic emotional makeup. For example, a person with latent dependency now has a singularly acceptable justification for satisfying such emotional needs, while one with latent depression usually exhibits apathy and inertia. However, his attitude is frequently conditioned by the words and actions of those about him--family, friends, co-workers, and personal physician. An over-protective spouse or associates who use him as a vent for their own latent cardiac anxiety may have a devastating effect, if their attitudes are not recognized and overcome. The attitude of his personal physician is more influential for not only is it projected with greater authority but also it determines the limits of activities that are prescribed.

The individual's attitude toward work--toward work in general as well as toward the job for which he is being considered--will also determine the outcome of the placement. Without proper motivation, the patient is not likely to develop the kind of work habits needed for successful job performance. Attitude toward the job is influenced by economic and tax considerations; by attitudes toward the company, supervisor, and co-workers; by union policies and programs; and particularly by attitudes and relationships in the home.

CARDIAC STATUS

Evaluation of the cardiac impairment is a fundamental consideration in job placement. Indeed, in view of Doctor DePasquale's remarks and of Sobin's finding that 10 to 20 percent of all patients referred to cardiac work classification units had no evidence of heart disease at all (see reference 1), it is imperative that the diagnosis be confirmed. In addition to the diagnosis, one must estimate the severity of the impairment and have some notion of the tempo of its progress.

The crux of the evaluation, however, is the functional capacity. How much work will the damaged heart sustain; how much of a load can this individual carry without symptoms and without risk of additional heart damage? What propensity exists toward acute episodes that could endanger the individual or those about him while at work? Then, having determined what the individual can do, one must decide what he should do.

Detailed discussion of how the cardiac status is determined is beyond the scope of this presentation. I will simply emphasize that it must go beyond the static diagnostic labelling of anatomic derangements to include a dynamic evaluation of physiologic mechanisms and functional capacity.

CONDITIONING

Conditioning through a supervised program of physical exercise is a singularly effective way of increasing and maintaining the work capacity of the person with cardiac disease. By more efficient use of energy expenditure, it permits more physical work to be accomplished without increasing the burden on the heart and circulation. It promotes a salutary sense of well-being and lessens the anxiety that the breathlessness and tachycardia of physical effort might otherwise engender in a cardiac patient. Regular physical exercise is also an effective antidote to the undesirable cumulation of emotional stress.

Conditioning to work and to a particular job is also important. Experience has amply demonstrated that the longer the period of disability following a cardiac episode, the less likely is success in returning the individual to work. By acquiring a "Knack" for the job (that is the skill, efficiency, and confidence developed through conditioning in a particular job) the cardiac patient may be able to perform strenuous work with less effort than he might have to spend in doing lighter tasks that are strange to him.

ENVIRONMENT

By environment, I mean the nature of the job, and the conditions under which it is performed, and the prevailing climate of employment.

Certain jobs by nature of their special risk to the individual or the risk to co-workers and the public are "off limits" to persons with heart disease; although there are fewer of these jobs all the time. Thus, patients with hypertensive and/or arteriosclerotic heart disease should not work on high scaffolds or other places where momentary dizziness might cause severe injury. Patients with ischemic heart disease should not be exposed to accumulations of carbon monoxide. All cardiacs tend to do poorly in hot humid environments, while patients with angina pectoris usually do not tolerate cold. In essence, except where the job placement is arbitrarily prohibited by legal or regulatory constraints (as in airline pilots), it necessitates a careful matching of the limitations of the individual to the requirements of the job.

Just as the diagnostic designation is inadequate without detailed evaluation of the patient's functional capacity as modified by attitudes and conditioning, the job classification must be accompanied by detailed knowledge of just what it entails in the particular environment in which it will be performed. This should include not only the physical and environmental hazards in the work place but also such items as the emotional climate in the job and the burden of commuting to and from work.

Where necessary, it may be possible to facilitate the matching of the patient's capabilities and the demands of the work by modifying the job. This can be done temporarily as the patient completes his rehabilitation following an acute cardiac illness or permanently to allow the cardiac to maintain employment that will benefit him, his employer, and society. Such modifications may include changing the work to reduce the muscular effort it entails, changing hours to avoid peak rush hour commuting problems, or allowing rest periods strategically interposed between periods of activity. Brouhà (see reference 2) has shown, for example, that modifying the environment in which the rest pauses are spent may further enhance their restorative value.

Such adjustments, however, may not be sufficient to overcome an adverse employment climate. The general state of the local labor market, the demand for the particular skills offered by the cardiac patient, his prior relationship with the employer, and the constraints specified by a current labor-management agreement--these may favor or prohibit the potential placement. The employment climate may also be influenced by the employer's perception of the threat of (what he regards as) a too-liberal interpretation of local workmen's compensation laws and by his concern over the possibility of excess drain on company-sponsored life insurance, disability and medical care coverage programs.

PREVENTIVE MAINTENANCE

The cardiac patient's adherence to a well-designed regimen, planned not only to enhance his current capabilities but also to minimize further advance of his heart disease, should be given considerable weight in evaluating his suitability for job placement. The availability of periodic re-evaluation by his personal physician, by the plant physician, or preferably by both, should help to assure continuing productivity on the job. The elimination or control of proven risk factors (such as cigarette smoking, hyperlipidemia, and elevated blood pressure) may justifiably be considered a requirement for employment. Although, as Klein (see reference 3) points out, the preventive value of exercise programs is not yet proved, there is sufficient evidence to include it in the preventive management of coronary heart disease. As noted above, regular exercise is helpful in maintaining an enhanced capacity for physical work.

TRIAL ON THE JOB

The multiplicity of factors and their almost infinite variability should make it obvious that one cannot be too rigid or precise in defining just what a particular individual can and should undertake. Although our knowledge of heart disease has advanced and we now have sophisticated techniques of physiologic testing, the ultimate factor is an actual trial of the work itself. This was pointed out by Paul Dudley White over 50 years when he said,

> The only sure way to determine whether a man with heart disease can stand a certain job is actually to try him out at it. No two jobs are exactly alike, and no two individuals have exactly the same kind or degree of heart disease... Having by observation, examination and some simple test roughly determined the fitness of the patient, the exact measurement of his strength, cardiac and general, in relation to a certain job must come from the job itself. We must remember, moreover, that it is possible to train a man for a job physically as well as mentally, although he appears incapable at the first trial. (see reference 4)

CONCLUSION

The first letters of the factors I have emphasized form a word that I pose as a challenge both to the practicing physician who cares for the patient and to the physician who evaluates his suitability for employment--ACCEPT.

1.) To ACCEPT the concept, already amply validated by wide experience, that most cardiac patients when properly placed can and should work productively and effectively without danger to themselves, their co-workers, or the public, and without undue liability to their employers.

2.) To ACCEPT responsibility for promoting in each cardiac patient an optimal capacity and motivation to work, and for encouraging the adoption by the employer of policies that favor hiring and continued employment of the cardiac patient, and

3.) To ACCEPT responsibility for individualized and objective application of the principles outlined above in evaluating the employability of the cardiac patient, in developing realistic and practical recommendations for placing him in a suitable job, and in monitoring both his cardiac status and his performance as long as he continues to work.

A final footnote--although I have addressed the problem of the cardiac patient, the same principles are applicable to all categories of disease that cause, or threaten, significant impairment and disability.

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CARDIOVASCULAR DISEASE IN OCCUPATIONAL MEDICINE

OCCUPATIONAL HEALTH SERVICE, SEVEN COMMON PROBLEMS
E. R. Plunkett, M.D.

E. R. Plunkett, M.D.: Consideration of day-by-day problems of the cardiac at work is a nice broad subject. To use a speaker's liberty, then, I should like to sub-divide this into sections that will allow isolation of problems common to all of us.

<center>EMPLOYMENT STANDARDS</center>

In the case of regulatory agencies, such as FAA and ICC (see reference 1), both preemployment and periodic cardiac standards are published and enforced. They are probably followed in most cases. The major corporations have the benefit of years of experience and competent medical advice. It is, however, that large group of small plants with part-time or on-call plant physicians who may lack consistent standards within their medical programs, to which I address this paper.

Some of you may have read the survey made by Doctor Weinstock and published last month in The Archives of Environmental Health (see reference 2). He gathered data on the effect of illness on employment opportunities from 45 company locations in a metropolitan area. To quote from his summary, "Patients with a previous myocardial infarction (MI) had only a 27 to 70 percent chance of being hired. Those with angina, 12 to 59 percent chance; rheumatic heart disease 9 to 33 percent; and hypertension 16 to 59 percent." He stated in his report that the physicians "...were asked to answer in terms of the company's policy, and not their own recommendations." To comment on this last statement, I believe it is the obligation of the physician to help set company policies in matters such as these.

However, returning to the study, using Doctor Weinstock's questionnaire, I gathered similar data from a number of corporations in our Pittsburgh-Cleveland-Akron triangle. I found that while the pre-employment rejection rate is a bit more for MI, angina and hypertension, our local firms are able to return to work 80 to 90 percent of their regular employees with these conditions, compared to 60 to 70 percent in Weinstock's study, and this pleased me. I cannot explain this 20 percent difference. It may have to do with workmen's compensation laws, physician influence on corporate policy, health insurance, and retirement benefits, or other factors.

Doctor Weinstock suggested that one solution to such a high rejection rate might be to form panels of "arbitrating" physicians who could examine an applicant who thought he had been

wrongly rejected. I do not agree and hope our discussion period will allow it to be further explored.

More and more individuals are undergoing routine health appraisals--FAA, athletes, hot rodders, job change, insurance, regular company exams, and so forth. This trend will grow and will afford all of us an increased opportunity to detect early changes. Thus, while there will be an enlargement of the population at risk, those of us doing the routine examinations will see these people much more often than the regular family physician, and it behooves us to exercise the opportunity for early diagnosis and correction. Similarly, with federal intervention on the work environment, as well as an increased social consciousness on the part of management, I think we shall see the time in which employers are more willing to hire their fair share of the handicapped.

In the final analysis, however, employment standards should be set by the physician who has or can acquire details of the job demands, and such standards should be in keeping with the pre-servation of the health of the worker.

FAIR EMPLOYMENT PRACTICES

Both state and federal regulations now require employers to "Take affirmative action to employ and advance in employment qualified handicapped individuals, i.e. there shall be no discrimina-tion because of disability." In most cases the laws allow disqualification if the health or safety of the individual or others is at risk. Unfortunately, we have no laws requiring cooperation of the individual. Let me review briefly two cases given to me by our local Board of Vocational Rehabilitation.

Case 1: A 40-year-old, chainstore-department manager who suffered a coronary in mid-1970 followed by a cerebral vascular accident. He has had openheart surgery with excellent results and is now asymptomatic except for very slight right sided residuals from the cerebral vascular accident. His old job is available, and his doctor has released him to work, but he will not return. His present income is $911 per month tax free; his wife is also employed; and his hospitalization covers 80 percent of all medical bills.

Case 2: A 55-year-old truck driver with 23 years experience. In May of 1972 he had a coronary and has recovered nicely. He has been approved to return to work. He no longer meets Interstate Commerce Commission standards, however, and his company has no alternate job. Be-cause of his approval to work, he has been denied Social Security benefits. He has successfully completed a welding course, but because he has no experience cannot find work in that capacity. He receives $160 per month company pension, and last week took a job as a handyman at $2.35 per hour.

WORKMEN'S COMPENSATION

In the June 1973 issue of Modern Concepts of Cardiovascular Disease, Mr. Robert Dalenberg authored an article entitled, "Coronary Heart Disease and The Law "(see reference 4). It is an excellent article covering the subject, and he observes that 12 states adhere to the rule that only unusual exertion may give rise to a compensable heart attack while 20 states will grant compensation where the attack does not involve unusual activity, and concludes that this latter liberality is the trend. He makes one very important statement pertinent to our discussion today: "The idea that awarding compensation may cause a class of heart patients to be unemploy-able has not had substantial impact on the courts."

In my state of Ohio the workmen's compensation people at the Akron office tell me that cardiac claims are less than 1 percent of the total and that most of them are disallowed. Where they have been allowed, the employers have been afforded relief under our handicapped worker's law where the bulk of the award is paid from the general fund.

TOXIC MATERIALS

Jean Stellman in her book Work is Dangerous to your Health (see reference 5) cites thirteen specific chemicals which she relates to heart disease as well as two physical agents. In some cases I would take exception to her conclusions, but there are considerations which must be given the individual with heart disease who is to be exposed to some chemicals.

Myocardial depressants such as aniline and nitrobenzenes (MET-Hb) and irritants such as ethylene, chloroform, trichloroethylene.

Agents with vascular effects. Azides produce severe vasodilation, carbon disulfide causes atherosclerosis, nitroglycerine and ethylene glycol dinitrate produce catecholamine release.

Some chemicals have profound systemic effects that could jeopardize the individual with diminished cardiac reserve, such as carbon monoxide, cyanide, insecticides (cholinesterase inhibitors), butyraldoxime (antabuse-like reactions).

Acute pulmonary irritants such as ammonia, chlorine, phosgene and the chloroformates may be quite hazardous to the cardiac. And, for the sake of completeness, mention should be made of the chronic cardiac effects of the pneumoconioses.

While Stellman suggests that noise and microwaves may increase the incidence of heart disease, in practice I am more concerned with the effects of heat and cold and, of course, electrical effects on pacemakers.

Care must be exercised in assigning cardiac patients who work with chemicals, and often it becomes necessary to educate the patient as well as the family doctor.

INDIVIDUAL AND HIS FAMILY DOCTOR

As was pointed out in the book The Heart in Industry, (see reference 6) edited by Doctor Warshaw, 70 percent of our cardiacs fall into Class 1 or 2 (above) and so do not need special job placement. Now and again, however, problems arise with the individual or his personal physician. It is possible for the outside physician to make an incorrect diagnosis of heart disease or to produce iatrogenic heart disease. While this situation is sticky, it can and should be handled with honesty and dispatch.

Again to quote from Doctor Warshaw's book, "The problem case is more apt to be the person who has some impairment but who: (1) lacks motivation; (2) cannot be retrained; (3) cannot be moved to another job because of union rules; or (4) does not suffer any income loss by electing disability."

The determination of the final job assignment should fall to the plant physician alone, and he should not be compromised by his company or the outside physician. This is not to suggest that the family doctor should not be consulted, but just as the plant physician would not dictate the digitalis preparation to be used, the family doctor should not dictate the specific job.

Doctor Warshaw has earlier suggested that the plant physician, by more frequent contact with the patient and a continuous evaluation of the job, is in a better position to judge the patient's adaptability.

UNIONS

In dealing with over 70 companies and their respective unions, I find if the plant physician does a better job of examining the employee than the family doctor, if he is more knowledgeable about the job content, and if he spells out realistic limitations, that we can avoid 90 percent of the union arguments that we used to get. Consistency is important also, and unions soon learn when management has asked a plant physician to be liberal toward a good employee in decisions on workmen's compensation or disability retirement.

Here, too, we shall see increasing demands for periodic health appraisals as part of the bargaining agreement. The health maintenance program of the rubber companies is a prime example. There is a need for better data gathering as it pertains to work environment and health effects, but often the physician must ask for it.

SUPPORTIVE MEASURES

Obviously, resuscitative equipment and emergency procedures need to be established at all plant locations. In the August issue of Emergency Medicine, Dr. William Greene from the University of Rochester makes another point that seems important. He suggests that the Buddy System for cardiac patients in the plant may be useful--someone to whom the man may turn if he is frightened or worried, someone who understands what steps should be taken.

SUMMARY

The plant physician then must (1) must educate the employer, (2) help identify proper jobs for cardiacs, (3) assume a responsible role in the early detection of heart disease, (4) educate the employee about his condition and the need for regular outside care, (5) help the company manage the non-compliant patient, (6) be firm with the union and management, and (7) if necessary, inaugurate supportive and referral procedures.

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CARDIOVASCULAR DISEASE IN OCCUPATIONAL MEDICINE

PREVENTION OF CORONARY HEART DISEASE
James A. Schoenberger, M.D.

Since time is limited, I will start with some conclusions. In my judgment coronary disease, with all its ravages, well known to all of you, can be prevented. This is not something which can be proven, but I think it is reasonable and prudent to adopt measures of intervention to prevent the clinical manifestations of coronary disease. Further, it is my contention that there is a vast pool of people who are at very high risk of developing coronary disease, who are unaware of their predicament or who are inadequately treated by their personal physician. And, finally, I believe that an exciting new area is opening up for industry to play an active role in this prevention. I hope today if time permits to tell you about a project which we have carried on with the Automatic Electric Division of General Telephone and Electronics in Chicago as a practical demonstration of coronary disease prevention in industry.

Now, I've made some broad claims, and I suspect you are entitled to challenge them. I will try to document what I am saying. Turning to the slides, I will review the background for the statements that have been made about risk factors and in particular justify the concept that we can control them.

First, we will turn to some data from international studies by Ancel Keys which really initiated the concept that we should be able to identify the man who is going to have a heart attack long before the disease manifests itself. This is based on the fact that there is a wide variation in prevalence of heart disease in different countries. The highest prevalence is in Finland, compared to the lowest in Japan, Greece, and Yugoslavia.

Why should there be this difference? Much has been said about physical exercise. I would like to dispel it right now. I think there is no evidence that it makes any difference. The Finns have a low rate of physical inactivity; yet their physical exercise has not protected the Finns in any way from the development of coronary disease.

How about obesity? Another popular concept, that fat people have more heart attacks, doesn't seem to be true. The Finns--hard working as they are, running out from the sauna into the snow and keeping themselves lean--still they have more heart disease than anybody else. I think that, all other things being equal, obesity by itself does not predispose to heart disease. Well, what does?

Here I think are some data that are fairly convincing. These are cholesterol values, (slide presented) a percentage of men with a value of greater than 250 milligrams per deciliter. One can see an excellent correspondence between the level of cholesterol percentage of men exceeding this arbitrary value and heart disease. It is a correlation which cannot be ignored. Why should

there be more cholesterol in the blood of Finns than in Japanese? Do you think diet has something to do with it? I do.

I know this is a controversial subject; however, the amount of saturated fat in the diet clearly has a great deal to do with the prevalence of coronary disease as you can see. (slide presented) An excellent fit.

Now going on to another one of the so-called major risk factors, cigarette smoking. I think the cigarette manufacturers must be very happy; however I am unhappy, because these data (slide presented) from a look at the world wide epidemiologic data fail to support the idea that cigarette smoking has anything to do with heart disease.

I think it can be said that we're dealing with a multi-factorial disease, and that, therefore, what may be an important factor in one country may not be in another. But clearly on the international level, cigarette smoking does not correlate well with the distribution of coronary disease. Here are blood pressures shown on the top systolic and on the bottom diastolic. (slide presented) I think there is a fairly good fit that there is more diastolic hypertension in the countries that have higher prevalence of coronary disease and a less well correlated fit for systolic pressure.

Now, turning from international data to data for the United States, here are data from the pooling project that was mentioned before. This is a combination of data from a number of prospective studies going on in this country for many years. The Framingham Study, the Western Electric Study, the Tecumseh Study, and others--all well known to you. I'm going to show you that in the United States, contrary to the world-wide data, smoking has a great deal to do with whether you develop a heart attack. Here you can see the risk of developing a first major coronary event. (slide presented) It is lowest for those who never smoked and a little higher for those who are former smokers. There is steady rise in the prevalence of coronary disease, either first major event or sudden death, associated with increasing amounts of cigarette smoking. Note that there is already a demonstrable increase, 60 per 1,000 for those who smoke less than a half pack of cigarettes. So don't deceive yourself that small amounts of cigarettes are safe. You can see the rate goes up astronomically for people who smoke more than one pack a day. There is a striking correlation between the number of cigarettes smoked and one's chances of dying.

It is clear that there is an increased risk of developing coronary disease for even very modest elevations of blood pressure. One of the tragedies of our times is the fact that most physicians take a very cavalier attitude about hypertension. A diastolic blood pressure between 85 and 94, already increases the rate from 48 per 1,000 to 87 per 1,000. It nearly doubles. This at a level that I think most doctors would not bother to treat. The same is true for sudden deaths, for all cardiac deaths, (slide presented) and as you can see, all of the incidence in this age group was low for stroke, but the coronary rate goes up three fold or more with rises in diastolic blood pressure. So, clearly blood pressure in the United States is related to premature development of coronary disease.

If one looks at combinations of risk factors, those who have one, two and all three of the major risk factors--smoking, hypercholesterolemia, and hypertension--have greatly increased risks of premature coronary disease, sudden death, total mortality. (slide presented) This slide is an extract of a table from the Risk Factor Guide, a publication of the American Heart Association. Everyone in this room ought to be aware of the fact that one can identify with a mathematical probability which is almost uncanny, the risk of the premature development of coronary disease by taking into account a combination of risk factors. Looking at those who are not smokers, there is a steady rise in the probability of the development of coronary disease for a 50-year-old man going from 2.5 percent in 6 years if his cholesterol is 185 up to 6.4 percent if his cholesterol is 310. One can see a steady progressive increase in probability with rising cholesterol.

Similarly for rises in systolic blood pressure, which is equally as accurate a predictor as diastolic blood pressure. And then if one has combinations of systolic hypertension and elevation of the cholesterol, this non-smoker can increase his risk of coronary disease as much as five fold going from 2.5 percent for the lowest risk, to 14.8 percent. This is actually double the

risk of the average man of this age in the United States.

Smoking cigarettes increases the risk by at least another 50 percent; 14.8 percent for the high risk man who doesn't smoke becomes 21.4 percent for the man who smokes. Now, it is not to be construed that changing these risk factors will by itself lower a man's status; because risk is something that is proportional to the length of exposure to the factor. It makes a difference whether you smoke cigarettes a month or 10 years. Similarly with hypertension and with the hypercholesterolemia. All of these factors are noxious influences on the vessels which exert their bad effects over time. Discontinuing or changing the risk status cannot be expected to reduce immediately one's risk. Sufficient time must be allowed to accomplish what may be potentially a reversible lesion. We know from animal experimentation that it is up almost to the point of fiber scarring, and there is every reason to believe that it may be for humans.

These data can be looked at another way. Given any combination of hypertension and elevation of the cholesterol, a non-smoker who had a systolic of 180 could be below average risk as long as he had a low cholesterol of 185. Similarly, somebody who had a low blood pressure begins to approach average risk as his blood pressure goes up. One can compensate to some extent for increased risk by judiciously giving up those habits or characteristics of the risk score which can be controlled by the individual. Perhaps the crux of this is that the individual himself must participate in the decision making and must modify his life style.

This is a summary of what I am talking about, (slide presented) from the Framingham data in which individuals were grouped according to a number of characteristics into deciles. At the top the highest 10 percent going, at the bottom to the lowest 10 percent of the 258 men who had coronary heart disease over the 12 year period, 126 came from the top 20 percent. This is a justification for my stand that we can identify the individual who is at high risk and that we hopefully can do something about him.

Now I will present some data from the Chicago Heart Association's project in industry: A pilot demonstration project, it is designed to determine if we could improve on the fact that about 175,000 of the more than a million deaths in this country from cardiovascular disease are premature. They occur in the labor force in men under the age of 65.

We were motivated strongly by the fact that coronary disease is extremely lethal, that 20 percent of individuals who have a myocardial infarction die before getting medical care. The irreducible minimum death rate in the hospital even in the best of coronary care units, is somewhere between 15 to 20 percent. Recurrent infarction and death takes about 5 percent a year so that at the end of one year, coronary disease has exacted a toll of about 40 percent in terms of mortality. It is far more lethal than cancer. This justifies the preventive approach.

There is no other way to look at the coronary disease problem in the United States except for the ultimate development of a preventive approach. We have begun to look at this in Chicago.

This is prevalence data on hypertension among industrially employed people. (slide presented) We had some remarks earlier about screening to detect people who have hypertension. Unfortunately, people who go to work at age 25 and have normal blood pressure may not have normal blood pressure when they are 45. Nobody as a rule seems to look at them. We have no data as to what happens in the intervening 20 years. There is a lot of hypertension. There is more of it in men at every age group, and there is more of it among black men and black women than in white men and white women.

It becomes almost astronomic when you see nearly 40 to 50 percent of the work force between the ages of 55 and 65 with blood pressures of 160 over 95 or greater, a significant hypertension. What is even more distressing is the fact that the majority of these patients have no awareness. But, of the 19.7 percent of the white men with elevated blood pressure at our screening examination, 13.4 percent or roughly 60 percent of those with hypertension, 60 to 70 percent denied any knowledge of being hypertensive. This is extremely upsetting, because a recent survey by the Harris Associates indicated that 77 percent of people in the United States claimed that they had had their blood pressure measured within the preceding year. Somebody's not telling the truth. In some cases the doctor may not be telling the truth, because he isn't telling the patient what

his blood pressure is.

This is my thesis: that if three-fourths of the population has had its blood pressure checked, but 60 to 70 percent deny any knowledge of hypertension, they either have bad memories, or they're denying it, or they were never told in the first place. Most of the hypertensives in the United States are undetected, have no practical knowledge that they have hypertension, or if they are aware of it they are not taking any treatment. Finally, of the few that are on treatment, only about half are successfully treated. It runs about 50 percent, so we have a pretty poor batting average. Doctor Stamler likes to say we have about a half of a half of a half-half of the people are undetected, half of the ones that are detected are on treatment, and only half of those are adequately treated. So we end up with about one-eigth of the total population under adequate treatment at the present time. This amounts to about 20,000,000 people with high blood pressure who are not adequately treated. This is a fact which contributes to the epidemic prevalence of coronary disease in this country.

What evidence is there that treatment is beneficial? I cannot understand why the American medical profession has not accepted the validity of these studies. These are the Veterans Administration studies on the value of treatment. (slide presented) Among the individuals with diastolics of 115 through 129 after 18 months the disasters that were occurring in the control groups on placebos, the 27 serious events leading to death or termination, were almost entirely avoided in the 73 men who were on adequate treatment. From a moral standpoint, a study which had been planned to go for much longer had to be discontinued because of the disastrous effects of untreated hypertension. For milder degrees of hypertension 90 to 114, this data shows by the life table method that it took 5 years to demonstrate without doubt that treatment made a difference. (slide presented) All morbid events, which would include all complications from hypertension, occurred in 55 percent of the untreated men in the first 5 years, and in the treated group only 18 percent had comparable complications. I think the benefit is clear. Only time will tell whether this view is right. My thesis would be that in those individuals who have other concomitant risk factors, even milder degrees of hypertension should be treated.

Our surveys show the prevalence of hypercholesterolemia, defined as 250 milligrams per deciliter or greater, to be significant and to go up with age. Overall, somewhere between 7 to 15 percent, both white and black men and women have elevated blood cholesterols and therefore are at least at double risk of developing coronary disease. And just as with hypertension, the vast majority, somewhere between 80 to 90 percent, denied any knowledge of hypercholesterolemia. This would indicate either that doctors don't routinely measure cholesterol; or that if they do, don't inform the patient or give him any advice about it.

Part of the problem here, I think comes from a noxious influence on American medicine; the SMA 12 gives us normal limits of cholesterol as 150 to 300 milligrams per deciliter, a grievous error in my judgment. I think anything over 225 is dangerously high. The problem is not what is normal in America but what is normal for man. We should learn in those countries that have cholesterols of 150 to 160 and low prevalence of coronary disease, and not be lulled by the fact that a cholesterol of 225 is average or normal in the United States. Biologically 225 may be high and a reflection of a very faulty diet.

A lot of people smoke. Black people smoke more than white people. As you can see 57 percent of black men were still smoking, 42 percent of white men. The prevalence of smoking is going down in certain groups. Both for white and black men in the older age groups there are fewer smokers than for the younger age groups. Certainly women are beginning to catch up with men not only in the number who smoke cigarettes but also in the number who are developing cancer of the lung and premature coronary disease.

Thirty years ago coronary disease was rare in women under the age of 50. Today in Framingham one out of four of those between the ages of 45 and 50 who has a myocardial infarction is a woman. The number of people who gave up smoking was considerably higher for men than for women. Fewer women in the older age group have quit. I think this may be of great importance in the future assessment of heart disease. Now finally, data showing, on the basis of 40,000 examinations made by the Chicago Heart Association, that the numbers of individuals having two or more risk factors goes up with age. For those at very high risk 2 to 3 percent of the population is involved.

Somewhere between 10 to 20 percent are at very high risk in the ages of 55 to 64.

Carrying out these surveys in 40,000 employed Chicagoans working in over 100 industries, we sent letters at the time of the examination to individuals urging them to seek medical care for evaluation. If we didn't hear from them regarding an effective referral, we sent a second letter and finally a third letter. There was a disappointing lack of effectiveness. For white men, 43 percent of the younger and 64 percent of the older did go to a physician. The figures are comparable for white women, and are strikingly lower, significantly so, for both black men and women. So the simple expedient of referring people for follow-up care is not all that is necessary. People who are planning screening programs in industry should be mindful of the fact that follow-up is the essential ingredient.

The effective referral rate was higher for people who were told they had a high blood sugar (64.5 percent) than it was for people with high blood pressure or hypercholesterolemia. The point is that the general public is well aware that diabetes is a life long chronic disease, and they don't want it but that they are unimpressed with the dangers of hypertension. I think most people in the American public don't even know what the word means. Since they have no symptoms, they are not inclined to see a doctor about it. Likewise, hypercholesterolemia has limited meaning to the American public.

Now, the effective referral on blood pressures in a re-screening examination which we did. (slide presented) Almost all of the screening studies reported have not bothered to look at what they accomplished in the most critical way, which is to re-screen the individuals who were initially referred. We had 105 individuals in this group with diastolic blood pressures of 105 or greater, and on rescreening there were 180. This reflects in part our inability to get the hypertensives into effective treatment by the medical profession, but also since this rescreening examination occurred 1 to 4 years later, it reflects new cases of hypertension. Here you can see that if we did get somebody to the doctor, the prevalence of individuals with 105 diastolic or greater was 27 percent. Among those who did not seek a physician, it was still 43 percent. Even though the accomplishments were modest, I think they're far ahead of doing nothing about blood pressure.

For cholesterol there is a very interesting phenomenon in contrast to hypertension. On the rescreening, whereas there has been 220 with cholesterols of 250 or greater, there were only 123 on rescreening; a significant drop. This may represent regression toward the mean, for those of you who are statisticians, but it may also reflect the fact that the screening process in itself is educational. Some of them on their own then took what is generally known about risk factors and diets, adopted it, and lowered their own cholesterol. Similarly, there were 790 who had been current smokers and that went down by a modest degree to 702.

Having found that screening itself and referral with three letters was ineffective, we developed in Chicago a second generation program. The first corporation in Chicago to have the courage to participate with us in the new venture is the Automatic Electric Division of General Telephone and Electronics. The program was designed to include not only screening and referral but also a more active intervention than had been done before. This consisted of intensive counseling of all individuals at high risk, then education regarding risk factors in groups, and finally establishment of smoking clinics where the smokers could go to get some assistance in quitting smoking. And, finally, perhaps the most important ingredient was a surveillance and long-term follow up of the hypertensive individuals. This has proven to be effective in other studies.

I refer you to the data in the classic study by Wilbur and Barrow from Baldwin County, Georgia, which show that at the start of this study 25 percent of the hypertensives in his population survey were on treatment but only 15 percent had good control. These figures correspond to the figures elsewhere. They reflect vast neglect of the hypertension problem. By the simple experience of having a visiting nurse make a call each month on individuals found to be hypertensive, and question them about adherence to drug regimens and adherence to appointment schedules in doctor's offices, they got 86 percent taking medication and 80 percent under control. Finally, when the grant ran out and the nurses stopped making the calls, things changed. Two years later the percentage on drug treatment had dropped to 55 percent and the number on effective control to 29 percent. There is a continuing problem in patient education, the medical profession is doing poorly at the present time.

III-21

At Automatic Electric we screened 1,031 individuals, 60 percent of those eligible. Even though you make the examination voluntary and assure confidentiality of the findings, a very sizeable percentage of the individuals who are eligible refuse to take the examination. Our population at Automatic Electric was almost entirely men, almost all white, and ranged in age anywhere from 21 to 64. Screening should be done on younger individuals perhaps even in the pediatric age group, since that's where the problem may start. This was a very select group, because 98 percent had a high school and 75 percent a college education. Obviously we were dealing with the executive corps of the company. We would have anticipated that this elite group would have known about their blood pressure, that they would be getting annual physical examinations. We found much to our amazement that this was not the case.

It was a sequential screening process; those who were found to be at high risk on first screening were rescreened, and the findings confirmed before they were referred to the doctor. In this highly educated group of men, 58 percent denied any previous knowledge of hypertension. Eighty percent who were found to have a high blood sugar denied any prior knowledge. Ninety-four percent with hypercholesterolemia denied any knowledge.

As a result of our screening of these 1,000 men, we referred 31 percent or 316 for further evaluation after the initial screening. We are now in the process of re-evaluating and following these men on a long term basis.

We offered them four 7-hour classroom sessions to learn about coronary risk factors. We built the education in at the plant. We did it off hours after work. For smoking, 11 of the 28 who went through the whole class are still not smoking. This is a preliminary result after about 6 months as opposed to 14 percent who quit smoking but didn't attend the classes. Those of you who have tried to intervene in smoking know that almost any method you use is effective and that you can get about 25 percent of the smokers to quit at the end of 1 year. We hope to improve that. We hope to get more people involved and coming in to smoking classes as time goes on.

Finally, for hypertension, we found the same thing, about 45 percent who were found to be hypertensive went to the doctor to get it checked out; 52 percent ignored it. Of those who went, less than half were put on treatment by the doctor, and of those who were not put on treatment, about 60 percent were still hypertensive when we rescreened them. Whether they are hypertensive or not is a technical question, because they should be closely followed. Many of them will subsequently develop a sustained hypertension. Again, for those the doctors put on treatment, only about 25 percent were effectively treated. So even though this innovative way of approaching heart disease in industry is encouraging, it seems that the big stumbling block may be in increased physician education and acceptance of the risk factor concept before we can effectively stem the tide of coronary disease in this country.

CARDIOVASCULAR DEMONSTRATION WORK EVALUATION STRESS TESTING

GRADED EXERCISE TESTING
I. Martin Grais, M.D.

My assignment today is to introduce the topic of exercise testing, and I am told that some of you are doing it and some of you are thinking of doing it. Some of you are even possibly opposed to doing it. What I'd like to do is show you one approach, one that I have used both in the clinical practice of cardiology as well as in research in a university setting. I suggest that this is just one approach and that there are many approaches to the problems of exercise testing. Those of you who are thinking of doing exercise testing can evaluate it on its own merits.

What I am going to do is direct my discussions to some of the aspects of equipment used in exercise testing, the methods that can be employed, and perhaps protocol that might be suitable. There are many ways of approaching each of these. I'll just show you one approach to give you something to think about, and to compare with pre-existing concepts and, perhaps, with what some of the other people on the panel are doing and using.

The problem we usually face in clinical cardiology is having a patient who has had some chest pain come in, and he wants to know why he's got this discomfort in his chest. Now, obviously for many of you who are industrial physicians, the problem is going to be different but even so you may from time to time be dealing with people who are already symptomatic.

Years ago we had available only one type of method for exercise testing, the two step. (slide presented) I know this slide is blurred, but it is purposely blurred, because that's often the way the patient looks to me when he is doing the two-step test. He is holding the electrode cable in his hand: he is going back and forth over the steps. It is very difficult to do anything with him while he is doing it. It would not be surprising that other methods of exercising him might have been tried along the way. However, there is a way of doing the two-step test satisfactorily with monitoring during the test which I think improves the two-step test and makes it clinically useful.

One of the things I have noticed in doing two-step tests on people is that you usually can't monitor them in the conventional way. There are some terrible things that can happen to people during exercise testing which can only be demonstrated by monitoring and perhaps some of the slides I have will illustrate this.

The purpose of the exercise, whether it is the two-step or the treadmill, the bicycle ergometer or the escalator ergometer, or just any sort of exercise is to increase the demand of the heart

for oxygen. It's strictly a question of supply and demand. You have a fixed supply which is generally the case with the coronary arteries, and you increase demand by increasing the heart rate, the blood pressure, or the inotropic state of the myocardium. If supply cannot keep up with demand then this whole thing tilts in such a way that demand is greater than supply, and you may begin to see some changes in the electrocardiogram. This isn't always the case, but presumably that's what all of these tests are doing. They're increasing the individual's heart and blood pressure, increasing therefore the amount of oxygen the myocardium is demanding, and if the coronary arteries can't keep pace with it you begin to see changes in the patient and in particular in his electrocardiogram.

Through the years, other methods have come along besides the two-step test. One of them is called graded exercise. I like this term myself, because I think the term graded-exercise test reflects exactly what you are doing. You are grading the amount of exercise to the individual's ability to perform that exercise. You are not making him do too little or too much. He's doing just the right amount of exercise to give you the information or for you to assess his functional capacity.

There are a number of protocols for exercise testing, but the one that is used in most of the exercise labs that I have visited utilizes a target heart rate. This is the rate to which you intend to exercise the individual, and it is based on age predicted heart rates which were established in studies reported in the 1960's by Lester et al in Circulation. This is a line which shows the maximum heart rate that can be generated if you exercise people in these age groups to exhaustion (slide presented). As you can see, a 25 year old can get up to a heart rate of approximately 200 whereas, a 60-year-old might generate a heart rate of approximately 160. We do not exercise people in the graded-exercise test to the maximal heart rate. Instead we arbitrarily select 85 percent of that heart rate as the target rate to which we intend the patient to go and beyond which we will not let him go more than 8 beats per minute. I believe that there are ample data to show that this is a safe and effective way of exercising people.

There are individuals who prefer to go above this level and feel that they increase the sensitivity of the test. This is one area of hot debate in the field of exercise testing. The protocol of exercising people to 85 percent of that rate is used in most of the laboratories around the country that I am familiar with.

Now if you put someone on a treadmill and exercise him, you can find one or another target at which you would stop the test. One is to get him up to that heart rate I've just shown you and maintain it for 2 minutes. Another criterion is that the patient develop some symptom or sign that would indicate that the test be terminated. If the ECG monitoring system fails and we are no longer able to monitor the patient during exercise, we stop the test. If he develops angina with or without S-T changes, we stop the test. If he develops diagnostic S-T changes, we stop the test. If a number of arrhythmias develop, we stop the test. Almost as important as the arrhythmias is that of the patient or signs that indicate low cardiac output, failure to generate an appropriate systolic blood pressure response, particularly if the systolic pressure should drop despite exercise, if he gets cold and clammy or begins to get dizzy. Any of these things are indications to stop the test short of the target heart rate.

There are many indications for exercise testing. Some of these may be applicable to your individual needs and others may not, but the list gets longer every year. The original one, of course, was to find out why people were having chest pains. A second indication that is being used with increasing frequency is functional capacity for work evaluation. You know the man has coronary artery disease, can he go back to the kind of work he was doing? Can he go back to sports? What is he able to do and do safely?

Many people use exercise testing to screen asymptomatic people for ischemic heart disease. It's used some places regularly in post mycardial infarction rehabilitation, to write an exercise prescription, for example. In people that have angina pectoris, it can be used to evaluate therapy and to determine the results of surgery, for example; or for medical management, or to see if their angina is indeed getting worse. You might argue that you can do that from the clinical history, but that isn't always the case. Many people don't tell you everything that is going on, and some people tell you more than is going on. There may be very little correlation between their symptoms and the objective assessment of those symptoms.

We use it quite often for arrhythmia evaluation in people that have symptoms that suggest exercise-induced cardiac arrhythmias. It can be used to determine bypass patency if you have a patient who's had a bypass graft. There are increasing data to show that the severity of the coronary disease can be measured by using the time on treadmill, the heart rate achieved, or some factor involving those parameters. In progressive coronary disease it seems likely that data will be forth coming to prove that we can assess.

Let's discuss T-wave screening. A lot of people, with non-specific S-T abnormalities seen by a physician, present the question whether this is ischemic heart disease or not. And I think one very important use of the exercise test is to see if one can convert a non-specific S-T change to a less non-specific one, that is ischemic S-T shift. On evaluation of therapy--you have someone whom you are treating; you want to know if he is getting better or worse? The list of congenital heart diseases is getting longer. Severity of congenital stenosis can be determined by an exercise test. Recently some authors of papers have used exercise testing to assess the postoperative patient with coarctation of the aorta. So there is a whole list of things that you can use exercise tests for, besides determining the nature of the patient's chest pain.

There are a number of things I use as absolute contraindications to an exercise test. Obviously you would not want to exercise someone with a recent myocardial infarction or someone who has unstable angina. Any sort of rapidly progressive angina would be a contraindication to exercise. Indeed, these are people that you generally put to bed. Other contraindicators are severe aortic stenosis, severe hypertension, recent heart failure, an active or current pulmonary or systemic disease process, uncontrolled arrhythmia, or refusal to sign the consent form after we have very carefully explained the rewards and risks to the patient. If he doesn't want to sign the consent, we must have some hesitancy about the test.

Relative contraindications include recent bed rest which can distort the outcome; hypokalemia, which can cause not only false positive responses but also arrhythmias during exercise. Certain orthopedic disturbances will prevent some people from exercising, but you can find other ways of exercising them as I'll illustrate. This is also true for central nervous system disorders. Cardiac hypertrophy and digitalis can both cause false positive responses, and they are relative contraindications for the diagnostic exercise tests, although one can certainly continue to do an exercise test safely if the question is other than diagnosis. If the patient demonstrates some doubt about wanting to have the test, this is usually an indication to pause and discuss it with him further.

For those of you who have never seen an exercise test or only the two-step test, what I am going to do is show you a movie of an actual graded, treadmill exercise test. There are many ways of exercising people, and I'll try to illustrate some of these for you. The exercise tests that I am going to illustrate were done in the cardiac physiology laboratory of Weiss Memorial Hospital, Chicago.

The patient is instructed in what's going to take place and then signs the consent. We pre-attach all of the electrodes for a full 12 lead electrocardiogram, and so that they do not fall off during the test, we remove body hair. We then mark the electrode sites with permanent ink with a felt tipped pen using Mason's formal lead modification so the limb leads go on the trunk. We then use a dental burr attached to a rapidly spinning motor using a motortool to just take off the ink. We're not drilling down to dermis, we're just taking off the carotene layer and reducing skin resistance. I think you can see this little white spot right here in the slide. All we did was just take off the ink there, and that is why we mark the skin so that we can see the ink.

We use fluid-column electrodes which are then jacked into a noiseless, shielded table which hangs around the patient's head so it isn't dragging on the floor. Then we wrap the chest; this kind of wrap is very easy to remove, and it keeps the cable from swinging, which would produce some baseline wander. The cable arcs up over the ceiling down to a triple channel ECG machine, and a lead goes from the ECG machine to a monitor scope. Here is our crash cart with the fibrillator and so forth.

Our triple channel machine has a computer terminal so that when a computer is available, we'll be able to use it; it's available in selected places. Now, we voice in on channel 4 the blood pressure, symptoms, heart rate, and here's a control 12 lead ECG. This is done in an automatic way with this triple channel machine. I ask you to notice that the base line doesn't wander which is a result of the very meticulous skin preparation. We do supine, sitting, and standing control ECG's so we know what the behavior of the patient's S-T-T waves are with posture. They can change rather dramatically as you know.

Then the nurse demonstrates how to walk on the treadmill, and how we'll be taking the blood pressures while the patient looks on. We try to make it an entertaining session for him.

The patient is examined briefly by a directed cardiovascular examination to make sure that none of the contraindications to exercise are present and to establish his baseline auscultation. Blood pressures are obtained sitting and standing, and then the treadmill is started.

We use the Bruce stages, starting at 1.7 miles per hour and 10 percent elevation. We record a strip of ECG every 60 seconds and a 12 lead ECG whenever we choose. We aren't very rigid about that. We can watch the leads on the oscilloscope, and we monitor AV, FV2, and V5 during exercise giving us an approximate orthogonal lead system similar to the XYZ axis.

Someone always holds on to the controls so that if the patient stumbles or slips, we can stop the machine, and he doesn't keep going round and round on the belt. Every 3 minutes the speed and grade of the treadmill are increased, our object being to get the patient's heart rate up to his target rate and to maintain it for 2 minutes, or else to reach some of the other targets that we have already indicated.

He gets pretty tired toward the end, and you'll see his smile when he finally sits down in the chair which we placed on the treadmill so that we don't lose any time getting the post-exercise electrocardiogram. We have an interval timer which stops right at the time the test was terminated so that that's locked in. We listen for gallops, murmurs, and simultaneously record ECG, and get blood pressures. The whole thing is on tape so that we can play it back. We record the ECG's on tape, and when we play them back we can do so at any paper speed. Fifty millimeter per second paper speed helps to amplify the S-T segment, AV, FV2, and V5.

Here's the first post-exercise ECG: 1, 2, 3, rlf V1, 2, 3 and then left chest. This is at 50 millimeters per second paper speed. Here is the calibration. We do two or three ECG's in the first minute post-exercise; we recorded the end of exercise also, because very often tests will be positive only at the end of exercise and never again. I'm going to show you some lengthy examples of the ECG to give the feeling for how straight the baseline is, and I'll show you why it is important to have a straight baseline. Here is one of the treadmill - AV, FV2, and V5 at 25 millimeter per second paper speed. You notice the S-T segment is a little more difficult to evaluate when the paper speed is slower. (slide presented) Here's the first post exercise ECG at the standard paper speed of 25 millimeters per second. (slide presented) Again, I think that it is a little more difficult to evaluate and eyeball at those S-T segments at the standard speed than at the fast paper speed. Using this particular protocol, there is relatively little tremor artifact no matter what position the patient is in. And, the baseline, I think, is much easier to interpret than when you have wandering.

Let me just take a couple more minutes to show you some examples of kinds of responses to exercise tests. If you take the standard PORST you will notice that the j point is at the baseline; for exercise testing we use the PR segments as the baseline. In one kind of response to exercise; a repolarization wave of the atrial activity, the TA wave which forms the base of the QRS, artifically lowers the j point. It can be misinterpreted as a positive exercise test. This is really one variant of normal. Another response is j point depression but with upsloping S-T segment. As long as that S-T segment is rapidly upsloping, we interpret this as a normal response. We might get into some discussion about that.

Another kind of response is with the j point at the baseline but with the S-T segment sloping down, and this also is not called a positive response. The positive response is segmental

horizontal or down sloping S-T segment depression, lasting at least 8/100th of a second and beginning with a j point that is at least 1 millimeter down. Now there are other criteria for positive response, each having its own sensitivity and specificity. The sensitivity here is about 85 percent and the specificity is about 95 percent. In other words, 85 out of 100 people with significant coronary disease (the find is 50 percent narrowing of one or more major coronary arteries) will have a positive response 85 out of 100 times. You will find such a response in only 5 out of 100 normal people or so-called normal people.

The T wave inversion without S-T segment shift is a non-specific response which is neither normal nor abnormal.

So to reiterate, the criterion we've been using is one millimeter of segmental S-T elevation or depression lasting 8/100th of a second. Here for example, is how we might measure this. We form a baseline, a dotted line, and then we measure the depression of the j point. (slide presented) Here it is about 3 millimeters, and note that the S-T segment is horizontal and and does not return to the baseline rapidly. That's much more difficult to measure if you've got a wandering baseline, and you'll see many exercise tests that have this kind of a baseline. A computer could interpret it, but I don't think that the eyeball can do so with much accuracy. In fact, some computer programs have been developed, because of inability to get a straight baseline. The computer just cancels out the positive and negative artifacts and gets one complex.

If you are measuring just one lead, you have got just one lead to look at but if you bracket it or measure adjacent leads, you get even more information, I think. It wouldn't affect some interpretation at all but other interpretations can be affected by having additional leads as I'll show you.

This was a 34- or 35-year-old man who had a black belt in karate. (slide presented) He began to have discomfort in his chest during exercise, particularly in bar-room brawls, and as you can see if he wasn't in danger from his opponents, he was in danger from his own coronary arteries. This is a 50 millimeter per second paper speed after only 3 minutes on the treadmill through stage 1.

This is much less exercise than this man would have had in a two-step test. In an unmonitored two-step test he could have this much S-T depression without pain, and I think that would be dangerous. In my personal opinion, some people would continue to exercise him despite this kind of S-T segment shift, waiting for angina. This was his coronary arteriogram with a main left lesion at this point of approximately 70 to 80 percent. (slide presented) This was a man whose chief complaint was dyspnea on exertion, and it was thought by most that he had pulmonary disease. My boss didn't think so and asked us to do a treadmill exercise test on him. He had a positive response of 6 minutes on the treadmill and coincident with it began to complain of the identical symptoms that had brought him to the physician, which he said was, "dyspnea right in here, doctor," and he pressed his hand against his chest.

CARDIOVASCULAR DEMONSTRATION WORK EVALUATION STRESS TESTING

EXERCISE TESTING IN PREVENTIVE MEDICINE
Jean Spencer Felton, M.D.

The Occupational Health Service (OHS) in the Department of Personnel of the County of Los Angeles, was established to provide a preventive medical activity whose objective is the conservation of human resources through the evaluation, protection, promotion, and restoration of the health of County employees. Currently, the County of Los Angeles employs 81,500 persons in a broad variety of departments, commissions, museums, districts, and courts. The facilities are are scattered throughout a 4,000-square-mile area, and the services provided the citizens range from law enforcement to health services to recreation to flood control to adoptions. The OHS is located in four buildings in or near the Civic Center, and preventive health field services are provided by teams from the central units.

The Cardiopulmonary Laboratory, which is in the County government headquarters building, was created in response to a Board of Supervisors motion requiring the development of a program to reduce the prevalence and severity of both cardiac disorders and back injuries among safety personnel with primary emphasis to be given to employees in law enforcement and fire suppression.

THE PREPARATORY EXAMINATION

Although the Laboratory is used for different groups of workers applicants for safety positions (sheriff and marshal personnel, firefighters, beach lifeguards, and district attorney investigators) safety employees, and executives, the procedures included are essentially the same. (see reference 1). The initial examination consists of the following elements:
    Completion of a medical history questionnaire.
    Visual acuity determination through use of a precision
      stereoscopic instrument.
    Audiometry based on the ISO standard.
    Measurement of height and weight.
    X-rays of chest and abdomen, the latter to detect aortic atherosclerosis.
    Resting 12-lead electrocardiogram.
    Urinalysis.
    Hematologic survey.
    Sickle cell trait test on all black personnel, and subsequent disease tests
    when indicated.
    Urinalytic drug screen.
    Blood chemistry: serum triglyceride, serum cholesterol, serum creatinine, glucose

alkaline phosphatase, total protein, serum albumin, serum globulin, albumin-globulin ration, thyroxine (T4), serum glutamic pyruvic transaminase, and uric acid.
Examination by physician.
Proctosigmoidoscopy on executives aged 45 and over.

On return of the results of the laboratory tests, if no ischemia had been detected electrocardiographically, the applicant or employee will be scheduled for the second phase of the study.

CARDIOPULMONARY LABORATORY PROTOCOL

The Cardiopulmonary Laboratory Protocol has been described in several articles (see reference 1). In the second phase of the study, the 12-lead resting electrocardiogram is repeated to determine if any changes have taken place. If the examinee is still free from evidence of myocardial ischemia, he will undergo some additional test measurements. The height and weight mensuration is redone, and using the Lange skinfold calipers, thickness determinations are made at four areas (subscapular, over triceps, over biceps, and suprailiac) and the percentage of body fat is calculated. Spinal mobility is measured by quantifying the range of lateral and forward trunk flexion, trunk rotation, and trunk extension. The devices used were designed by Laboratory personnel.

The grip strength is obtained bilaterally using a handgrip dynamometer, and the side of dominance is noted. The strength of the hip extensor muscles of the back is checked, and measurements are made of the strength of the thigh musculature, using a laboratory-designed leg and hip dynamometer. Strength measurements are taken after dynamic electrocardiography.

Blood pressure levels are recorded before and after exercise, with the subject recumbent, on quick-standing, and after 1 minute of standing. Pulmonary function is tested with the Collins' Stead-Wells spirometer to determine the forced vital capacity and the maximum breathing capacity. Just prior to ergometry, the heart and lungs are examined.

Twenty minutes of graded exercise electrocardiography is accomplished by means of a stationary bicycle ergometer. During the test, the work is increased to produce a heart rate response of 100 within 5 minutes, 120 at 10 minutes, 140 at 15 minutes, and 160 at 20 minutes (85 percent of maximum), followed by a rest period of 6 minutes. The ECG, blood pressure, oxygen consumption, and carbon dioxide production are monitored and recorded throughout the test.

The physical facility of the Laboratory for exercise testing consists of two separate rooms with a bicycle ergometer and resuscitation equipment in each room. A third room holds the central processor and the gas analyzer. A display and control console in each testing room consists of an ECG strip-chart recorder, a long persistence ECG monitoring scope with a second channel for holding patterns of interest, a magnetic tape recorder with 30-second delay for ECG playback, a pneumotachometer and integrator, and a CRT-keyboard terminal to the computer.

On completion of the test, the control terminal is used to edit the test data before storage and listing on five record pages from a printer. Pre-set standards for blood pressure level, frequency of premature ventricular beats, and S-T segment changes will effect an early termination of the test, when exceeded.

TESTS ACCOMPLISHED

The exercise test protocol was initiated November 1970. Through July 27, 1974, the following numbers of persons have been examined:

Examinees	No.	Percentage of Total
Applicants------------2,571	42
Employees------------3,582	58
Total-----------6,153	-

During that period, abnormal findings--distrubances in rhythm, or in conduction, or evidence of myocardial ischemia--were encountered as follows:

Examinees with Abnormal ECG's	No.	Percentage of Total		
		Examinees	Applicant Examinees	Employee Examinees
Applicants-----------70	1	3	--
Employees-----------307	5	--	9
Total------377	6	-	-

Fifty-four of the applicants tested, 1 percent of the total examinees seen, and 2 percent of the total number of applicants tested, were not recommended for employment. (It should be noted that a small additional number of safety position applicants were disqualified on the basis of findings in the resting ECG's and did not proceed to exercise testing.)

Of the safety employees tested, 67 or 1 percent of the total examinees and 2 percent of the total employees, were recommended for removal from or change of duty because of significant electrocardiographic findings.

BASIC OPERATIONAL CONCEPT

Although the measurement of the cardiovascular response to the challenge of work has been part of clinical medicine for nearly half a century, it has only been in recent years that there has been a loosening of faith in the diagnostic value of ECG's taken in the resting state. Both physicians and patients have placed confidence in tracings interpreted as "normal," in spite of the growing body of knowledge that such a procedure may be only the most superficial of screening techniques. Many observers have reported positive findings on dynamic electrocardiography conducted on persons declared to have normal resting ECG's.

This platform has been the philosophic base for our cardiopulmonary laboratory operation, and parallels the excellent statement of the American Heart Association's Committee on Exercise, (see reference 2) when reference is made to risk factors. "Persons with risk factors but without manifest heart disease are, nonetheless, coronary-prone. These individuals should undergo exercise stress testing for detection of possible asymptomatic, early manifestations of ischemic heart disease. Persons with identifiable heart disease should not engage in unsupervised, strenuous physical exertion without first submitting to exercise testing."

Further, and possibly more importantly, the Committee has stated (see reference 2) that, "Exercise stress testing may reveal the presence of latent ischemic heart disease in persons without increased risk factors, although testing in such persons under the age of 35 is not likely to disclose more than one case in a hundred. However, as age progresses, ischemic responses to exercise testing may be expected with increasing frequency, as high as 10 percent in some series."

In substance, it has been our belief that, in the employment situation where there are workers exposed to significant physical and emotional risk, exercise testing is mandatory to identify, by the best non-invasive technique available, those persons (applicants or employees) with covert coronary artery disease, so as to avert the major devastation that such pathologic changes may produce.

OBJECTIVES

The Committee formulated succinctly the objectives of exercise testing (see reference 3).

1.) To establish a diagnosis of overt or latent heart disease.
2.) To evaluate cardiovascular functional capacity, particularly as a means of clearing individuals for strenuous work or exercise programs.
3.) To evaluate response to conditioning and/or preventive programs.
4.) To increase individual motivation for entering and adhering to exercise programs.

USES OF EXERCISE TESTING BY OHS

With the establishment of the Laboratory, the OHS has applied the findings generated by exercise electrocardiography to the employment process, to the evaluation of workers in service, to the initiation of exercise programs by individuals and by groups, and infrequently to the return-to-work review of cardiac patients.

IN THE EMPLOYMENT PROCESS

All applicants for safety positions receive the complete two-phase physical examination for several reasons. First, both the training period and the work are arduous, very physically and emotionally demanding, and the applicants must be able to meet the requirements of these jobs. No differentiation is made in the test protocol between men and women applicants for deputy sheriff positions. As yet, no women have applied for a firefighter position.

Second, under California law, anyone engaged in law enforcement of fire suppression who manifests heart disease comes automatically under the so-called presumptive clause, which means that the heart disease is presumed to be job-related. Thus it is covered by workmen's compensation. The current compensation costs for a safety worker with heart disease are estimated to be $40,000, and if surgery is involved, this figure rises to $50,000. It may be seen, therefore, that the 54 applicants disqualified for employment, if not detected, may have cost the County of Los Angeles at some time during their service approximately $2,160,000.

Third, the findings on testing may be used to aid in the placement of the candidate in other available, less-demanding positions. Although applicants for deputy sheriff or firefighter posts rarely wish other work, the mechanism does exist for selective placement in other departments.

IN EMPLOYEE EVALUATION

Safety personnel are re-examined at intervals to determine their cardiovascular status and receive the complete examination outlined previously. If the examinee is a member of the Sheriff's Department and a limiting cardiac disorder has been encountered, an assignment can usually be found for him consonant with his capacities. Because of the multiple activities of the department, there are many positions extraordinarily different in their physical demands.

Comparable possibilities for personnel mobility are not the same in the County's Fire Department. It has been decreed by the Chief that "all men must be able to pull hose." In specific support of this requirement, there are spelled out in the form used in returning a firefighter to work after an illness absence the tasks which he is expected to perform. They are:

1.) Lift weights of 50 pounds or more repeatedly.
2.) Go long hours without sleep (24 hours or more).
3.) Pull hose lines up steep mountainous terrain (at altitudes of 5,000 feet or more).
4.) Drive steep and narrow roads in mountainous terrain as well as through heavy city traffic under emergency conditions (red light and siren).
5.) Be exposed to toxic fumes and heated gases.
6.) Be exposed to overheating, chilling, and wet clothing.
7.) Be aroused out of sound sleep by fire alarm gongs.

If the returnee cannot function as required and the condition is not reversible (as it most usually is in coronary artery disease), the incumbent if offered other employment in the County or goes on to a disability retirement. Rare is the fireman who selects other work; he chooses instead, to take a year of sick leave at full pay, and then retire. In essence, the exercise testing is used to determine the continuing fitness of an individual to retain his physically rigorous job.

Those men who present normal ECG's in response to an exercise challenge continue at work, knowing that their health status still meets the needs. Many of this group, of course, are involved in individual or group exercise programs, and the testing, if the results are good, offers testimony to the worth of the activity program.

A smaller group of employees undergoes the same protocol, at their own choosing. The program is offered to executives, and their acceptance has been particularly good. Noteworthy in their use of the examination system have been department heads and judges. The usual yield of lesions in older groups have been encountered, and a moderate number have been diagnosed as having myocardial ischemia. The additional step offered with these staff members is an individual review of findings with the patient by the Medical Director, and referral, with complete report, to his private physician. Further, in cases of hyperlipidemia or overweight, the consultative assistance of the OHS nutritionist is sought.

Extremely infrequently, another use may be made of the exercise test, and that is as an adjunct procedure in the battery of steps employed in a medical re-evaluation or what is generally termed a fitness examination. If a questioned job performance or attendance record can be given greater understanding by an exercise test, such a set of diagnostic steps will be included.

IN EXERCISE OR CONDITIONING PROGRAMS

The Cardiopulmonary Laboratory has exercise equipment in the form of stationary bicycles and wall apparatus, as does an outlying facility for use of executives. Before anyone is permitted to use these facilities, he must have completed the full examination. If his cardiovascular system shows no deficits, one of the exercise physiologists will outline the correct program of reconditioning. If certain ECG changes are uncovered, the private physician will be apprised of them, and if the patient wishes to exercise, a written and signed exercise program will be requested for the individual.

Some departments have obtained their own exercise equipment or have established a compulsory exercise program. In such instances, it has been mandatory that each person wishing to participate must be cleared by the OHS by means of the complete examination.

FOR MISCELLANEOUS USES

On rare occasions an employee's physician will request on his patient's return to work after a cardiac episode that an exercise test be performed on his patient, or that one be done as the

basis for an exercise prescription.

Most encouraging is the repeated use of the ergometer by the truly ardent exerciser as a check device of his progress. When his work load increases with the programmed heart rate increments over his previous record, there is solid evidence of the worth of the undertaking.

Infrequently exercise testing will be conducted in response to a filing of an appeal by a medically disqualified applicant for a safety position. If, for example, the resting electrocardiogram proved to be positive, it might be that an exercise test will be carried out under close cardiologic monitoring to confirm the presence of coronary artery disease. In public employment, the appeal system permits an applicant to appeal any decision regarding his physical or emotional capacities for a position. Unusually low levels in the measurements of strength and mobility have not been used to disqualify an applicant. These data are being collected so that one day a correlation might be made between the strength and mobility data and the occurrence of back or other injuries.

SUMMARY

1.) An Occupational Health Service serving an extremely large County government provides exercise testing as a portion of the examination protocol of safety personnel and executives, both applicants and employees.
2.) Carried out in a special Cardiopulmonary Laboratory, 20 minutes of graded exercise electrocardiography is accomplished by means of a stationary bicycle ergometer to 85 percent of maximum.
3.) In over 3 1/2 years, 6,153 persons have been examined, 42 percent being applicants and the remainder employees.
4.) Of the total, 377 examinees displayed abnormal electrocardiograms.
5.) It is believed that in the employment situation with workers exposed to significant physical and emotional risk, exercise testing is mandatory to identify those persons with overt coronary artery disease so as to avert the major devastation produced by such pathologic change.
6.) Exercise testing is used primarily (a) as part of the employment process to identify latent heart disease in applicants; (b) to serve as a portion of the re-examination procedure of employees in demanding positions; and (c) as a requirement prior to utilization of exercise facilities provided by the County of Los Angeles. Infrequently the procedure will be used on an employee's return to work after a cardiac episode as a re-check of persons on exercise programs or as part of a review occasioned by a disqualified applicant's appeal.

CONCLUSIONS

1.) Exercise testing should be a mandatory inclusion in the preplacement physical examination and periodic examination of all persons engaged in extremely physically or emotionally demanding positions.
2.) The procedure can be life-saving to the applicants and employees, and cost-saving to the organization.
3.) The data generated by such test procedures can aid in future research in coronary artery disease.

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CARDIOVASCULAR DEMONSTRATION WORK EVALUATION STRESS TESTING

EXERCISE THERAPY FOR ANGINA
John E. Smith, M.D.

In the airlines we have a peculiar problem because we deal with an unusual group of people--airline pilots. This group make over $40,000 a year. When we use exercise therapy to attempt rehabilitation of a pilot grounded for a suspected heart condition, we find he can be easily motivated to cooperate. If a man might drop his pay from $60,000 to $12,000 on the basis of whether I can make his ECG normal or not, I have no problem getting him to comply with an exercise and physical fitness program. Perhaps this also explains why we get much better results with exercise as a form of therapy than most groups reported in the medical literature.

I came to work for United in 1962. I worked out a company program using exercise stress testing for the treatment of angina, based partly on my own problem. I developed angina in 1961, and I had a 3 millimeter RST segment depression; I could walk about two blocks. I found the conventional treatment of angina by good doctors was inadequate, at least as far as I was concerned. Everything my own best medical friends would do would depress me. After about 6 months, I consulted an old professor of mine up at the University of Vermont, who felt that angina was a little bit of a hoax. He thought a lot of it was catechol effects. My wife kept saying, "You've had a nervous breakdown; you haven't got heart trouble!" I had been with the Federal Aviation Administration just before that.

Anyway, I started my own program of exercise. I measured a mile from my home up a slight grade. I walked that mile four times a day. I had nothing else to do at that time, because I was convalescent, just out of the hospital. I would walk up the hill to a certain point. Time after time for 2 months I would make it up to that point and then would have to stop on account of chest pain. I began to think, "Maybe I'm not going to make it." About 6 weeks later, one morning I went out and walked up the whole hill. The pain was gone. Then I found peculiarly enough that I would get this pain, not quite as severe, as I kept the program going, but then if I stopped, I would walk 10 miles. This is now described by some as walk-through angina. I think this is one of the most important things in the treatment of angina, because I believe that angina is as much upstairs as it is downstairs as far as the heart is concerned.

I've treated now about 40 cases of angina among airline pilots. Once they have angina, they don't fly. But they think they're going to fly. We try to maintain the principle that they come back to work in some capacity if we can get rid of the angina. The first group I published was of 21 patients. Four of them didn't respond to treatment. For some reason there are always a few people who don't respond. I don't think it has anything to do with their heart trouble; it's a mental condition. I think sometimes it is their wives. Sometimes it is the changing of living habits that these people desire; they don't want to get rid of the angina, because they could perhaps retire and go someplace else to live. But the ratio (about 4 out of 21) was the same for

the second group. What I have done with 40 angina cases is do for the group the same thing I did for myself. I have had no chest pain now since 1962. I used for the group that same technique of a gradually progressive exercise stress. I try to get these people to walk 4 to 6 miles a day, starting at a 20 minute mile up a little bit of a grade and then gradually increasing the pace to about a 15 minute mile. This varies a little with the structure of a person's legs--how short or how long legged he is. We've had some rather remarkable results.

I'm convinced that airline pilots also have another variant that you people may not see, what I call reactivity, cardiovascular reactivity. The good literature on this was written by Hitler's doctors about 1937. It never crept into the American literature much. Their flight surgeons noted that some people would react to stress peculiarly by increasing their flow rates. And these were called ergotropes. Their hearts would pound, they developed mild hypertension as a situational stress. Using an ultra-low frequency ballistrocardiograph, these people commonly react to situational stresses by increasing their ejection velocity. Yet, they maintain a normal pulse rate and may maintain a normal blood pressure.

Now in this business of cardiovascular reactivity the German doctors noted two types of reactions to stress. One they called an ergotrope for lack of a better name, and the reason they did this is because these people, aviation cadets who wanted to be pilots, were responding to the sympathomimetic drugs of the ergot type. There is another reaction to stress which they found which they called histotrope, and this is the one actually whose cardiac output and stroke volume went down under stress. So one would look from an empirical point of view like a hyperthyroid, and the other would look like a hypothyroid. But these were reactions to situational stress.

Now we find among pilots many who don't detest doctors, they hate them. Yet that's not the right word either. A pilot once put it this way to me, "How would you like to have your job at stake when you have to go to a guy you got no respect for, you know he's an alcoholic and you know he's not interested?" This is not a bad way of putting it because pilot certification is a compulsory program. I can sympathize with this and it is the beginning of what I call cardiovascular reactivity. Many pilots are conditioned as cadets. A doctor says, hmm, got a little high blood pressure, that's all he say, and they are rejected. They come back a month later and may make it or they may not. This is the Pavlovian type of conditioning in which the pilot never does get over it. Every time he takes a physical examination, he's like one of Pavlov's dogs. His job is at stake; he reacts.

I think the symptoms of cardiovascular reactivity deserve much more attention. You have all seen this, and maybe you've paid more attention than I think you have. But, I see an awful lot of people with abnormal ECG's under situational stress. Their palms sweat. My electrocardiograph technologist, when I was reporting on the use of propanolol as treatment for this, maintained that she could tell the reactivity by measuring the diameter of the sweat pool under the armpits on the paper on the electrocardiograph table.

They also very often have atrial gallops. Now an atrial gallop is a very important diagnostic sign, but not in reactivity. They also have bruits over the carotid arteries and fairly loud. At the same time, these people also will slip their T waves over, and many times it will be on the resting ECG. Yet the exercise test will be either a little more normal or will remain unchanged with no change in the RST segment that has sagged down.

Now whether you get this among people that you see, I don't know. I think we tend to get an awful lot of it among pilots, because their jobs are at stake. There is another thing that goes with angina, and I've seen this often. I was on a panel with a psychiatrist, and we were talking about angina, and he said, "I see all these guys with mental depression now with chest pain, and I don't pay any attention to it." And he was right. I think an awful lot of the cases that I see are mentally depressed. They're not mentally depressed in the psychiatric sense, they can work. But, they also have problems within the family structure and the psychological problems of the middle aged male. The middle aged male in America today is a pretty poorly treated specimen in some respects.

An example: One of our Captains came in with angina. Negative resting ECG, positive exercise test. Trying to get a history out of him--no, no stresses.

His wife happened to be there. I try to get all these men to come in with their wives, because you can get a better history stress-wise from wives than you can from a pilot. So while he was having some of the lab work done, I brought her into the office. Beautiful woman. I was talking sex as a variable. I said, "When did you have sexual intercourse last?" She said, "Three months ago." You know that man must be depressed! I could look at this girl and tell he couldn't possibly be normal and have sexual intercourse 3 months ago. Then she told me why he had angina. This was his second marriage. Living with them was a son by the first marriage whom he almost worshipped, and the son was in jail for stealing. The Captain hadn't slept since that time. He wouldn't give me this history. But the wife would. Many times the wives will let me into the situational stresses, the things that are going on with these men, that I can't ever get out of a pilot. A pilot thinks he's very manly. He wouldn't want to admit anything like these stresses.

Another case: A man has a very aggressive wife; he's got two kids in college, and there are a lot of family difficulties. One of his kids gets on drugs, is arrested. The man's angina appears in two weeks. I think many times we don't pay enough attention to this type of angina. While these people do have an underlying coronary disease, I think the catechol effects from the stressful situations are the precipitating factors. I think within this group we get the best results by using progressive exercise as a treatment. I'm not certain under a progressive exercise program how much good we do to these people as far as developing collateral circulation is concerned, but I know we change their psyche tremendously. They get treated very poorly by most doctors. Practically all of my cases have been treated many times by outstanding physicians. These men are depressed. They're told not to walk upstairs, they can't do this, don't have sexual intercourse, everything is a denial, and I think it is basically wrong.

What do we do with a man who develops a bundle branch block who seems to be in excellent physical condition? We could find nothing else. Obviously, he should be catheterized and other studies done, but I just want to illustrate this case. I run two of these a year out of an airline crew of 6,600 pilots. Out of this group this is a man who was 57 in 1968. He retired at age 60. This was picked up 30 years ago! So am I a lot smarter than the guys who are using the dyes in the heart, because he went all this long? I would like to bring out on the ballistocardiograph that his is a perfectly normal ballistocardiograph at the age of 57. I think these things are good clues with the work that we have now done by angiographic staff. We now have 18 left bundles that I've picked up since I've been with United over a 13 year period. We have 14 of them flying. All these have clean angios, but I'm not particularly happy about the interpretation of the angios, because when we didn't have angios I think we were doing all right too.

This is an illustration of why I get upset when everyone tells me the RST segment depression is proportional to something. I've been in cardiology too long to believe this. In the first regional group of 21 cases that we used exercise stress as a treatment for angina, here was a man who couldn't walk up a flight of stairs. He was sent in to me and this was his first ECG. (slide presented) He had the most ST segment depression of any case, that I did and this is a Double-Two Step. As you can see, this is a good 5 millimeters, yes, more than that.

Many of these cases I walk with because I like to. Most of my cases come from out of town, and when I get through with their workup, I don't eat; I go for a 2 mile walk every day. It is good experience for me to walk with these men to really get a feeling for how much angina they have and how bad it is. This man had it so bad that I had to stop three times to get him back on the airplane. So I put him on progressive exercise stress program. This is a 6 months later ECG. (slide presented) His angina has gone completely, but he still has some little ST segment changes. Normal 1 year and look at the date on that, don't forget, that's 1968. I first saw him in 1966. This man now hasn't had a chest pain since. He's a working executive in United Airlines at the vice president level, and this man is absolutely convinced that pills are the worst thing in the world, and I believe it.

Now this is a typical case of cardiovascular reactivity. (slide presented) I see about 50 of these a year. Forty-three years of age, a top physical specimen six kids, an airline captain making $60,000. This is February 1966. I put him on exercise. I told him not to run, whatever he did. To go from $60,000 down to $12,000 with six kids, age 43, you'll be surprised what motivation you can get. This is his ECG a month later. (slide presented) These are the same things

with the exercise test. (slide presented) The man's been flying safely. He was so reactive that my technician told me he had the widest diameter sweat pool under the armpits of any man we've tested. We find in this group very often they responded to propanolol very nicely. You can give them 30 milligrams or 40 or 50 depending on how big they are and with a little psychotherapy get them to relax and they'll do very well. You can get a normal ECG on them. I was almost convinced that propanolol would be the answer to the problem of the so-called ergo-trope, to the person who's reacted to the examination with a sympathetic nervous response that gives us abnormal ECG s, except for the following case.

This one is also interesting, because it shows why I like the ballistocardiogram, though I didn't have sense enough to pay attention to it. You will notice that he has no I wave on this ballis-tocardiogram. (slide presented) This in my opinion has been done by computerized methods at Hopkins. It's done by Ikstar. We find this one of the most significant findings on the cardiac acceleration curve, because it means very much lower ejection velocity. Now here's a man that comes in with angina, positive exercise test, so I repeated him with 30 milligrams of propanolol, and the propanolol curve was almost made normal. I saw him late. I can forgive myself for this, because with this type of thing in his ballistocardiogram, I would have been much more cautious. But he is always jogging. Someone else had seen him; he was from the West Coast, and he'd been jogging, so I figured well if he's got away with it, let him keep it up. I'm not going to stop him, because the chest pain has disappeared. Three weeks later they found him dead out in the school yard with his jogging pants on. So we're dealing with something that isn't quite as insignificant as a chest cold.

I would like to say that I'm enthusiastic about exercise as a form of treatment for middle aged males, but I'm not certain how it works. I know that I can take everyone with an abnormal T wave pattern for practical purposes and make them normal. And many of their exercise tests, if they are asymptomatic, I can make them all normal with exercise, with the exception of maybe one or two. It's so infrequent that I can't that I expect them all to become normal. These are asymp-tomatic people. I can take almost all of my angina cases and get rid of chest pain if I can get them motivated well. This isn't all the airline's problems and don't forget we have a good economic bases. I hate to put things in terms of money, but if we lose an airline captain at the age of 45 and he lives 10 years, United Airlines loses $250,000.

MEDICAL MONITORING FOR OCCUPATIONAL DISEASE

A NEW PHASE FOR OCCUPATIONAL MEDICINE
Irving R. Tabershaw, M.D.

<u>Irving R. Tabershaw, M.D.:</u> To set a framework for our discussion on Medical Monitoring for Occupational Disease, I would like to review briefly major landmarks in the development of medical practices in industry and some of the current forces which make medical monitoring for occupational disease an increasingly important facet of occupational medicine. Perhaps the first thing is that occupational medicine in its practice is markedly influenced by law and social forces. This is unlike other aspects of medicine where a disease or organ system is the focus of a group of specialists. In our field, the target is a population—the worker. The legalities, the social framework, and the interpretation of the worker's health are important elements which impinge on the way we practice occupational medicine.

This is illustrated in the history of the development of this specialty. The first, and still a major force, is surgery—taking care of the individual who is injured on the job. This was given its basic initiative by the workmen's compensation laws in the 1910 to 1920 era when it became mandatory for industry to provide medical service for workers who had been injured on the job.

After World War I, a second element crept in, and it is now reflected in medical monitoring for occupational diseases. That element is the appreciation that there are subtle toxic agents in industry which do in fact affect human beings over a long period of time and that many of them also have acute responses. I'm talking about the whole field of industrial hygiene and the discovery of toxic or chemical exposures, which people like Alice Hamilton were so instrumental in bringing to attention. But since then, industrial hygiene seemed to become an engineering problem—to prevent exposures. Once you physicians described and made a diagnosis of the effect, it became somebody else's purview, namely, the industrial hygienist's, both to measure and to control. The doctor's role has not been very great.
Except for original identification, a third element crept in during World War II. I say crept in because these were subtle influences which developed the appreciation that medicine had another role in the practice of occupational medicine. Here it was a matter to some degree of law or social situations. During World War II, there was a critical need for manpower and the doctor was helpful in assisting individuals with impairments to become part of the labor force. The followup of such individuals, including the medical management at work and the job placement relationship, became important. This has been essentially what has been going on in industry.

Yesterday, for example, we devoted a whole day to cardiovascular disease in industry. With the exception of those cases that are labeled compensable, cardiovascular disease concerns the issue of the doctor in industry who keeps the man at work through health maintenance. There is no real legal or social basis for that. But, you heard expressed yesterday a philosophy that supports the idea. Why should we use industry as a basis for studying even preventive aspects of coronary disease?

We are now coming to the question of the specific occupational diseases. For those who are not familiar with the field, let's appreciate that in industry there are toxic agents, there are processes, there are situations, and there are products which can produce disease in man. It is toward these that the current emphasis on monitoring in a medical fashion is being directed.

There are laws that influenced to some degree the kind of practice that is carried on in the sense of medical monitoring. Early examples are the regulations of the FAA or the ICC which define health qualifications of airline pilots and truck drivers, in the interest of safety or the public. To that degree we develop some legal basis. Since World War II, this specialty has continued to develop in this direction with components of surgery, of medicine, and of interest in industrial hygiene.

Actually from a medical standpoint, there have been relatively few people interested in the toxic chemicals in the larger area of this specialty. But, in 1955 we became a specialty group. Here a great deal of the direction was given by the AMA which set up the basic ethic of practice that we were preventive, that our function should be practically entirely preventive medicine, and that treatment was to be diverted or sent to physicians in practice outside the industrial plant. A part of the rationale behind it, and this has validity, is that the field is so broad and the scope so great that in a sense it is a general practice. You have to know so many organ systems and so many other factors that you can't even define the specialty.

I was involved for some years in attempting to develop a curriculum that described this occupational medicine. It was very difficult for me to focus on what one should teach, and I dare say that has been one of the major problems in our recruiting younger people to enter the field. It's been difficult to define. We really have a smorgasbord. You can pick anything you want in medicine and say that it applies to an industrial population, and you can find a specialty within that particular area of interest no matter how narrow.

So from the clinical standpoint, it has been rather indefinite. Industrial hygiene in the work environment is part of it. One should know the work environment. We constantly say that. But, if you look at most practice in occupational medicine, you'll find that essentially it is directed at the impairment or the potential impairment of the indivdual. This is the preventive medicine, this is the internist who does an executive health examination with very little regard as to what the potential of the environment is, when perhaps the environment plays a minor role in such instances.

We have identified that group information is important in industrial epidemiology in order to define a health standard. That perhaps has been the single largest area of medical involvement. In a general way, when I describe the practice of occupational medicine, particularly the practice of my full-time colleagues, I say, "A good deal of their work is administrative." Administrative work is an important element in tying together all of this wide variety of information that has to be put together for the benefit of the worker, because that's the target.

In 1970, a law was passed--the Occupational Safety and Health Act--which is having a profound influence. It is obviously changing the scope, the direction, and the importance of the practice of occupational medicine.

It's interesting to me that up to this point except for the asbestos standard only one regulation has been adopted. In 1972, the Department of Labor adopted a first aid rule (Paragraph 1910.151) which was taken over from previous federal legislation. That rule says that you must provide a first aid facility or its equivalent in an industrial establishment. And that's about the extent of it. The wording says that in addition to certain first aid requirements..."the

employer shall insure the ready availability of medical personnel for advice and consultation on matters of plant health." Matters of plant health being so broad, for all intents and purposes it doesn't tell what you should do or not do.

As part of this law, the Occupational Safety and Health Act (OSHA) was created; its function is to create and enforce standards--standards for the workplace. You all know that during the first 3 years or more OSHA still emphasizes safety and has handled health very gingerly. The National Institute for Occupational Safety and Health, (NIOSH), was created by the law to provide the scientific and professional base for the standards to be promulgated by the Labor Department. It is the NIOSH criteria documents and the OSHA standards I will concentrate on to set the basis for medical control.

To make a general statement about policy, it seems to me that the policy of OSHA has been to worry about the health of the workplace, not the health of the worker. There is a truism in all this that says if the workplace is healthy, there are no noxious agents; obviously, the people working there will also be healthy, and this is about as far as they want it to go. But our concern is with the health of the worker, the health of the individual.

Now what is the reason for this emphasis on the workplace instead of on the worker? I believe part of the reason is that there were threshold limit values available. These were guidelines that the profession clearly said are engineering guidelines and not by any means standards that we can live by or that we can enforce. Engineering description is easier than a medical description. You could have monitoring by relatively untrained people. And, the data that you develop could stand up in a court. So it's easier to put the emphasis on workplace controls, than to control the worker himself. And, if you try to control him, you find a great many problems.

Perhaps one of the major forces that deters examining people is the laborer himself who says we should make the workplace safe and not use the worker as a guinea pig. The other problems were such things as the following: What are the qualifications of the doctor to do the examination? Who's available; what do they know; where are the laboratories? Also it's more costly to deal with physicians than with engineers, and ultimately you get to the fundamental question of ethics. How do you handle the records? What is the responsibility to the individual? As physicians we do have that kind of responsibility, and it is important to him and to us. I mention this, because we still at this point have very few standards that involve examining the worker. But the evidence is accumulating that we must move toward examining the worker, and we must be responsive in that effort to all the questions that I've raised.

Now, since 1970 OSHA and NIOSH have developed 20 criteria documents or equivalents which include the best thinking at this time about what should be involved in medical monitoring for certain substances. We have 17 criteria documents, each a publication of NIOSH which proposes the criteria to be examined in establishing the recommended standard. We have one standard adopted, the one on asbestos; and two proposed emergency standards are still being debated one on carcinogens and the other on vinyl chloride. This makes a total of 20 documents that might be used as a basis for defining the medical role in the developing field of occupational safety and health.

A criteria document gets to be a fairly substantial monograph; it has a lot of information in it. I am impressed with the quality and the amount of reduction of information that's been accomplished. We as part of our consulting activities did assist in some of them; that is, in finding the basic information. I can tell you it is an overwhelming job with a great deal of attention to professional and scientific detail. The criteria documents cover many aspects, some in great depth, of the clinical responses, the diagnosis, the epidemiology, the toxicology, personal protective devices, and other matters such as analytical methods to be used, the frequency and type of environmental analysis that's indicated, and the scope and use of the agent. There is a little bit on treatment, and some words on prevention. But, the bulk of the document is still a medical one. It involves the health effects on man. And, we now begin to have in these 20 documents a body of knowledge that's been reduced to manageable form. It has been seriously thought through and is no longer a matter of a single opinion. At least there is now a text book, if you wish. We've never had a text book in occupational medicine in such detail.

I've already indicated the need for medical background, and the reason there hasn't been previous medical emphasis. Now you can't avoid it. Obviously, emergencies take place; there are acute exposures; you have to have some medical background for those. There are a number of diseases which represent a long latent period, that you must constantly monitor.

There are a number of situations in which the medical monitoring has no immediate value but has epidemiologic value in that the disease is subtle, takes a long time to develop, and at some point you must recognize whether or not the threshold limit or the standard you set is applicable. We've had some potent examples of failures of this kind. In some instances, it is even the sole method of really telling what goes on in the worker, because what happens to the man is really an integration of the total exposure by skin, by inhalation, by ingestion occasionally or frequently.

Now my point is what these 20 documents at this point represent. Remember there are at least 20 more in the works and perhaps 40 in the next year or two. There will be 386 additional mini-documents, which I think Doctor Bundy will react to. In the next couple of years, we are going to have a flood of information on specific toxic chemicals, and if you look at industry broadly, any doctor involved with industry must sooner or later meet one or more or perhaps dozens of them. If he is in the chemical industry, he obviously will meet more than one. On the other hand, practically every job has a noisy area or has a hot environment somewhere. If you practice occupational medicine, you cannot neglect this text book that is developing.

Furthermore, it is now expected by industry that physicians will know what's going on, and your own managements will ask you what you know about this. Perhaps more important, labor will ask you because part of the OSHA law requires that you must educate the individual regarding his potential exposure.

I think we can expect it intra-professionally also. We'll ask one another, what do you know and how are you handling your particular plant? And we'll expect a reasonable response indicating that the man has at least read the basic literature. There is still another factor here, one not to be overlooked. There is beginning to be a real liability not just financial but also in terms of the conduct of the plant. A strike, imminent shutdown, and so forth are possibilities resulting from this law. Thus, the doctor's opinion is not merely a private one between him and the worker or between him and his employer. What he is saying can become a focal point. I'm sure we can give you examples of the repercussions of a careless remark or an ignorant remark or even of an insufficient response because of lack of knowledge.

In a general way, there are four areas covered in these 20 documents. Of the 20, 19 demand that you take a history without specifying what you are to ask for. And I'll go into those a little bit more in a moment. Nineteen demand a physical examination; the one that doesn't is the one on noise.

Now I'm referring to directions in these documents that demand procedures you should be doing if you are to be responsive to the control, the diagnosis and the management of that particular toxic chemical or hazard. Bear in mind that even if these are not yet adopted as standards, they do represent the best thinking of what we now know in occupational medicine regarding any one of these specific toxicants. Even if the eventual standard doesn't fit specifically what is suggested here, nevertheless the basic medical information and environmental information is incorporated into these criteria documents. So there is no question in my mind that this will become what one can reasonably ask an occupational physician to know about noise or about trichlorethylene or about the heat environment.

Eleven of the 20 documents have specific laboratory demands, indicating what you must do, and 12 demand a special examination of some type. Some of the latter concern evaluation of the worker for potential need of a respiratory protective device.

Briefly, the 20 documents say the following about the history: Five specifically say you should have a personal history. Two want you to take a family history, (I don't know how important that is in occupational medicine.) Two want environmental factors, whatever that means; (I suppose where the worker lives and whatever other extraneous influences might act in consort with or

inhibit the occupational toxic agent or exposure.) Eleven ask for an occupational history; two want you to look at genetic factors, whatever they are; five specifically demand that you go into a smoking history; two, that you go into the alcohol intake history; four, into respiratory symptoms. Then you are asked in at least one document to look at reduced immunological competence, exposure to steroids, exposure to cytotoxic agent, hepatotoxic agents and so forth. One asks that you inquire about pregnancies; one wants to know about blood transfusions as part of your history; and four specifically demand neurological symptoms.

In physical examinations, the spectrum that you are supposed to cover includes a general examination in four; five ask for eyes, (and, I think they mean the external aspects of the eyes, the conjuctivae, not the phorias and so forth.) Eight want you to look at the skin; ten, at the respiratory tract; one, at the abdomen; one, at the endocrine system; two, at the kidney; three, at the liver; two require that you do a dental examination; and two that you weigh the patient. I'm just putting together all the specifics that you are going to have to put into your system if you've covered all these 20.

As for laboratory examinations, four specifically ask for variants in the peripheral blood; one, a liver profile. (The liver profile is an interesting one because that's the vinyl chloride standard; it calls for some exotic tests, not demonstrated to be effective in telling the story.) Five demand pulmonary function; six, a chest X-ray, and four, a urinalysis.

The specific examinations demanded depends, of course, on the particular toxin; one asks for urinary phenol (benzene); the lead document demands a blood and urine lead; the coke oven emission, asks for a sputum cytology; in noise they want an audiogram; and the bulk of the special examinations are to determine whether or not an individual can wear a respirator.

So let me just review now with you what this broad spectrum of criteria documents and standards means and what the future looks like. First, it is a body of organized knowledge, a knowledge we've never had before. We can agree that some of the best minds in the country have assembled what we do know at this time, and what is fact separated from undocumented experience. Second, we have definitions for a reasonable level of practice in medical monitoring controls--at least you do what's asked for these specific toxins. Third, there are legal and economic implications of your decisions--it is no longer a matter of your just practicing medicine full- or part-time with your employer or with your worker group. Fourth, you must now begin to distinguish this from some other aspects of clinical practice; for example, the easy way to manage the liver profile is to run a SMA12 but that way you get a plethora of information that you need a computer to sort out for you. We must find some device or devices in occupational practice that focus on special problems, in this case the liver profile.

Another aspect of this practice is that you now have a goad; you must contend with the worker's knowledge. He's entitled to the information; he'll be getting it; he'll ask the question. It's like Today's Health in your waiting room which tells the worker what to expect in the way of new treatments. He will anticipate this. And another aspect, you must keep a record. You have to be involved in recordkeeping, sometimes for as long as 20 years; it is now required by law. Obviously, the record is not only just what you put down but also what else is pertinent to the worker's health--environmental data and so forth. So, we're in a new phase of practice; no longer are we simply involved in practicing occupational medicine as we ourselves saw it or as we determined it as a specialty.

MEDICAL MONITORING FOR OCCUPATIONAL DISEASE

HOW CAN MONITORING BE ACCOMPLISHED?
Rita Dingman, R.N., M.P.H.

Today, many organizations have received instructions to do monitoring. But there appear to be a few questions to be answered. WHEN is monitoring necessary; WHO is to be monitored; WHAT is to be monitored; and HOW can monitoring be accomplished?

I will assume that my physician colleagues on this panel will discuss WHEN monitoring is necessary and WHO should be monitored. A few of the criteria documents have stipulated that these are decisions which should be made by physicians. They quite likely will also cover WHAT is to be monitored or WHAT parameters are included in the monitoring. Doctor Tabershaw already discussed some of these considerations.

At first glance, "Medical Monitoring" would seem to imply "Physician Monitoring." But, as one reviews the required and recommended standards to date, there are relatively few tasks directly assigned to "physicians" per se. More often the references are to "medical personnel" or a "competent person." Quite likely this was done deliberately, so there would be as much latitude as possible and still get the job done.

And, the job is massive. In Colorado, which is not an industrial state but which is typical of many states; we have less than 100 organizations with more than 500 workers each. Some of them are actually umbrella organizations for a number of small units. We do have 82 organizations with full- or part-time occupational health physicians and nurses. However, we have over 44,000 workplaces with less than 250 workers each; probably 42,000 of them have less than 50 workers each. I'll talk about Colorado, because I am familiar with it.

The Federal standards, so far, affect all establishments engaged in interstate commerce. They are not limited to those large industries with full-time qualified occupational health personnel.

Most of the established occupational health service units have been doing some medical monitoring for years. But some haven't. Especially the one-nurse unit with an on-call physician. In my frame of reference, these are the medium-sized industries--from 300 to 700 employees. Many of these and even smaller organizations have indicated on various occasions that they are frustrated in their attempts to contract with a nearby general practitioner even for emergency services. They say, over the phone, "The physician in the office near our plant isn't interested in an industrial contract. Furthermore, he won't even take any new patients."

I've been told that health maintenance organizations will be the answer. We do have several of these developing in various areas of our state. I have heard that they are supposed to be

"prevention oriented." I haven't found much activity yet to verify that. Most of the staffs seem to be as "acute care" oriented as everyone else in town.

It must be very difficult for the general practitioner who has a few small industrial contracts for emergency care, to be suddenly asked to participate in "medical monitoring." In this era of galloping developments in the medical field, he is hard put to keep up with what is going on in maternal and infant care field, changing childhood diseases, symptoms of adolescents on drugs, new treatments for heart disease, the push for hypertension surveillance, and the ever increasing and perplexing problems of the aged. To expect him to get excited about monitoring apparently well, working adults in a nearby plant and to involve himself with still another government agency, may be unrealistic.

But those who do allow themselves to get involved, to treat work injuries and diseases, are perceived as industrial physicians. Some do get acquainted with the hazards in the workplaces and are usually familiar with the requirements, under workmen's compensation, for medical evaluation of impairments of injured workers. But, it must be increasingly difficult for them to keep abreast of the standards which undergo amendment and change and to comprehend the impact of the flow of criteria documents that can be anticipated with over 500 occupational disease exposures under study. There really is no structured method for these physicians to learn the role of other workers in the occupational health field, such as industrial hygienists and safety engineers. And, they cannot perceive the need for reporting and recording systems which develop accident and disease frequency statistics or for epidemiological studies of the employee population. They need an expert occupational health physician consultant who can tell them, in their language, just what they should be looking for in such-and-such plant. This appears to be a growing need in many states. It certainly is a need in Colorado.

Another need is someone to do the medical monitoring. Instant solution! "How about using nurses?" And each physician who makes that statement thinks of "a nurse"--the one who works in his office or one of those at the hospital. But let us look at nurses as a possibility. What can nurses do?

Any Registered Nurse, newly graduated and licensed, can perform basic nursing functions. One reason that the profession of nursing has not been defined is that the body of knowledge and the basic functions keep changing. Taking blood pressure was not considered a basic nursing function in the 30's. Today, it is not only a basic nursing function, it is a task that can be delegated by nurses to trained Aides. So there is some disagreement in the nursing profession about the current scope of basic nursing functions.

However, if the graduate RN has worked in an institution with expert nursing supervision, she will have refined her patient care skills and developed responsible judgment. If she has worked for a public health agency or graduated with a degree, she will be skilled in taking a health or medical history. If she has worked in a Coronary or Intensive Care Unit for a while, she will be expert in auscultation of heart sounds and probably lung sounds also. Further, she will know how to get into a vein to either start an IV or draw blood, quickly. If she has worked in an Emergency Room, she will be able to function well under stress and handle trauma problems with skill, including minor suturing.

I've just talked about the relatively recent graduate. But studies have shown that the majority of those functioning in an industrial setting, especially in one-nurse units, with limited medical direction, are over 40 years of age. These studies have also shown that the turnover rate, after the first year, is very low, as compared with statistics from other organizations which employ nurses. Fifty percent have been on the job more than six years and 10 percent have been employed in the same position from 11 to 20 years.

So, the number of monitoring activities an occupational health nurse can perform, will depend on her level of preparation, and what she has learned since. The nurse in industry today, for the most part, possesses more skills than she did seven years ago or her counterpart did seven years ago. One reason has been the support industry has given to her continuing education. Another has been her own drive to cope. She often uses her own time and money for additional preparation.

In Colorado, almost every Occupational Health Nurse has gone to educational programs on alcoholism and drug abuse and cardiac resuscitation. Many have gone to coronary care courses and can do auscultation and read a heart monitor. Some have attended workshops put on by Emergency Room Nurses, Family Planning Nurses, and others. We have five who have completed the 11 week Adult Health Practitioner Course and some others who have completed the one month Expanded Role or Primary Care Course.

Because the nurses who work in industry have a history of longevity on the job, it is a wise investment for industry to support financially the efforts of its nurse or nurses to acquire more skills.

A word of explanation is probably useful at this point in regard to the courses just mentioned, which have become very popular with nurses. The original Pediatric Nurse Practitioner Course was developed several years ago at the University of Colorado; it was one semester in length and required the nurse applicant to have a BS degree. The Practitioner was trained to do physical assessment and could diagnose and prescribe within identified limits. Since then a certification program has been instituted by the American Nurses Association for this group. Other Nurse Practitioner courses have been developed in Geriatrics, Adult Health, and School Health at the University, with different applicant requirements. Different organizations offer Critical Care Practitioner courses. Practitioners function in a so-called expanded role but the title "Practitioner" is limited to those who have completed the specific course requirements.

Because of the popularity of the role expansion concept, two shorter courses were developed in Colorado and probably other states--Expanded Role Course and Primary Care Course. They are "field courses" offered in many regions of Colorado and Wyoming with the 15 to 18 day didactic material presentation given in 2 or 3 day segments over a 3 month period. There is built in a requirement for a number of hours of clinical practice in physical assessment with a sponsoring physician. The Expanded Role course is offered by the University of Colorado, the Primary Care Course is offered by the Colorado Department of Health. For the purposes of this presentation, the term Expanded Role Nurse will mean one who has attended the shorter, one month course, has physical assessment skills, and is expected to perform a high level of primary care.

Most of the monitoring activities described in the standards can be performed by a Nurse Practitioner or an Expanded Role Nurse. The only difference between a physical examination by an Expanded Role Nurse and a physician, is that the nurse is oriented to what is normal. She can tell a deviation from normal, but will not pursue the investigation to determine why a parameter is abnormal or the cause. That is the physician's job. The patient is referred to the physician for further study, diagnostic workup, and medical regime prescribed.

So the Expanded Role Nurse can take the health or medical history and be alert to areas of potential concern. The nurse can use a sphygmomanometer to take blood pressure; she can use a stethoscope to do auscultation, an otoscope for ear examinations, an ophthalmoscope for eye examinations, and look into the nose and throat with a flashlight. She can test reflexes with the percussion hammer and has some skills in percussion, palpation, and inspection. She can put blood samples on slides and do pap smears.

Many Occupational Health Nurses today are audiometric technicians and can do hearing screening and testing. Many also can do more sophisticated vision checks than the old Snellen E. Some also take X-rays although I think this should be done by radiologic technicians. These are all the parameters of medical monitoring recommended to date. History, physical examination, perception tests, and laboratory studies. I am not suggesting that the nurse be trained in laboratory work. If an industry wants to do its own laboratory work, it should employ a qualified laboratory technician. But the nurse can obtain the specimens and understand the significance of the reports.

Many nurses are or can be skilled in the parameters of medical monitoring, but what is the nurse's legal liability and will her observations be accepted?

Occupational Health Nurses have been the most vulnerable of all nursing groups to malpractice suits. While there is exposure, the actual numbers have been few, and most of the problems have stemmed from a nurse FAILING to act. The courts have judged nurses to be responsible for performing those acts for which they have preparation and which the community expects. The courts have not been involved with the Nurse versus Physician Practice Acts. Last year, the Colorado Legislature passed a Nurse Practice Act which expands the definition of the practice of professional nursing to include the "...diagnosing and treating of human responses to actual or potential health problems."

The cardiologists and emergency service physicians were strong in their support of this change, for obvious reasons. It is my understanding that many states are changing their Nurse Practice Acts for the same reasons. However, the Act is not limited to nurses functioning in a Coronary or Intensive Care Unit or Emergency Rooms. The change will affect what is perceived by the community as a nursing function. Therefore, Occupational Health and other nurses, who function in the "traditional" manner of 1954 and even 1964, may not be functioning at the level of today's public expectations.

It is a reality of life that when an individual accepts more responsibility, there must be more accountability. But, presumably the increased compensation the nurse receives for these higher level nurse functions will offset the increased cost of Nursing Liability Coverage.

As for acceptance of her observations, the credibility of anyone, on any level, must be tested and proven. While the Nurse Practitioner and the Expanded Role Nurse are assuming responsibility for primary health care, they must have enlightened physician back-up. There is nothing more frustrating to a nurse than to see some deviation from normal, suspect the cause, refer to a physician--only to have him return the patient with a "nothing to worry about, honey" comment. But she does worry. And, because she's bright and careful, she frequently turns out to be right.

Maybe, after a number of years as the one doing the medical monitoring or giving the primary care, the nurse will become complacent and even careless. But, don't under-rate her now. The ones that have the skills, really have them, and they are confident and sure about what they know and do. They also recognize, even more acutely than before, what they don't know.

I asked the head of one of the unions in Colorado, how he thought the membership would feel about nurses doing medical monitoring, especially physical examinations. He said, "Well, almost any nurse could do as good a physical as some I've had. Actually, I guess they would do a good careful job. No, I don't see anything wrong with that idea, though there might be a few guys who wouldn't want a woman examining them."

He then went on to talk about the health and medical records of workers. He was caustic in his comments about nurses who don't keep information confidential and even more critical of personnel officers and managers who "paw through records just to get something on a guy so they can fire him." I looked up the statement on record confidentiality made in 1960 by the American Medical Association House of Delegates: "Disclosure of information from an employee's health record should not be made without his consent, except as required by law."

Earlier I mentioned the need for medical monitoring in small industries which do not now have occupational health services. One method discussed in our state is to have these industries contract with the local Health Department for part-time nursing services. We have many Public Health Nurses who have gone through the Primary Care and Expanded Role Courses and all kinds of Nurse Practitioners utilized in various kinds of clinics--Pediatric, Geriatric, Family Planning, and so forth. These personnel could do the medical monitoring and many other occupational health nursing activities. They would have to be clued in on the mysteries of Workmen's Compensation and insurance benefit programs, profit and loss rationale, disability and absence controls, the role of industrial hygienists and safety engineers, labor-management relations, and the philosophy and purpose of the records used.

I have the feeling that this last item will give us the most trouble. Public Health Nurses are accustomed to exercising complete record confidentiality. They will most comfortably share medical monitoring information with the physician. They will be most resistant to sharing any

health record information with anyone else. This is as it should be. The credibility of the nurse will be mortally damaged if workers feel she violated their confidence. I'm not sure that management personnel in some plants, especially the smaller ones, accept the AMA statement.

Accomplishment of the required and recommended medical monitoring is a formidable but not impossible task. However, I am very curious about how we handle it, and what we do with the information developed.

MEDICAL MONITORING FOR OCCUPATIONAL DISEASE

ADMINISTRATIVE PROBLEMS RELATED TO
MEDICINE MONITORING
Merle Bundy, M.D.

I'd like to spend a little time with you this morning talking about certain administrative problems related to medical monitoring. I'm only going to talk about three of them, because we could spend a long time if we were to get into all of them.

However, before I get into my discussion, Dr. Tabershaw used a term that I think needs a little bit of expanding. Yes, the criteria documents may be "the best information," but some of the interpretations of some of that information may stand a little closer scrutiny than the term "best knowledge" implies.

The three areas that I'd like to address are primarily related to the mini-documents, and here are four of them, so that you can see what they look like in draft form. (slide presented) There are 382 more to come within the next 2 years. I submit that this will only be a beginning. The Toxic Substances List, published by NIOSH, now is, I think, approaching 25,000 toxic substances. I suspect we'll have at least a mini-document on most of them, if not all of them in the next 5 to 10 years. This gives you some idea of some of the things that we're going to have to address. The mini-document is not as extensive as the criteria document. It's primarily a good work practice and a good medical practice document, but it introduces some problems for us. These are the things that we'll talk about.

I'd like to speak of the action level that the mini-document tells about. Now where does that come in? The criteria document has set certain levels that are suggested for a standard. The mini-document calls this an exposure level, so that it isn't a standard. The standard is promulgated only in a specific way according to the law. So the exposure level is what probably will ultimately become the standard, or it will come close to it.

Below that exposure level there is what is called an action level. The level where the physicians and nurses will be involved with monitoring individuals, when exposures reach that action level. And, you as the physician and/or nurse in industry will have the responsibility for that medical monitoring; actually, I prefer the term medical surveillance, which Ernest Dixon was proposing a year and a half ago at the American Industrial Health Conference. Most of the time I'll try to stick to that term, because I think it is a better term.

The second area that I want to talk about is instrumentation. Not only instrumentation in our medical departments, but instrumentation that directs us to do things at certain levels. And a third area that I think needs a lot more attention, the identification of the susceptible.

Let me assure you that I am in favor of medical surveillance. I think this is an area that we

as physicians must accept, and I don't know of anyone who doesn't really believe that this is a good thing to do. It's really the guts of an occupational medical program. It's really the guts of a good preventive medical program. We're going to need time to get there, because the requirements in all of these are going to take some tooling up to do. There are going to be some areas where the kinds of services and requirements that are demanded aren't available from physicians because they aren't available in the area for one of the following two reasons: (1) no interest on the part of the physician to do these kinds of things, or (2) the number or amount of medical manpower just isn't thick enough to split it into all of these requirements.

In these areas, particularly in small operations in small communities, the use of the nurse, the use of other paramedical or physician assistant trained individuals with the review of positive findings by a physician either in the community or a consultant nearby or in a corporate head-quarters will, I think, be necessary. I was delighted to hear Doctor Grais yesterday say that he didn't think a cardiologist necessarily should be the one to monitor stress testing. I think that same kind of philosophy is going to have to pervade in the occupational medical field. Somehow regulations are going to have to cover this. They don't presently do so.

The responsibility is on a physician and we're going to have to provide certain information as physicians to the employer. Alternate methods I think are going to have to be considered, and after good faith efforts and with full knowledge of the employees and their representatives, we're going to have to try to solve the problem in a variety of ways. I don't think there is any one single way. Another method would be to provide mobile units that go from location to location with medical supervision either from a private group or a corporate group or however it might be proposed. I think these ways are going to have to be initiated if we physicians are going to do the job that we are going to be required to do, and if we're going to be able to do it at all.

Now let's get back to the action level. I agree, and I think most of you would agree, that at some level below a standard of exposure, we ought to begin to take a pretty good look at the people who are in those exposures to see what subtle changes might be occurring. I think my argument with what's in the mini-documents is what I think is lack of sophistication in being reasonable when they set an action level. For instance they say 50 percent of the exposure level in one document. They'll say 10 percent in another document. In manganese the exposure level is 5; the action level is 0.5 of a milligram per cubic meter. This flies in the face of logic. I think someplace along the line there has to be a more reasonable "look-see" at where that action level is.

What I'm saying I guess is let's practice good preventive medicine in an occupational setting. I think the surveillance of individuals from time to time and on a periodic basis is something that we as physicians need to be constantly doing. Let's not call action levels necessarily something that is due to a hazardous exposure, because many of the levels that they are talking about don't reach that if the standard was set properly. I guess maybe one of my hangups is semantics. Let's don't do things under the guise of something else.

For instance, my concern is if we've got an action level that is there because of an instrumentation problem that we can't accurately measure, and we set this as a safety level, let's say so; let's not say it's because the level is hazardous. Let's say it's because we can't measure that accurately. I think this takes some of the sting or the onus off of the individual who is being exposed. And I think that his feelings should have some consideration in this rather than for us to blithely say we're going to set the action level way down here.

If we have a degree of accuracy for which the instrumentation is excellent, then why not pick a level at 80 percent? If we want to do monitoring or surveillance under that level, this is good occupational and good preventive medicine practice, but let's try to keep our terms and thinking a little nearer what I think follows logic. In another area along this same line, I don't believe enough consideration has been given to the yearly negative examinations that you're going to be doing. I think people generally believe if they've got a negative finding this is good insurance, and it is; but year after year after year they may become complacent; they may become careless. I would much prefer to see some consideration given, after three consecutive negative examinations, to consider doing it every 3 years, if they're under 30 years

of age, because we get a great many of our work force in the younger age groups. I see no need to monitor such individuals that often. Under that age we don't do it in many other areas. We use age or some combination of age, levels of exposure, and length of exposures, as criteria for such a procedure. I think they mention sputum cytology in the coke oven emissions. There are pretty good studies to show that even for those individuals who smoke one does not begin to find any kind of cytologic changes under the age of 32. That means they either haven't smoked long enough or the effect doesn't occur (and for non-smokers under the age of 35), but there is no mention of this in the criteria document.

I think these kinds of considerations are important, because when we start initiating sputum cytologies and making sure we get it either every 6 months or every year, we'd better be doing it on the people where the results are going to show up. Let's not go back to the same old ballgame. If we're going to improve the quality of our medical practice, let's really do it.

Now a few words about instrumentation. Instruments are not infallible; people who operate instruments are not infallible. What arguments I've heard over a single test! I think we have to be awfully careful as physicians and nurses in saying this is that level in this individual. If it is abnormal or deviates from the normal, I prefer always to have a second test that confirms it. I think we can get into an awful lot of difficulty if we rely on a single test to determine abnormality.

Some of the instruments measuring the atmosphere that require us to initiate an action are as much as 50 percent inaccurate. I'm not the originator of this statement, but I think it expresses exceedingly well how precise we try to be with such imprecise instrumentation. These are some of the problems you are going to face as you deal with the patient, his representative, or with management. We have to be rather perceptive, and we need to be awfully careful when we make some suggestions. If it is poor instrumentation, I think we'd better admit it. I think money in far greater quantities needs now to be poured in to develop the kind of instrumentation that will make our job easier, because we can depend on the result that we get. This is I think a most important area and one that you're going to be constantly asked questions about: "What does this mean?" I urge you to be prudent before you make your judgment that you really have instrumentation or results that you can stand on.

And, lastly, let me say just a few words about the determination of susceptible. I think with all due respect to the criteria documents and the mini-documents that are coming, people are not alike; they react differently. I think we need to do a great deal more in trying to identify susceptible individuals so that we don't put them in a spot where their health is even going to be jeopardized. I think it is possible for us to do far better job placement, if we get a better way of identifying the susceptible. I don't have the answer for you; all I know is that there ought to be an awful lot of money spent to try to identify the individual whose pulmonary system is going to react to smoking, to a dust exposure, to a mist, or to some chemical pollutant.

I think this is needed because we can have people work side-by-side for a lifetime; one individual has devastating results from that exposure and the other individual has no measureable changes. I'd like to distinguish between the two. Don't wait until we do our surveillance to find that he is responding either adversely or in some fashion. I'd like to get that identification before I place him. I trust that more money can be directed toward that area.

I don't mean that we ought to neglect the development of criteria documents or the minidocuments, not in the least. All I'm saying is here is an area where we as physicians, as nurses, as medical people charged with maintaining the health of our work force, where I think we could be immensely assisted if we had a way of developing or identifying that susceptible individual.

MEDICAL MONITORING FOR OCCUPATIONAL DISEASE

MEDICAL MONITORING AND TWO NEW STANDARDS
Miles O. Colwell, M.D.

INTRODUCTION

Since the previous speakers were committed to cover the general scope and extent of occupational medical activities directed at monitoring for occupational disease, it was agreed that I should use the case history approach to demonstrate what my company has been doing to monitor two very important worker exposures in the aluminum smelting industry (heat and inorganic fluoride) and how we have interfaced with government activities concerned with these same exposures.

The increasing influence of government on the practice of occupational medicine has been clearly demonstrated by prior speakers. I favored the passage of the Occupational Safety and Health Act of 1970, I still support it in principle, but I do have some concern about the manner in which certain facets of it are being implemented. I am most concerned about the lack of cooperation between industry and government and have expressed my views in some detail on the subject in the literature (see reference 1). My remarks today will again emphasize this concern.

I have served as a member of the NIOSH Advisory Committee for the criteria documents dealing with hot environments and exposures to inorganic fluorides. The document dealing with hot environments (see reference 2) was finalized and forwarded to OSHA in 1972; the second, concerning inorganic fluorides, is in the draft stage.

OCCUPATIONAL EXPOSURE TO HOT ENVIRONMENTS

The second draft document on Hot Environments (the first did not involve the Advisory Committee) was sent to me as a member of the NIOSH Advisory Committee with a cover letter dated April 26, 1972. The document was voluminous and the bibliography contained 130 references. The cover letter listed 42 questions concerning the document with the request that replies to them be in the NIOSH office by May 8, 1972.

My consultant and staff complained bitterly that a proper response could not be made in so short a time, and I agreed, but we did our best and did reply. The Advisory Committee met for one day on May 11, 1972, to discuss the document. We were told we would have no opportunity to comment on the revisions which would result from our meeting prior to publication of the next draft. This, to me, was not the proper use of the assembled talent. Eventually, a final document was

sent by NIOSH to OSHA. To date, a standard based on this document has not been promulgated due to the lack of consensus by the many consultants called in by OSHA. In my opinion and with due respect to NIOSH personnel, such an eventuality should have been recognized early in its deliberations for the following reasons:

1.) The need for the proposed standard had not been demonstrated - an attempt was not made to document the frequency and severity of heat illness in U.S. industry.
2.) A validated data base for setting the proposed environmental measurement standards did not exist.

I base this opinion on the vast amount of environmental and physiological data we have obtained in our smelters since 1961. Alcoa heat stress studies have been performed under the guidance of Dr. Steven M. Horvath, Director and Professor, Institute of Environmental Stress, University of California, Santa Barbara, California. We have performed 14 studies, each lasting a few days to three weeks, involving 273 subjects. A computer program has been developed at the Institute of Environmental Stress to process all recorded data and to compute the necessary derived values such as WBGT (^{o}F), radiation (BTU/hr), convection (BTU/hr), evaporation required (BTU/hr), evaporation maximum (BTU/hr), evaporation maximum (BTU/hr), and VP air (mm Hg).

During the summers of 1971 and 1972, studies on maximum working capacity were made on volunteer aluminum smelter workers prior to and immediately after a normal 8 hour shift. The metabolic energy expenditures (oxygen uptake of various representative tasks undertaken by the workers) were also determined. The methodology and results of these studies have been reported. (see references 3 and 4)

Time does not permit me to present the evidence in detail as to why I say a validated data base did not exist for establishing the proposed environmental limits which would dictate remedial action in the workplace, however, for those interested, this information has been published and a reprint will be furnished you upon request (see reference 5), "Heat Stress and the New Standards." ·

Regarding the need for a standard, I am not aware of any recent published data documenting the frequency and severity of heat disorders in the U.S. We do record all such disorders in Alcoa, and we couldn't justify our studies on the basis of excessive heat disorders. Our data are used to assist the industrial engineers in establishing workloads and patterns that promote maximum worker efficiency--this is good for the employee and for the company.

I assume that one day a feasible work practices standard for hot environments will be promulgated and I will applaud such action. However, if such is not the case and an impractical, unworkable standard is established, the economic and social cost to U.S. industry could be staggering.

I will close this subject with one final comment. In the preface of the criteria for a recommended standard, Occupational Exposure to Hot Environments (see reference 2) published in 1972, is this statement.
    "It is intended to present successive reports as research and epidemiologic studies are completed and sampling and analytical methods are developed. Criteria and standards will be reviewed periodically to ensure continuing protection of the worker."
This is 1974 and much data has been gathered and analyzed since the 1972 document. Obviously, this is not an emergency standard we are considering. The involved parties--industry, union, and government--should take a look at the evidence to date; wouldn't it be shocking if we were to find that after all this discussion about a standard we had during the interim, failed to adequately monitor for the disease (the frequency and severity of heat disorders)?

OCCUPATIONAL EXPOSURE TO INORGANIC FLUORIDES

INTRODUCTION

Fluoride is ubiquitous in man's environment. All foods contain traces of fluoride, and drinking water in the U.S. may vary from trace amounts to as high as 16 ppm (see reference 6).

Soluble fluorides are rapidly and efficiently absorbed from the stomach and intestine, in most cases as the fluoride ion (F^-). Insoluble or slowly soluble fluorides, or soluble fluorides in the presence of complexing elements, e.g. calcium and aluminum, are absorbed slowly and incompletely.

Once absorbed, fluorides, like chloride, are distributed rapidly throughout the body water exclusive of that inside cells. A part is speedily deposited in bone and essentially all of the remainder is excreted via the kidneys, although under hot, moist conditions McClure, et al, found up to 46 percent of ingested fluoride in sweat (see reference 7).

Fluoride is a prototype bone seeker; it offers the best example of an element that is stored in bone and practically nowhere else. The larger the daily fluoride intake, the higher (but not proportionately) the ultimate bone concentration. The first demonstrable effect of chronic fluorosis in humans is X-ray evidence of increased bone density. This takes many years to occur. Studies of populations whose drinking water contained high fluoride levels show that increased bone density, or osteosclerosis, was not apparent roentgenographically where the fluoride concentration was less than 4 ppm, and that 10 to 15 percent of individuals drinking water containing 8 ppm showed evidence of increased bone density (see reference 7). Since excretion in the urine parallels intake those individuals would show about 4 mg/L and 8 mg/L respectively.

Aluminum smelter fluoride exposures consist of both particulate and gaseous components as do most industrial exposures except where the process involves the manufacture or use of hydrogen fluoride. The route of entry into the body is via the lungs and gastrointestinal tract. (The latter includes particulates inhaled and subsequently swallowed, plus those ingested because of poor hygienic practices such as eating, drinking, and smoking in the area of exposure or in clean areas without first washing one's hands.)

ALCOA FLUORIDE EXPOSURE SURVEILLANCE PROGRAM

The surveillance program since the early 1940's has involved air monitoring, urinary fluoride analyses and medical evaluation of exposed workers.

URINARY FLUORIDE SURVEY PROGRAM

Analysis of increments of 24-hour urine samples in the 1940's gave evidence that a spot sample taken at the end of day towards the end of the work-week could be used as an index of worker exposure at that time. If enough workers could be sampled at one time one could make some assessment of the fluoride levels in the area in which they worked. The spot urine program, although of some value, never gave the quantity of data needed for a good evaluation of the workplace. The men were not informed as to why the urine sample was being collected and, therefore, cooperation was poor.

In 1965, it was decided that the union representatives, the smelting workers, and pertinent government agencies should be fully informed concerning our urinary fluoride survey program. This was done in one plant and cooperation of the workers was so great that the same approach was taken in all other smelters. We now collect urines under specified conditions at appropriate intervals during the year in all Alcoa US smelters. Both post-shift and pre-shift (after away from work 48 or more hours) urines are collected.

In 1970, the data from the urine fluoride survey programs was computerized. As of July 1974, about 6,000 smelting workers were involved and 55,000-man fluoride surveys had been processed in the computer program. This includes data from all Alcoa US smelters and those with which we are associated in Australia, Suriname, and Brazil, some Norwegian data has also been processed in our computer program.

We have learned much via this expanded data base and through our ability to develop computer programs to find answers to many questions which would be impossible to solve without such tools. I will discuss some of the opinions we have developed in this regard when I discuss the NIOSH criteria document on inorganic fluoride exposure.

AIR MONITORING

We have conducted air monitoring programs to check the general level of fluoride in the work environment and to determine areas of highest exposure. We think air monitoring is of limited value in determining worker exposure because of the variability of work patterns and because it excludes fluoride ingested through the unhygienic practices of workers (eating, drinking, smoking in areas of exposure or clean areas without washing hands).

MEDICAL EVALUATION OF WORKERS

We have published the results of a study of workers in an old smelter with no environmental controls in 1946 (plant closed in 1949) and compared those findings with those of a study of workers in a smelter with environmental controls in the 1960's (see reference 8). In the old smelter, urinary fluoride levels were much higher than in the more modern smelters. In the smelter with no controls, 76 of the 79 workers X-rayed had evidence of increased bone density (Fluorosis). In the smelter with controls, none of 231 X-rayed had increased bone density. An X-ray survey last month of 55 of those employees still working in the potrooms showed no evidence of increased bone density, and none of these 55 had less than 15 years of service in areas of exposure.

CRITERIA DOCUMENT FOR OCCUPATIONAL EXPOSURE TO INORGANIC FLUORIDES

My experience as a member of the Advisory Committee on this document was similar to that I had when I served on the heat stress committee. I received the first draft with a cover letter dated July 8, 1974. It included a list of 24 questions to be answered by August 19, 1974. The bibliography contained 284 references. Members of the Committee were informed at the end of the one day meeting that they would not have an opportunity to review the next draft prior to its publication.

The goal of the recommendations is to prevent increased bone density in workers exposed to fluorides. The recommendations for standards in the document include an air fluoride standard and a post-shift urine fluoride standard. The recommendations were based on data presented in one paper, and they totally ignored all other documented evidence concerning industrial exposures and that very important data concerning populations exposed to high levels of fluoride in drinking water. The Hygienic Guide (see reference 9) for atmospheric levels and the Biologic Monitoring Guide (see reference 10) for urine fluoride produced by the American Industrial Hygiene Association were also ignored. Incidentally, the paper on which the NIOSH contractor made his recommendations was available to AIHA when their Guides were developed. The air and urine standards recommended in the criteria document were both more restrictive than those recommended by AIHA. Alcoa experience would support the latter rather than the NIOSH draft document.

Aloca's experience would indicate that a post-shift urinary fluoride is of no use in assessing body burden of fluoride or as an index that workers might develop fluorosis. It is only of value on a group basis for assessing environmental controls and hygienic and work practices.

The criteria document does not deal with preshift urinary fluoride levels but, in my opinion, this, rather than the post-shift value, is an index of fluoride body burden and could therefore be used to predict the possible occurrence of chronic fluorosis in some members of a group if not in a particular individual.

The suggested follow-up air and urine monitoring requirements in the event either or both were high at the time of survey were not practical. They failed to take into account the fact that nothing happens to the worker who is exposed very quickly--it takes years to develop increased bone density.

There is the question of standardizing the procedure for determining increased bone density roentgenographically. We now rely on the experience of the very few available experts.

The paper used as the basis for recommendations noted above suggests that the albuminuria found in exposed workers might be related to fluoride intake. We asked our computer to check this for us last month and found that 36,023 urines of exposed workers showed 12.5 percent albuminuria and that 637 controls showed 13.2 percent--no statistically significant difference.

There is no need in my continuing to describe areas where I differ with the draft criteria document, because it undoubtedly will be changed. I only do so to emphasize some facts and inferences I shall now make in summary.

SUMMARY

First, I do not doubt the sincerity of NIOSH and OSHA in their effort to protect the safety and health of the worker.

I do question the approach NIOSH has taken in the development of the two criteria documents with which I have been personally associated.

I realize that NIOSH personnel and their contractors are expected to base their recommendations on published data, but in my opinion, that approach, in view of today's fast-moving developments in our field, is antiquated. Why can't or don't those developing the criteria by which we must monitor for occupational disease examine data available in industry such as that I have described today?

And, finally, there always will be emergency situations needing prompt and forceful attention, but the bulk of our potential occupational medicine problems is not in that category. Programs for monitoring to prevent disorders can be developed on a programmed basis that will result in better scientific, social, and economic acceptance.

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MISSED AND MIS-DIAGNOSES IN CHEMICAL EXPOSURES

Dr. Eckardt: In the treatise "De Morbis Artificum" (Diseases of Workers) written in 1713, Bernardino Ramazzini urged that physicians add one more question to the list of those recommended by Hippocrates. That question is "What occupation does he (the patient) follow?" Unfortunately, this is a question not often asked and pursued by the practicing physician. As a result, the diagnosis of a patient's condition can often be missed or a mis-diagnosis made.

My own introduction to this concept, except for a few conditions such as dermatitis in machine shop workers or lead poisoning which I received in medical school, began before I actually entered the field of occupational medicine. While I was serving in the Army as Chief of Medicine of a small station hospital, a GI was brought to my office from the stockade by an MP. He had been accused of being drunk on duty and summarily placed in the stockade. After being there several days, he still appeared drunk, which puzzled the stockade officer who sent him up for medical evaluation.

As the boy entered my office, it was apparent immediately that he was unsteady on his feet. He staggered and lurched from the door to the chair I motioned him to sit in. In talking with him, it became apparent that his speech was slurred, that he really seemed to have little concern about his predicament, and that his attention continually wandered away from my history taking. Having seen "The Lost Weekend" and having dealt with alcoholics before, I was inclined to believe that in some way he had been able to maintain his source of alcohol. When I probed this line of questioning, he steadfastly maintained that he had consumed no alcohol since being placed in the stockade. Although not fully believing him, my thoughts turned to some central nervous system disorder, such as a cerebellar tumor, a large frontal lobe tumor, or perhaps a subdural hematoma. But continued observation of him and questioning disclosed no other signs or symptoms of brain disease. He just appeared drunk!

It was then that I asked him what he was doing when he was placed under arrest, and he gave me this story. His job was to fumigate the clothes of soldiers returning from the European theatre. To do this, large bundles of clothes were taken to a shed away from the main area of our operations. They were placed in the shed, doors and windows were sealed with tape, and the fumigant released into the shed. After 24 hours, the shed was opened wide, and their

instructions were to leave it open wide for 24 hours before removing the clothes. However, since this delayed their work, they often went into the shed and removed the clothes and began sorting them before the 24 hours were up. He said they often still had the sweetish odor of the fumigant, but it didn't seem to bother them at all. He had been on the detail for two weeks, while other men had been assigned in and out of the work on an almost daily basis.

When asked what the fumigant was, he said he wasn't sure, but he thought it was methyl bromide! At this, my ears immediately perked up, and I ordered an immediate blood bromide. I dismissed the MP, called the stockade officer, and told him I would keep the boy in the prison ward until the blood bromide was back. It came back and was several times the toxic level, so we moved him to an open ward, began chloride therapy, and got the charges dismissed against him. This was a simple case of bromide intoxication which was mis-diagnosed as drunkenness, and the diagnosis was missed until questions about what he actually did were pursued. It is not enough to know the occupation--in this case, a private in the Army--but what the person actually does on the job--in this case, fumigation--and whether or not he follows the rules laid down for the job.

Upon return to civilian life and more residency training, I picked up extra money by working my off Saturday mornings in the Personnel Health Clinic of my hospital. One day I saw an employee who had been admitted to the hospital for infectious hepatitis. Since his course did not follow that of a usual hepatitis, he was discharged from the hospital with a diagnosis of toxic hepatitis of unknown cause. When I saw him he was well but was in for a follow-up visit. He job title was electrician, but in questioning him I found out that his job was to clean the electric refrigerator motors. In this job, he would take a pail of carbon tetrachloride, go from refrigerator to refrigerator in the hospital, and clean the motors with a rag dipped in the carbon tet. Since the motors were underneath, he had to lie on his back, put his head into a confined space, and then clean the motor, which was also in the confined space. He would clean perhaps 10 or 12 refrigerator motors a day and had been doing this for some time before his hospital admission. Here was a case of carbon tetrachloride liver damage, a missed and mis-diagnosis, because no one had inquired about what he actually did. His job title of electrician was certainly no clue. This emphasizes again the importance of knowing not just what a man's title is, but what he actually does on his job.

My third case concerns a man I saw at our refinery in Aruba. I was doing a series of skin examinations on these men. One man had very large calluses at the proximal interphalangeal joint of the fourth and fifth fingers of his right hand. I thought to myself, "Aha, here is an unusual and exotic occupation to produce such unusual calluses!" Imagine my chagrin, however, when he told me in response to my questioning that he got his calluses playing cards! He loved to play cards and played a great deal, in the excitement of the game, he always banged his cards onto the table, rapping those two knuckles continuously in doing so!

It appears that Ramazzini's admonition was a good one. It is important to inquire of a man's occupation and just what he does in his job if we are not to have missed or mis-diagnoses of his ailments.

MISSED AND MIS-DIAGNOSES IN CHEMICAL EXPOSURES

CHEMICAL EXPOSURE, THREE CASE HISTORIES
Carl U. Dernehl, M.D.

One of the challenges of occupational toxicology is the fact that the toxic responses of humans are seldom specific for a toxicant. Nausea and vomiting is indistinguishable as to origin except perhaps after hemorrhage. Respiratory symptoms from chemical exposure are initially indistinguishable from acute viral or bacterial bronchitis. To confound the physician even more, an acute illness need not become evident at the time of or immediately after exposure. Here are three cases which point out the need for a high index of suspicion when dealing with the health problems of a chemical worker. I hope they raise some questions for discussion.

CASE I

JHB was a 27-year-old chemical engineer. On a Wednesday evening he returned home from work feeling a little bad, and by bedtime he had a severe headache, mild nausea, and a marked aching of his back and testicles. When he voided at bedtime, his urine was reddish black and seemed to contain a lot of blood. He called his family physician, who sent him to the hospital and ordered laboratory work and a KUB X-ray. The urine was found to be grossly hemorrhagic, and the X-ray did not show the presence of a stone. By morning the urine contained considerably less blood, and an intravenous pyelogram was read as normal. The following day only traces of blood remained in the urine, and the patient was discharged with a diagnosis of bleeding of unknown origin, possible stone. The patient was released to return to work on Monday by his family physician and a plant nurse.

EPILOGUE

On Monday morning JHB was in an office he shared with BBC when he commented that his testicles were aching. BBC commented that his were aching, too, and had been since the previous Wednesday when he had felt badly. After further comparing experiences, both men then reported to the medical department, where the following story was developed. The illness of JHB was reported in detail, and BBC stated that he also suffered from headache, malaise, backache, and testicular aching on Wednesday evening. Early in the evening he noted his urine was highly colored and a little "pinkish," but by bedtime it was much lighter in color. On the following day he had only a mild headache, not relieved by aspirin, and testicular aching which continued to the time of the interview.

Obviously both men had been exposed to something at work which resulted in a hematuria on Wednesday and Thursday and somewhat similar symptoms, of which testicular aching brought them to the medical department.

On that Wednesday, both men were making a special experimental run of polyvinyl chloride catalyst. In this case, acetic anhydride was reacted with hydrogen peroxide to form diacetyl peroxide. The special part of the reaction was the addition of cadmium acetate to the reactor kettle. The reaction was carried out in two kettles cooled with brine and stirred by sparging with nitrogen. The kettle used by BBC had automatic temperature control, so all he had to do was occasionally lean over the tank and read the thermometer to check the operation of the cooling system. The tank used by JHB had manual temperature control, so he had to lean over the vessel frequently to read the thermometer and adjust valves controlling the flow of the cold brine solution of the cooling systems. In both vessels the nitrogen sparge bubbled to the surface and carried fine droplets into the air above the vessels. Being forced to lean over the vessels, both men inhaled some of the droplets, with JHB inhaling the greater amount because of his need to both read the thermometer and adjust the refrigerant flow.

A review of these events suggested that both men had inhaled droplets containing cadmium acetate, and cadmium is known to be a severe kidney irritant causing varying degrees of hematuria.

Urine samples were collected in specially cleaned pyrex bottles from each man and were sent to a laboratory for analysis for cadmium. Three control urines were also collected and analyzed. The control urines contained no measurable quantity of cadmium. The urine from BBC contained 45 micrograms per liter and that of JHB, 66 micrograms per liter. Thus, 5 days after exposure to the soluble cadmium acetate, each man was still excreting cadmium, with the more heavily exposed JHB excreting the greater amount. The cases were accepted as occupational in origin and were illnesses which would have gone unrecognized had not aching testicles precipitated an investigation. Both men recovered fully and 23 years later still have normal kidney function.

CASE 2

Close to the end of the work day, operating difficulties in a production unit resulted in a spill of about 50 gallons of ethylene dichloride, 1,2 dichloroethane, on a concrete pad. An operator, HB, assisting in corrective action, was exposed to breathing vapors of the chemical for a long enough time for him to become lightheaded and to stagger when he walked. Having brought the situation under control, he staggered to fresh air and sat down for a while. After about 10 minutes, his lightheadedness passed, and he felt normal. Since it was now shift change time, he did not want to hold up his car pool. He left the plant without reporting the incident to the medical department as he should have done. He arrived home some 45 minutes later feeling well.

EPILOGUE

HB ate a good supper, chatted with a neighbor, watched TV a while, and went to bed about 10 o'clock. At 11 o'clock he awoke with nausea and pain in the abdomen. He vomited several times in the next hour, and the abdominal pain grew worse. He went to the emergency room of a local hospital, where he was seen by an intern who made a diagnosis of probable appendicitis and notified the staff surgeon on call that night.

The staff surgeon arrived while the operating room was being set up and the patient prepared for surgery. The surgeon was disturbed by a fever of 102° F, an essentially normal white blood count, and pain localized closer to the umbilicus than to McBurney's point. In addition, the patient's nausea was practically gone. Further questioning by the surgeon about how the patient had felt the previous day and evening brought a recollection of the episode at the plant.

The surgeon called the plant physician and asked if an illness such as this could result from exposure to ethylene dichloride. He was told that nausea and vomiting were commonly delayed for 6 to 8 hours after an acute exposure to ethylene dichloride and that abdominal pain occurred rather commonly in the more heavily exposed cases. He was advised to keep the man under observation, since it was felt the man's condition was due to his exposure. Abdominal pain disappeared within the next 2 hours, although tenderness remained for another day.

Delayed nausea and vomiting are common with some chlorinated solvents, and I have seen it with carbon tetrachloride and dichlorethyl ether also.

CASE 3

A barge, which had just been returned to the plant after carrying a load of vinyl acetate, was found to be so full of loose rust that cleaning was required. Steaming and airing was used to remove monomer vapors, and on the following day, a labor gang proceeded to sweep up and remove the rust. In spite of ventilation, the dust concentration became unbearable, and the men were issued dust respirators. These apparently became clogged with dust, and most men took them off and tied bandanas over nose and mouth. The barge was cleaned by 2:00 p.m., and the men finished that day at various other assignments after showering and putting on clean work clothes.

EPILOGUE

SJ went home after work, ate supper, and went to watch a soft ball game. About 9 o'clock, while at the ball game, he became nauseated and developed a severe headache. On the way home he started to ache all over, and shortly after arriving home he vomited. He went to bed at once, and about 10:30 he had a hard shaking chill, and his temperature rose to 104° F. He called his doctor who had him admitted to the hospital, where he vomited again and had repeated episodes of chills until about 3:00 a.m. A thick blood smear for malaria was negative, and blood was drawn for culture. The only blood abnormality was a white cell count of 28,000 with 70 percent neutrophils. By morning his temperature had dropped to 100.2° F and his aching was much less. A tight feeling with a desire to cough which had been present was lessening, and a chest X-ray was normal in appearance. Had it not been for intervention by the man's plant physician, his discharge diagnosis would have been fever of undetermined origin.

As it happened, when the plant physician came to work that morning, four of the five laborers who had worked in the barge were waiting to tell him the same story of delayed nausea, vomiting, chills, fever, headache, and body aching which had practically cleared by morning with only a vague general malaise remaining. All had a leucocytosis in excess of 18,000 with pronounced neutrophilia. One man recognized his illness as the same as the "zinc shakes" he had once previously after oxyacetylene cutting of some galvanized metal. The missing fifth man was located in the hospital, and his physician was given the diagnosis, metal fume fever.

The mystery was how the disease came about. Iron oxide fume or dust does not cause fume fever. zinc oxide fume causes the disease, but the dust does not; and besides, there had been no welding anywhere near the barge.

A laboratory man was sent into the barge with a fine brush to sweep up any remaining fines he could, and these were taken to the laboratory for analysis. Spectrographic analysis showed a high copper content in the dust, which could not be explained until a laboratory analyst remembered that copper naphthenate was sometimes used as an inhibitor in vinyl acetate shipments. It was concluded that one micron or less copper particles had precipitated out of the copper naphthenate or perhaps had plated out on the rust particles, as several chemists believed. Whatever form the copper dust was in, it caused metal fume fever in the five heavily exposed men just as copper fume can.

MISSED AND MIS-DIAGNOSES IN CHEMICAL EXPOSURES

CARBON MONOXIDE AND LEAD EXPOSURE
Bertram D. Dinman, M.D.

When you get called at two o'clock in the morning, you get out of bed not too fast anymore, and your mouth still tastes not too great, and you reluctantly answer the phone. You know you have to answer the phone. The call on this February night was one which sounded all too familiar. It was: "The family is sick; we're throwing up, we feel sick in the stomach; we have a headache. We're just sick." You know very well there is a lot of viral gastro-enteritis going around. Well, you say: "Why don't you take them down to the emergency room?" "Well, we're just too damn sick, Doc. We just feel awful. The kids are upset; the wife's upset; I'm upset; the family dog is upset. So, maybe you still make house calls under these circumstances."

You rouse yourself to get to that house this February night. It's cold out and the car doesn't start too easily, but that is what you expect for February anyway. You think, maybe next time you'll put the car in the garage before you go to bed at night.

You get to the house, you're ushered in, and there's everybody sitting around looking sort of flushed, and their guts feel just bad, and they're throwing up. You look at the kids. There is no previous history. They've been feeling so-so the last couple of days, but that's about the worst of it. You'd expect that, too. They've eaten fairly well, but they've been off their feed for the last couple of days, too. This is pretty typical, of course, once more.

You check their bellies and there is no localization, no real tenderness, actually. There may be some increased peristalsis, but nothing to write home about. They just look punk. Instead of being a little pale or a little shocky looking, like a severe gastroenteritis, they don't look particularly pale. Well, maybe it's the light; and the family dog is not very happy either, by the way. Well, you know there is a lot of gastroenteritis going around town, and you know it is a viral gastroenteritis, and this is what it looks like. So perhaps you might leave some rectal thorazine or a prescription to at least knock down this vomiting and nausea. And you go home.

There is an epilogue. The next morning you don't feel particularly clear about the diagnosis or very comfortable about it, and you call up, and there's no answer. All too often this family is dead, every one of them--dog, kids, mother, father. Very simply, you've missed the diagnosis of incipient carbon monoxide intoxication in a tight house that is being heated by a space heater or a furnace which is inadequate. The only ventilation is by way of the chimney. You didn't make the diagnosis because you didn't see any staggering, you didn't see any impair-ed consciousness.

These symptoms are what you have been led to believe are necessary for diagnosis. Oh, there was a headache, in retrospect a very nasty severe headache, but this is also associated with viral gastroenteritis. And maybe if you had looked closely, you might have seen a pink face, but it wasn't a real cherry red color. Unfortunately, you fall into the trap of expecting some impaired consciousness, which is not present at about 20 to 35 percent carboxyhemoglobin. This table demonstrates the range of signs and symptoms with varying percentages of carboxyhemoglobin and what you might expect. (slide presented) And you'll see here that in this range of about up to 20 percent there is very little symptomatology. There may be head-ache, and it is without doubt the most common persistent reproducible symptom that you find, just plain headache. The headache starts at about 15 to 20 percent carboxy, and that is all there is. When you get past the 20 percent range, the headache is really persistent, and this is the presenting symptom. After that range you start getting into abdominal discomfort, and that's all it is. It's nothing localized, no abdominal rigidity, no guarding. There is nothing indicative of increased or decreased peristalsis. They just feel punk; they don't feel like eating.

There is some nausea and vomiting as you get up to the 30 percent carboxyhemoglobin range. This is essentially the range that we're talking about and which this family demonstrated. Headache, severe. It may be frontal; it may be occipital. There's nothing characteristic about that. There is a severe headache, abdominal discomfort, and vomiting; and that's about all you're going to hear about. Now if you walk these people up and down two flights of stairs, you may see some dyspnea on effort. You can get them down to about 15 percent in about 3 hours through exposure to fresh air. But if the diagnosis is not made now and the house is closed up again, it progresses.

Now, about this cherry red color. If you've seen a lot of cases of carbon monoxide poisoning, you can pick this up at around 25 to 30 percent. If you haven't seen cases or haven't worked with it, you will find that you will not notice it. The cherry red color only starts appearing at around 30 to 35 percent; and here you have to be very sharp and on your toes, because you are likely not to see it, even at that stage. It's around 35 percent that you start getting a really distinctive cherry red color.

Among people who work with carbon monoxide, the major symptom is headache. The patients complain about it bitterly, and they will know it's there. If the range is not a wide one, which will happen with a concentration around 200 to 275 ppm in the air, over the course of about 3 to 4 hours you will get only a headache. As long as the concentration doesn't go any higher and stays in equilibrium, that's all that you're going to have. The people who work with it know that they don't like it. But the layman who does not work with it, the unfortunate contact with it at home, the headache, abdominal discomfort, nausea and vomiting can and do masquerade in the whole family as an acute viral gastroenteritis. The consequence of missed and mis-diagnosis is severe.

My next subject is another old turkey. And I think it is common enough to be repeated often. This concerns once more the apparently severe, acute, hot abdomen. This is a man you see in the emergency room; he has a severe abdomen. He has a hot belly; it looks like one; and this is what the surgical resident thinks is indeed the case. The man's age is 55. He has had this developing within the last 6 to 8 hours. He has had some cramps before over the last several months. These just come and go; and he put this down to a long history of uclers in the past, though these cramps were a bit different from the usual ulcer symptoms. They didn't have the food association as far as relief was concerned, but he thought he was getting older and this was the result of a long history of an ulcer problem. In addition, he had diabetes, and he thought maybe this had something to do with it, too. He presented the emergency room with an acute abdomen. The medical history had been reviewed including the ulcer history; some questionable ulcer bleeding in the past was presented as a differential diagnostic possibility.

His pulse was 134 when he was seen having this acute abdomen. And, after a short period of time, there was some remission. The pulse went down to about 93. During the period of acute abdomen, you found a rigid belly, no localized tenderness, some questionable rebound tender-ness, practically an absence of peristalsis, and the man was gray. He was sweating--a cold sweat--supporting the worst fears of the admitting resident, a perforated viscus. It certainly looked like a real possibility; except that this acute abdomen, after you had watched it for

about 5 minutes, gradually cleared, and now the belly was soft. There was no guarding, there was now no tenderness. The pulse was down. Peristalsis was still not present. Had he perforated, and would he die in collapse? No, there wasn't any collapse; his pulse and blood pressure were normal; his belly was soft. This immediately should raise a red flag.

Acute bellies, acute surgical abdomens, don't suddenly clear like that, unless they have perforated quite a while and the man is going into shock. Yes, then it may get soft; but that's a pretty unusual and pretty late situation, and it doesn't happen after 10 minutes of a tight belly. The lab work was not very interesting, except that he did not have a leucocytosis. At this stage you wait. The belly comes back again and it's severe. It's a real tight, hot looking belly; except there is no leucocytosis, there is no fever, and unfortunately, in too many cases the abdomen is cracked. Diagnosis is entertained of acute appendicitis or of perforated ulcer in this particular patient (which was not the case).

There is one very simple remedial action, or at least diagnostic action, that can be taken. It is simple to make a differential diagnosis within 2 minutes. It doesn't require a laboratory; it doesn't require any great acumen except asking what the man's occupation is. If there is any possibility that you're dealing with lead intoxication, one very simple thing should be done. You can't hurt the patient by the maneuver, and yet you can save him from the surgeon's ministrations. Give intravenous calcium gluconate, 10 cc's slowly injected of a 20 percent solution. Make sure you get it intravenous, because if you get it outside the vein, you'll get complaints. That's all you need to do. Let the surgeon wait a moment. If this cures your hot surgical abdomen, you've got no surgical abdomen, because it will very clearly and very rapidly clear that belly like no surgical treatment yet invented.

Very simple, does not require laboratory; all it requires is a bit of cerebrum. Now that's the only reason I bring this up. It is a very simple maneuver; it's a very easy maneuver. Most hospitals have some calcium gluconate, and it would save a few surgical abdomens. It would save a few hospital red faces; it would save on our spiraling hospital costs; and it would save the man an unnecessary tour in the hospital. This is a turkey. This is an old story. Everybody knows about lead exposure. If you will just ask about the possibility of the work exposure, you can really be a hero to yourself and to your own professional conscience, too, by this very simple maneuver.

Textbooks mention it, but they don't give much of the details; 20 percent solution, 10 cc's slowly intravenously. Simple, an old turkey, but it always works if lead poisoning is what the patient has. Of course, if you've got an acute abdomen, you're not going to relieve it, but you're not going to harm the patient either. So you've got nothing to lose by this maneuver, except if you get it outside the vein. If you get it outside the vein, you've got something to lose. The patient is really very unhappy about it, and he darn well should be. Now, that's enough of the old turkeys...

MISSED AND MIS-DIAGNOSES IN CHEMICAL EXPOSURES

OCCUPATIONAL DERMATOSES
Donald J. Birmingham, M.D.

After the very nice watch and see and understand descriptions already presented, you may find the melange of disease I shall present a bit bewildering. Your attendance at a meeting like this is evidence of your trying to unravel the bewilderment, I'll try to help you make skin disease look like something we can really diagnose. You are obviously aware of the tremendous spectrum of diseases that the skin is able to conjure up for us.

While many solely dermatologic lesions result from contact with physical, biological, and chemical sources, numerous other skin diseases of similar appearance may be the expression of an internal disease, a drug eruption, or indeed a systemic reaction to an environmental toxicant. Some of these things were described earlier today, such as Dr. Dinman's reference to the cherry red color of the skin. That was a good indicator of something gone wrong resulting from an inhalation phenomenon. The dermatologic signs attending such systemic reaction may not be as profound as those associated with the central nervous system, the gastrointestinal system, the pulmonary tree, kidneys, or liver. An understanding of the interplay of the skin in certain of these systemic affections, including environmental intoxications, can be exceedingly useful to you in your diagnostic capability. If one is unfamiliar with these possibilities, the lowered index of suspicion obviously leads to missed or mis-diagnoses.

Now, admittedly, it is not a simple task for the non-dermatologist to readily recognize and catalog a skin disease as local or systemic, as occupational or non-occupational. Indeed, the well trained dermatologist himself has his bad days in his office as well as in court, and sometimes he never reaches resolution for these cases. The most reliable way to approach the diagnosis of a dermatologic condition is to apply the same simple tenets of diagnosis you learned many years ago. May I just take a moment to remind you of them. I think they are pretty obvious things; but most of the time, in our haste to get a case in or out of our offices, or in or out of an emergency room, maybe it is we who are at fault. How often do we really take a good history on our cases? How far do we really go into finding out what kind of a lesion we are looking at? What's the distribution of the disease? What has been the clinical course? What will be the clinical course as we prognosticate? How do we interpret our clinical tests?

These diagnostic categories are just as important to the dermatologist as they are to the cardiologist, the pneumonologist, and the renologist. Our bench marks in the study of occupational diseases are the reasonably recent revisions of the basic clinical types of occupational skin disease. (slide presented) Obviously, the skin has so many cantankerous ways of behaving that you couldn't possibly have an orderly classification confined to seven different orders of disease. Nonetheless, these seven are the ones you would encounter most often if you were to study 1,000 cases, or indeed even 100.

First, is acute eczematous contact dermatitis which may be a fooler. I think all of you will agree this is an acute contact dermatitis. (slide presented) The young man presented himself at the emergency room of our hospital some months ago. He was sent to us from his company. They gave up on him and thought he needed more acute care than they were able to render in a very small plant. Everything here hinged on his history. He worked with chromates in a small bumper and auto parts plant where the parts were chrome plated. Obviously, the manager thought that this was the major cause of this man's disorder. The actual cause of this fellow's disease had nothing to do with his exposures at work, but it had everything to do with his exposures in a moonlight job where he was employed as a paint stripper. This actually proved to be the major problem. We see here an acute eczematous disease with all the cardinal signs of inflammation. I think none of us needs to be a first class dermatologist to make this diagnosis. It's in a classical site of involvement, the face. Notice the eyelids, with their edema, acute redness, swelling, pain and in this instance, certainly impairment of function of his normal cutaneous physiology.

On the other hand, look at this young lady (slide presented) also presented as a potential occuaptional case. She has redness of the face. She has a very strange type of spurring circum-orally, generally, not totally. The most interesting part in this photograph are her fingers. I think you can see them here as portraying a rather good, deep red color. You can see punctate hemorrhages, and also under her fingernails you have some splinters. This young girl did not have an occupational disease, but somebody had to think of one of the connective tissue disor-ders. Here we have an acute systemic lupus erythematosis allegedly, but not actually, due to her work. Although they honestly think that their rash and their job are almost invariably connected, most patients can be reasoned with; and they do try to understand a rational approach to the disease if you explain it to them in detail.

More recently, we have encountered a very strange and frightening phenomenon, toxic epidermal necrolysis. It has been referred to by the original describer, Dr. Lyle of Scotland, as the "scalded skin syndrome." Heretofore, we used to think this disease was confined to little chil-dren, a pediatric problem, where the cases were almost always due to a staphylogenous infection, a phage affair, phage to strain 71. With adults, however, this is not the case; more often than not, the adult has been taking a drug, notably butazolidin, sometimes penicillin, or sometimes a sulfonamide. Within the past 2 months, the Archives of Dermatology reported four such cases in one family from Florida. The house had been debugged with a mixture of acrylonitrile and chlorinated hydrocarbon. Two of the four patients died. This is a very frightening disease that can reach its climax within 48 to 72 hours, and the patients go into extremis and death. It has to be treated vigorously with substantial amounts of steroids. If you are certain that you are dealing with a non-infectious type, you can go forward without too much of a problem. In chil-dren, obviously, you have to avoid the steroids because of their propensity to enliven the existing staphylococcal infection.

We are still talking about acute eczematous contact dermatitis. Obviously this a severe form, but I remind you that contact dermatitis is confined to the epidermal layers; so is this disease. It really is a stripping off of the epidermal layer. You can see why it has been aptly des-cribed as the "scalded skin syndrome."

Another vexing problem to all of us is the person who claims he has never had sweating of the hands and vesiculation of the fingers until he went to work. What do we do with them? We usually say this is a non-occupational disease. We don't know what causes dyshidrosis except we know that it seems to be made worse by certain things: stress situations, chemical exposures at work, etc. But my only answer to this is that the only people who have dyshidrosis are dys-hidrotic. It sounds a bit facetious, but it's not unlike the situation with psoriatics. Here is the classical palm of the dyshidrotic patient (slide presented) tense, with very painful and discomforting vesicles. How do we get rid of them? Obviously, the patient has to remain away from work for perhaps a week to 10 days, sometimes longer. He will make rapid recovery on steroids. This is an easy way out; sometimes it is the most expeditious and genuinely sensible way, but he will get well without heavy dosage of steroids. The only thing is the disease will come back to haunt him and ourselves, as well.

The chronic eczematous contact dermatitis in this slide is the psoriatic. (slide presented) "I never had this disease until I came to work at this plant" is an age-old axiom. He is unhappy with his disease. He has to do manual work; he has lots of fissuring and cracking, and obviously he has a real impairment of his integument. Again, is psoriasis an occupational disease? Heavens, no. Is it made worse by occupations? Yes depending on what the man does. Office work, no. Blue collar workers with manual tasks may indeed have aggravated psoriasis. I think in our normal sense of compensation today, a patient is compensated for the aggravation of pre-existing disease.

In this particular case, we have to eliminate the possibility of a mycotic disease, one of the trichophytin infections. Most of the time a good clinician can recognize this as psoriasis. May I remind you once again, we do have a very good technique in dermatology that's available to all of us; it's the biopsy. We have good histopathologists who do dermal pathology exclusively, and they are quite capable of interpreting the presence or absence of a disease of this nature.

Here is something to remind all of you, you recognize this as an ichthyosis. (slide presented). But, when ichthyosis appears in later life, you'd better become suspicious that something is happening inside. Not uncommonly, this is the forerunner of an indwelling neoplasia. It's a good thing to remember that the sudden appearance of this disorder should telegraph to you to look at the gastrointestinal tract or perhaps the hematopoietic system.

Follicular and acneiform dermatoses--maybe 15 percent of the cases at the most. Fiber glass causes these, also cutting fluids and dirt. More graphically illustrated here is the type of folliculitis that you might see today among our youngsters who like to wear tight pants. They get frictional abatus from the tight-fitting denims or other types of clothing. The hairs break off and you have an indwelling infection, a very mild impetiginous dermatosis which is really a folliculitis. In the working man's mind, this would automatically become a follicular disease due to his work. Sometimes it is if he works with cutting fluids of the insoluble type.

Most of us at some time in our life develop acne. Acne can be an occupational disease. One of the worst causes of acne, of course, is a group of chlorinated hydrocarbons. Several of the solid chlorbenzols, chlornaphthalenes, diphenyls, and triphenyls are notorious producers of chloracne. You've heard a reference today to carbon tetrachloride. Carbon tetrachloride is not an acnegenic agent. Chloracne is not only a serious disease from the standpoint of causing disfigurement but also it is serious because it is hepatotoxic. And, now we know that it can be accompanied by a very interesting disorder called "porphyria cutanea tarda." This is severe chloracne; here is an even worse case, a high price for this gentleman to pay for having to make a living. (slide presented). He was engaged in the manufacture of 2,4,5-T. This process started out with the chlorination of benzol.

We now get into the pigmentary disturbances. There is an overlap between this and the chloracnogenic response. For example, excessive pigmentation accompanies chloracne. We have here a gross destruction of the sebaceous glands, which leads to unquestionable scarring of a permanent nature.

Now a little bit about pigmentations. Case for diagnosis--we have a fellow with purple lips, purple ears, and purple fingernails. Obviously, he works with something that interferes with his hemoglobin. This something happens to be aniline. This is a nice graphic description of "look and see something." We all look at things, but how often do we really see them? It pays well to look and see. We're teaching our students to do this all the time; and I guess our problem is that we look a lot but do not see.

Pigmentation of the nails again (slide presented) but it has nothing to do with the girl's work. She thought it did because she worked with a spirit duplicating fluid. She was taking Declomycin, and this is a photo-sensitivity phenomenon associated with Declomycin and sunlight.

Sometimes pigmentation can be minus instead of plus. You may remember that many years ago we thought the only chemical capable of inhibiting the tyrosine-tyrosinase reaction, which leads to the reduction of melanin, was monobenzyl ether of hydroquinone. Now we know several related phenolics--tertiary butylcatechol and other complex phenolics--that can indeed bring about this interruption in the enzymatic reaction which produces melanin. Sometimes the aftermath of an acute

dermatosis happens to be a temporary inhibition of melanization. It's not a permanent affair. It's a frightening affair to the patient, particularly if he is black; but this will usually go away in 3 to 5 months.

This example is a more profound form of depigmentation. (slide presented) The lady has something else. She did not allege that it was due to her work, but often scleroderma is associated with one's work. If the patient has worked in a cold environment, we may be hard pressed to really say what we think. We think that cold or lower temperature may aggravate scleroderma, as with Raynaud's disease, but it is not the a priori cause of the disease.

Here is a connective tissue disorder somewhat related to rheumatoid arthritis and perhaps to lupus erythematosus dermatomyositis. (slide presented) We don't know the cause. We call them all autoimmune diseases in our normal parlance. I think it is a bad term. Immunity to me means protection. What is the protection conferred to a person when he has scleroderma or lupus erythematosus?

This lady is losing her pigment. (slide presented) This is inexorable with her because of the packing that is going on in her cellular layer of skin. Her connective tissue is becoming very tight. Finally, there may be no vascular supply, sometimes producing ulcerations. As you know, this is a progressive systemic disease involving the esophagus, the gastrointestinal tract in general, and the pulmonary system.

Here is one of the fascinating diseases that can be missed or mis-diagnosed. This man has porphyria cutanea tarda. Look at the resplendent development of hair in areas in which he shouldn't have hair and the ashen color of his skin. Chlorinated hydrocarbons allegedly induced this in him and perhaps eight or nine other workmen.

More pigmentation, people taking the "pill" may exhibit this. (slide presented) If they have a job, they'll almost always associate the disorder with their work. Working with certain chemical compounds can induce porphyria cutanea tarda, particularly in the ethanolicprone individual. Try not to fail to associate the two.

An interesting problem is shown in this example of facial flush. (slide presented) You're probably aware of the serendipitous affair that led to the discovery of Antabuse: one of the physicians in a rubber company noticed that certain compounders who worked with tetraethyl thiuram disulfide had this terrible flush, and sometimes urticaria and the feeling of great weakness, after they took a few beers or a few shots of whisky. There are other things that will do this—for instance, butyraldoxime, which is put in inks to prevent skinning of the inks, and recently reported is perchlorethylene. Other chlorinated hydrocarbons also will produce the effect, but whether the chlorinated hydrocarbon or some additive is responsible, I don't know. Nevertheless, there is some gross enzymic interference with the degradation of aldehydes which causes these people to respond in this very strange way.

This is another photo-sensitivity phenomenon that almost always involves occupational exposure. (slide presented) Notice the "V" of the neck, the face, and the forearms. (This person worked outside.) This is one of our newer diseases that is part of our modern way of life, our social stance today. We all have been sold by Madison Avenue and others that soaps have to contain chemicals which keep us from smelling. Some of them are chlorinated compounds—the salicylanilids, or relatives thereof—and these are photoreactive materials.

Neoplasms, this man has a photosensitivity dermatosis that was originally diagnosed as a lymphoma. (slide presented) Next, a tumor called a keratoacanthoma. (slide presented) There are allegations that this is occupational. I have yet to be convinced that it is, but I'm willing to be convinced. I will not be, as Dr. Schwartz used to say, "that man convinced against his will is of the same opinion still." I'm willing to accept the allegation when there is sensible proof.

The basal cell epithelioma should be recognizable to all of us. In Texas, I do believe there is no such thing as an occupational cancer; it would be deadening to consider. But here is an individual allegedly with an occupational dermatosis: multiple basal cell epitheliomas from head to foot. (slide presented) He thought this was due to having worked as a radar repairman at a

communication center during the war at Ft. Monmouth, New Jersey. His radiologist and surgical friend who took care of him went along with this allegation. The man actually has an inherent genetic aberration. He has the basal cell nevoid syndrome; he'd develop these neoplasms no matter what he did. He also had mandibular cysts, bifid ribs, and other genetic aberrations.

Another age-old thing is beginning to rear its head again: Is arsenic a true carcinogen to many organs? We know that it is a carcinogen to skin. These views show typical carcinogenic squamous cell carcinomas that we see in the individual who has ingested arsenic. (slide presented) I'm not al all sure that the inhalation thereof has led to this.

This case is more easily recognized as coming from one of a few things--arsenic trioxide, zinc, chromium, etc.

Some years ago, I had a most fascinating experience, I saw a group of adults and children who worked in a Nevada smelter. The typical lesion of the children included interdigital ulcerations of the webs of the toes, etc. The reason for this was that the playground on which they played had something like 12 percent arsenic trioxide in the soil.

Sometimes people tell you they have had a cement burn. Cement burns occur when people kneel in wet cement; otherwise, they don't get it. When they kneel in the cement for a long period of time, they truly get third-degree burns.

Finally, this case is the one of the sclerodermic ulcerations that obviously would result in severe debilitation.

And then we have the granulomas, caused by silica, for example. There are any number of substances that cause granulomas. This isn't a serious disease for the individual, but it is vexing if we don't know what causes the lump or whether it is malignant. All we need to do is take it out and use a polarizing microscope to identify whether we have a silica granuloma.

What do you think caused this granuloma? It's beryllium, looking like a rheumatoid nut. (slide presented)

Or this (slide presented), the more modern problem in granulomatous disease, where we have these new tuberculosis or microbacterium marinum, among several others, that don't obey the normal culture behavioral patterns of the tubercle bacillus, and we have to have all kinds of tricks to grow them out.

At any rate, I think this takes us through the little "derm show," and you can see here that there is a lot for you to think about. It's easier to think about it if you have some kind of an orderly classification. Just as we taught you as students, we try to teach the students today. We want you to know what a macule, a papule, and a vesicle are. Some people call it garbage, but it isn't. These are bench marks. We learn bench marks in cardiology and pneumonology. We also learn them in dermatology. These are the things that enable us to make diagnoses.

MISSED AND MIS-DIAGNOSES IN CHEMICAL EXPOSURES

NOISE, HEARING, AND AUDIOMETRY
Richard A. Nelson, M.D.

Noise induced hearing loss has been recognized as an occupational hazard in certain trades for several hundred years. Little was done to prevent this injury before the past quarter of a century. We now have Federal regulations requiring industries to initiate active programs to conserve the hearing of all employees exposed to hazardous noise. The Occupational Noise Exposure Standard recommended by the Occupational Safety and Health Administration Standards Advisory Committee on Noise will probably become law soon. It will give specific guidance in the areas of hearing conservation which most concern the plant physician.

There are five elements basic to a hearing conservation program. These are (1) noise measurements and analyses, (2) audiometry; (3) personal protective measures; (4) education; and (5) engineering control. For the purposes of this discussion, we will concentrate on the aspects of hearing conservation which most affect the practicing physician.

AUDIOMETRIC EQUIPMENT

The needs for conducting screening audiometric examinations are minimal--an air-conduction audiometer and a sound proofed room for testing. The expense one may incur in obtaining the equipment is variable and not necessarily related to the quality of the results.

I am of the opinion that any physician responsible for conducting screening audiometry on industrial workers, either in the plant or in his private office, must insure that his equipment meets all the requirements of applicable regulations. To do less than this is wasting the physician's time and the plant's money.

The proposed OSHA Standard would require that the audiometer meet ANSI S3.6 (1969) standard for either manual or self-recording audiometers. The test frequencies must include 500, 1000, 2000, 3000, 4000, and 6000 Hertz. The tracing of the self recording audiometer must cross a horizontal line at least six times for each frequency.

The Standard would require monthly biological calibration of the audiometer plus annual periodic calibration and exhaustive calibration every 5 years. Monthly biological calibrations are inadequate for most operations and should be performed at least weekly, if not daily.

The proposed Standard would allow the use of either manual or self-recording audiometers. I believe that in most situations the manual audiometer is the better choice. An experienced audiometric technician can complete a routine examination in less time manually than required for self recording and can recognize an invalid examination sooner and re-instruct the testee. Also, the initial and maintenance cost differences between the two types of machines are significant. A manual air-conduction audiometer meeting the ANSI standards can be purchased for approximately $375. The maintenance costs for a manual audiometer will average considerably less than $100 per year. An acceptable self-recording audiometer will cost from $1,500 to $2,500. Annual maintenance will cost $150 to $200, and the cards for self-recording audiometers cost up to $31 per 1000.

The proposed OSHA Standard lists maximum allowable sound pressure levels for audiometer rooms. These criteria would be difficult to meet without the use of a specially prepared room or booth. Single wall mini-booths can be purchased for approximately $1,000. The attenuation characteristics of most of the commercially available mini-booths are such that they would be satisfactory for use in most medical facilities. Large, double walled booths are considerably more expensive.

Audiometric booths need to be tested initially to assure compliance with the Standard and then retested every time a change is made in the booth or booth environment. Booths should be routinely tested biennially.

TRAINING OF MEDICAL PERSONNEL DOING AUDIOMETRY

Industrial audiometry should be performed only by appropriately trained individuals. To do less will result in legally invalid audiograms. Most plants must depend upon their physician consultants or plant physicians to identify and provide legally and medically satisfactory audiometric programs. Therefore, it behooves us as physicians to know what is required to satisfy state and Federal regulations and provide such a service.

The proposed OSHA Standard specifies that audiometric tests shall be administered by a physician, an audiologist, or a certified audiometric technician. The same proposal defines a certified audiometric technician. This requires documentation of satisfactory completion of training meeting the minimum standards specified by the Intersociety Committee on Audiometric Technician Training or certification by the Council for Accreditation of Occupational Hearing Technicians. I am of the opinion that any physician, other than an otologist, who administers audiometric examinations on industrial workers should also be a certified audiometric technician

Satisfactory audiometric training programs are in existence in most areas of the country. These programs may be provided by nursing associations, continuing medical education centers, speech and hearing clinics, or private groups. The fees are variable.

RECORDKEEPING

Good recordkeeping in an industrial audiometric program is essential for good medical practice as well as for the protection of both the industrial worker and the plant.

A serial audiometric testing sheet which can be maintained in the individual's health record is the preferred means of recording industrial audiometric findings. This form should contain a questionnaire about past and present otological conditions, noise exposure, and use of hearing

protectors. The serial audiogram portion of the form should allow for eight or more sets of test results with spaces for date, time, audiometer identification, calibration standard, signature of the tester plus numerical results of testing at each frequency in each ear and a single frequency retest for each ear. If a self recording audiometer is used, the individual cards should be maintained as well as a serial form. These records should be maintained for 5 years past termination of employment (this time may vary between states).

Records of calibration of equipment must be retained for legal purposes. The biological, periodic and exhaustive calibrations of the audiometer must be carefully recorded. The exhaustive calibration should cover the specifications of American National Standard Specifications for Audiometers. A "bench certificate" listing all of the calibration tests and results should be kept on file.

Hearing test rooms should be tested after installation to assure that they provide the attenuation required by applicable regulations. The room should be retested every 2 years and whenever a local change is made which might effect the ambient noise level or the attenuation characteristics of the test room. The results of all attenuation evaluations of the test room should be recorded and filed.

PERSONAL HEARING PROTECTIVE DEVICES

The selecting and fitting of hearing protectors frequently presents a dilemma for medical personnel charged with this responsibility. One must be concerned about cost, comfort, attenuation, durability, ease of fit, etc. Identifying the best protection for the individual worker becomes a compromise between the aforementioned factors and the needs of the worker.

Personal hearing portective devices basically fall into three categories: earplugs, ear muffs, and ear canal caps.

Earplugs are the most commonly used hearing protective devices. The price of earplugs may vary from a few cents for common-size plugs to approximately $6 for a set of personalized molded earplugs. The standard V-51r earplug is available in five sizes, gives good attenuation, and is one of the very least expensive. This plug is satisfactory for most general usage. Even so, a variety of hearing protectors should be stocked to give the individual worker a choice of protection. The standard saying, "the best hearing protective device is the one that is worn," applies.

Earplugs are best fitted by a person who can examine the canal with an otoscope and recognize common abnormalities. Devices are available for insertion into the ear canal to determine the appropriate size of plug needed. At the time of fitting the individual with hearing protective devices, it is important to instruct him in the proper placement, use, cleaning, storing and replacement of the devices.

Ear muffs are somewhat more expensive than standard plugs but generally provide better attenuation and comfort. Individual workers who cannot readily be fitted or for medical reasons should not be fitted with earplugs, and those who cannot find comfortable earplugs, may satisfactorily wear ear muffs.

Ear canal caps are most useful for persons spending only short periods of time in noise hazardous areas. These devices generally become uncomfortable with continuous use.

The attenuation value of the hearing protective device used, as well as the ambient noise exposure of the individual worker, must be known to the person responsible for providing the protection. Some work situations may require the use of double protection to give a margin of safety.

AUDIOGRAM INTERPRETATION AND OTOLOGICAL REFERRALS

The proposed OSHA Standard gives guidelines on screening interpretation of employees' audiograms.

The proposal would require that each worker's annual audiogram be examined to determine if significant threshold change has occurred in either ear. A greater than 10 dB average decrease in hearing acuity at 2000, 3000, and 4000 Hertz will be considered significant change relative to the baseline audiogram. The employee found to have such a shift in hearing acuity shall be retested within 1 month. If the shift persists, the employee shall be referred for appropriate medical evaluation, according to the proposal. Also, the employee must be told what his hearing level is and be provided a copy of the audiogram.

The physician evaluating screening audiograms for industry must be aware of the gross audiometric findings typical of noise induced hearing loss as well as the findings associated with conductive hearing loss, tumor of the auditory nerve, infection, and even malingering. These interpretive findings can best be found in a text such as those listed at the end of this paper.

One must be very selective in choosing an otologic consultant. Many practicing otolaryngologists are not well versed in the area of noise-induced hearing loss or in the laws affecting occupationally induced hearing loss. I believe that the plant physician should question the prospective consultant thoroughly before referring industrial workers. When referring workers for evaluation, one must provide the consultant with all available pertinent information such as noise exposure history, all audiograms, head injury history, etc. The more information the consultant has available, the better service he can provide for the employees and the industry.

SUMMARY

Our goal in industrial hearing conservation must be the prevention of any loss of hearing due to the individual's occupation and the early recognition and appropriate referral for hearing loss due to any other cause.

The Occupational Safety and Health Administration's noise standard will soon be published and become law. This document will provide requirements for personnel training, equipment, record-keeping, and personal protection. It is important for the person responsible for administering the medical aspects of a hearing conservation program to realize that the contents of this standard will constitute minimum requirements and not goals to attempt to achieve.

The preservation of industrial workers' hearing requires adequate and appropriate medical participation. Without this, the entire program is in jeopardy.

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INDUSTRIAL HYGIENE DEMONSTRATION OF ENVIRONMENTAL MEASUREMENT TECHNIQUES

MONITORING THE OCCUPATIONAL ENVIRONMENT, GENERAL CONCEPTS
Jerome T. Siedlecki, M. S.

Jerome T. Siedlecki, M.S.: I will start off this session by talking about general concepts of Monitoring the occupational environment. In occupational health surveillance, four monitoring systems are used for the prevention of occupational disease. These are personal, environmental or area, biological, and medical.

Personal monitoring is the measurement of doses of airborne contamination to workers. In personal dosimetry, the measurement device is placed as close to the portal of entry of the contaminant into the human body as possible. In the case of a substance which is toxic by inhalation, the measurement device is placed close to the breathing zone. In the case of noise, the device is placed close to the ear.

Environmental or area monitoring is the measurement of a contaminant in a workroom. The measurement device is placed adjacent to the worker's normal work station. The effect on the worker is calculated or estimated from the measurement.

Biological monitoring is the measurement of changes in composition of body fluid, tissues, or expired air in order to detect absorption of a contaminant into the body of the workman. Examples are the measurement of lead in blood or in urine to determine excessive lead absorption or the determination of phenol in urine for excessive benzene absorption.

Medical monitoring is the measurement, by medical personnel through clinical means, of the human response to a contaminant.

Biological and medical monitoring provide information only after the fact of absorption of contaminant. Personal and environmental monitoring provide information necessary for design of effective environmental controls such as engineering controls and proper work practices to prevent exposure to excessive concentration of the contaminant and hence absorption of the contaminant into the body. Such monitoring must be continued to determine adequacy of engineering controls and maintenance procedures.

Each of the four monitoring systems may be necessary to evaluate adequately the effect of the work environment on the worker. Human biological responses are non-specific in nature and both medical and environmental quantification is necessary to determine whether a casual relationship exists between the worker's condition and the workplace. For example, a

physician may suspect that a worker's anemia has resulted from his exposure to benzene. Only when personal and environmental monitoring provides information that sufficient benzene was present in the environment, can an evaluation be made of any casual relationship.

When significant absorption of the contaminant occurs through the skin, personal and environmental monitoring cannot provide sufficient information in a surveillance program. Medical and biological monitoring are necessary to assist in adequate evaluation of the exposure.

Epidemiologic studies using the four monitoring systems develop information on the health effects of exposure to a potentially hazardous substance. From such studies, permissible limits of exposure can be established.

Personal monitoring is necessary to establish a baseline of exposure to determine the need for biologic monitoring and medical examinations. For example, the recently released NIOSH criteria document on benzene defines an "action level" of exposure as one-half of the recommended environmental standard. If this limit is exceeded, laboratory examinations must be conducted at 3 months intervals and more frequently if the results are judged to warrant more observation.

Accordingly, there must be on-going relationship between the two disciplines, medicine and industrial hygiene, to develop appropriate engineering and medical controls for the prevention of exposure to a potentially hazardous substance.

Our discussions today on environmental measurement techniques will be limited to personal and environmental monitoring, the measurement devices and air sampling methods and the evaluation of the data from the monitoring procedure.

RECOGNITION OF HAZARD

An environmental investigation must define the exposure of a worker to a toxic substance, biological agent, or physical energy. The concentration and the movement of the worker with respect to time and location are not constant but are highly variable. Air currents in a room, variations in process, changes in work practices and variation in emission rate of contaminant are some of the factors which result in continual change in concentrations of the contaminant. The five questions--What? Who? Where? When? and How?--must be answered before any sampling or measurement can be initiated.

What is the potentially hazardous material? Is it a chemical, biological or physical agent? What is the physical state of the chemical? Is it a solid, liquid, gas, vapor, mist, dust, or fume? If the potential hazard is a physical agent, is it electromagnetic or ionizing radiation, noise, extremes of temperature, humidity or pressure? The characteristics of the hazardous substance must be known, but the concepts of defining exposure are similar whether it be a chemical or biological agent or physical source of energy. For simplification, I will limit the discussion to toxic chemical agents.

Who is the worker being exposed? The worker at the source of exposure should definitely be studied, but workers at nearby operations and maintenance workers in or in the general vicinity of operations may also be subject to exposure to the contaminant.

Where is he exposed? The worker may be exposed at his "normal" work station where he most frequently works or only in areas where leakage may occur or at abnormal or infrequent process operations.

When is he exposed--the cycle of the operation--over what period of time, day, night, summer, winter? There may be a period of air concentration build-up at some period during the cycle of operation or shift. Production rate may be variable during different days of the week or month. There may be variability in climate conditions such as temperature, humidity, wind speed and direction, particularly in outdoor work which can cause exposure variations.

In what manner and how long must the worker be sampled to obtain a representative evaluation of his exposure?

SELECTION OF MEASUREMENT PROCEDURE

The selection of the air sampling method or measurement device depends on the physical and chemical characteristics of the air contaminant. Such factors must be considered as the particulate sizes involved, density, solubility, vapor pressure, dew or sublimation point, freezing point, chemical sensitivity, radiant energy absorption, thermal conductivity, and ionization potential in solutions. Other considerations are whether the presence of other substances interfere either with the collection of the contaminant under investigation or with the reliability of the analytical method. It may be necessary to collect simultaneous samples by different methods to determine the concentration of the contaminants. Other factors to be considered in choice of a particular measurement device are (1) portability and ease of use, (2) efficiency, (3) sensitivity, (4) reliability under various conditions of field use, (5) availability, and (6) the information desired.

In recent years, methods and instrumentation have been developed to allow full-shift sampling for many dusts, fumes, gases, and vapors. These methods include the use of filters and miniature cyclones to sample airborne dusts and activated charcoal to sample many gases and vapors. These will be fully described by the other members of this workshop.

The method of sampling and analysis should be thoroughly investigated. The method must have the desired limits of sensitivity. The size of the sample must be such that a definite result can be reported. There is a limit to the amount of asbestos fibers on a filter for accurate counting. There is a limit to the absorptive capacity of charcoal tubes used for sampling of gases or vapors.

The instrument or sampling equipment should be calibrated prior to monitoring. The apparatus may have to be checked for calibration during and after the monitoring is completed to be certain the results are valid. Where a definite volume of air is sampled, it is essential that the air sampling device operate at a known rate of air flow. The pump of the air sampling device must be calibrated against a standard air flow measuring device before and after use in the field. The exact rate of flow must be known so that when it is multiplied by the sampling time, the total volume of air sampled or collected will be known. This volume of air is used in calculating the concentration of contaminant to which the worker was exposed. Direct reading instruments such as detector tubes must be calibrated against a known concentration of the substance for which they are used. All instruments have limitations and require considerable care and maintenance.

MONITORING PROCEDURE

Monitoring is initiated after selecting the appropriate measurement or sampling device. The purpose of sampling is to determine the pattern of occupational exposure so that the average time-weighted exposure for an 8-hour work shift can be calculated. It is necessary to sample at the breathing zone of the worker, as well as to obtain samples in the general area adjacent to the operation or general room air.

Sampling should determine the duration of exposure, usually a complete cycle of operation. This may require a series of samples, that is, sampling for a period of time every hour. Full-shift integrated personal sampling is desirable if the result is to be calculated as a time-weighted average exposure. This is essential, because many of the permissible limits of the Occupational Safety and Health Administration are based on ceiling or peak limits as well as time-weighted average concentrations. Ceiling and peak limits are maximum concentrations which are allowed for any time period.

It is desirable to obtain area samples to determine whether employees located at distances from an operation should be sampled or whether the area should be isolated so that employees do not enter a contaminated area. This can readily be determined by locating samples at a fixed

position at different distances from a source of contamination.

The volume of the sample or duration of sampling depends upon the sensitivity of the analytical procedure, the permissible limit of the contaminant and an estimate of the air concentration. It must be adequate to permit a determination with reasonable accuracy. The volume of the sample may vary from a few liters when it is estimated the concentration of a contaminant is high to several cubic meters where low concentrations may be anticipated.

A blank of the sampling solution, filters, or other collecting devices carried in the field should always accompany the samples shipped to the laboratory for analyses. The purpose of the blank is to establish a baseline for the result and to determine interference from contaminants inherent in the solution, filters or collecting devices, or picked up in the field.

EVALUATION OF DATA

After the samples have been obtained and analyzed and the data from direct reading instruments assembled, the final step is evaluating the environment. Time-weighted average concentrations should be calculated and a determination made whether ceiling limits have been exceeded.

A great deal of judgment must be used in interpreting the results. The conditions under which the monitoring was conducted should be carefully evaluated. Was the day on which the samples were collected representative of all other days? Was production normal? Did the worker wear respiratory protection? Was the respiratory protection effective? Was the exhaust ventilation turned on? It may be necessary to monitor on several days under different operating and climactic conditions to evaluate adequately an exposure to a hazardous substance.

The results of the environmental study are compared to standards of the Occupational Safety and Health Administration or the Threshold Limit Values (TLV's) of the American Conference of Governmental Industrial Hygienists. Where no permissible limits are available, a judgment must be made whether exposures are excessive from available toxicological data. Results should also be compared with previous data to determine any changes in airborne concentrations and the adequacy of existing controls.

I have given a few general concepts on personal and environmental monitoring. Our next three speakers will elaborate further on these concepts when they discuss the sampling devices and instruments for the measurement of particulates, the measurement of gases and vapors and the measurement of physical agents.

INDUSTRIAL HYGIENE DEMONSTRATION OF ENVIRONMENTAL MEASUREMENT TECHNIQUES

MEASUREMENTS OF PARTICULATES
Robert Weidner, M.S.

Robert Weidner, M.S.: Before we talk about sampling equipment used to collect particulates, and I can only give a cursory view in so short a period of time, we should consider its purpose.

Particulates are classified generally into two categories. There are particles and there are fibers. The industrial hygienist's definition of a fiber is anything that has an aspect ratio--the proportion of its length to its width--of three to one. So that if a particle has an aspect ratio of three to one, it becomes by definition a fiber. Fibers of asbestos, wool, or glass generally have aspect ratios much greater than that. Sometimes the aspect ratios reach as much as 100 to 200 to 1. The size of the particles we are talking about is very important in relation to their effect on man. Why is this? When we're talking about particulate matter, we're primarily concerned with airborne particulates or airborne fibers that are harmful to the lungs.

You can walk into a plant and see a very large dust cloud. That doesn't necessarily mean that there is a serious problem. The particles that you can see with your naked eye are usually too large to get into the lungs. They are retained in the upper respiratory tract and don't create a pulmonary problem. There might also be particles in that cloud which you can't see with the naked eye and, being small, could get down into the lung and cause harm.

Therefore, we break dust down into two categories. Based on a pathological examination of lung specimens from persons who were known to have various pulmonary diseases (such as silicosis, farmer's lung, byssinosis, and so forth), we have been able to determine the size of particles and the extent of their penetration into the pulmonary system. As a rule, we have come to consider a respirable particle as that having a diameter of about 10 micrometers. Some people consider anything that is 7 micrometers or above will not be respirable and others consider a cutoff at 5 micrometers. In most cases, however, the actual shape of the particle has more effect on its aerodynamic qualities and respirability than does the actual diameter. Be that as it may, the important thing to note here is that the design of the sampling equipment must be capable of discriminating between those particle sizes which are respirable and those which are not. In effect, the sample must approximate as nearly as possible the size-selective capability of the human respiratory system so that we can determine not only what is in the plant air, but whether or not the level of particulates is safe.

In the two-stage sampler, there is a first stage that keeps out particles which are larger than about 7 micrometers, but allows the smaller particles to pass on to the second stage. The latter is generally some type of filter upon which the dust or fibers collect; the filter medium is subsequently removed at the end of the requisite sampling interval and the deposited

material then weighed and/or analyzed visually or chemically.

All right, this gives us so many milligrams of dust on that filter. Because we have properly calibrated the equipment before taking it out into the field, we know how much air we pulled through that filter. Thus, we can arrive at a mass concentration of so many milligrams of respirable dust per cubic meter of air sample. This figure may be then compared with the current standards for threshold limit values.

The standards that we're talking about are those that were initially promulgated by the American Conference of Governmental Industrial Hygienists (ACGIH); these are published in their TLV booklet (TLV means Threshold Limit Values). These TLV's have been in existence for many years; in the foreword of the booklet, you will find that when the ACGIH devised these limits as guides for the industrial hygienist in evaluating the hazards of a particular environment, they not only included dusts, but also many different chemical hazards. ACGIH specifically states in the book that these TLV's were never intended to be strict levels, above which you are going to have a toxic hazard problem and below which you will not have a problem.

But when the Occupational Safety and Health Act was written, its framers wanted enforceable regulations under the law--some numbers on which to hang their hats. They came upon this book-let of Threshold Limit Values. They thought these were really practical. So they decided to adopt the values into the regulations. So now you have the Threshold Limit Values, which were intended to be used merely as guides, but which now have the effect of law. And when you exceed a TLV for a particular substance, be it carbon monoxide, beryllium, nuisance dusts, or whatever, you are subject to being cited and fined by the Occupational Safety and Health people. I throw this in because what we are dealing with here is more an art than an exact science, especially when you are talking about particulate matter.

When you actually get out into practice and collect samples in the field, you don't work with nice, spherical shapes, because most dust particles are created as a result of chipping, grinding and the like, and the particles are irregularly shaped. However, the studies in the laboratory that went to develop the techniques and equipment are normally based on "unit-density spheres," nice, round spheres. Actually your chances of running into nice rounded spheres when you are collecting a sample in the plant are very remote, so there has to be some give and take here.

Years ago, when evaluating dusty environments, they did it by collecting samples with an impinger. The impinger is a special glass bottle with a known amount of liquid in the bottom; air containing dust is drawn through the liquid and the dust becomes trapped in the liquid. A small aliquot of that liquid is put into a counting cell, allowed to sit for a period of time until the dust settles. The specimen is examined under a 100X optical microscope (a 10X eye piece with a 10X objective); comparing the size of individual particles to a series of known diameters in the graticule, the particles are both sized and counted much as blood cells are compared. The procedure described is subject to many variations, there are differences between individuals in the way each judges the diameter of an odd-shaped particle, or one may have naturally better eyes than another person. So you have all these and other variables that enter in. Even so, after they instituted controls based on this "inexact" method of evaluation, in the Vermont granite sheds, they had no more problems with silicosis. So to me no matter how inexact the method or equipment we're using, the main criterion is, does it work? The many drawbacks in the impinger method of evaluating dust were overweighed by the fact that it did work; and it did reduce the health hazard. This is also true of other equipment that I'm going to show you.

There are now also community air pollution problems. Now plants are turning toward the industrial hygienist and telling him the environmental pollution agencies won't let us exhaust contaminated air out in the atmosphere. You now have the job of controlling the outside atmosphere also. One must put in some type of collectors. This is a sand blasting operation. (slide presented) These two workers are really working under safe conditions. It's not the most comfortable way to work, but they are in cooled, air supplied suits. Some workers object to using them, insisting that a canvas hood with a glass window in it, to keep the sand from coming up and hitting them in the face is sufficient protection. It is not. Another operation

involving dusts or fumes is welding. Here we're talking about very small particulates, welding fumes. Obviously, the welder has no local exhaust to control the output of his operations.

There are scales, giving relative sizes of some of the more familiar particles. By definition, a fume is anything from about 1/10 of a micrometer in diameter up to 1 micrometer. Dust by definition is anything from 1 to about 150 micrometers in diameter. Rain, drizzle, mist, fog each is defined by its actual size. The pollens that cause hay fever range in size from around 18 micrometers to around 75 cr 80. This is a representation of the human respiratory system, indicating the various particle size ranges for each area of deposition. (slide presented) In stage 2, we are talking about 9.2 to 30 microns. A micron is a micrometer, which is 1 meter times 10 to the minus 6. We're saying that particles from 9.2 up to 30 don't usually cause any problems. From 5.5 to 9.2 particles get down to the pharynx. 3.3 to 5.5 get down to the trachea and the primary bronchi. From 2 to 3.3 they get down to the secondary bronchi. From about 1 to 2, they get into the terminal bronchi, and from around 0.1 up to 1, we're talking about the particle sizes that get down into the alveoli. The values I have listed here do not apply to fibers because of their odd shapes.

This is an Anderson Sampler which enables you to collect samples at the various stages. (slide presented) Larger particles are first stage. The next size is stage 2. That's the sampler on the left, and on the right is the pump that pulls the air through the unit with a flow rate of 2 cubic feet per minute. The air is pulled down through tiny holes in this plate. Underneath is another plate on which the sample is collected. Each succeeding plate has smaller size holes. As the air flows through the holes, it increases in velocity as the holes get smaller. On the bottom plate you collect what's left in the air. Here is a similar smaller unit with only four stages (slide presented), and the last stage in this particular unit is a filter. This separates particles into the respirable and nonrespirable portion; it is a personal pump which can be worn by an individual, collecting the sample as he performs his job.

This sampler collects a large volume of dust in the air for detailed qualitative analysis by a chemical lab. (slide presented) It has a vacuum sweeper motor, and this particular sampler was originally developed by the air pollution people. This one is the same except that it has a cyclone. (slide presented) The cyclone enables us to collect a high volume sample, and the portion which is collected on the filter is a respirable sample. The particles which are smaller than 7 micrometers are collected on the filter itself. This unit is the piece of equipment used to evaluate beryllium (slide presented), and you may recognize it as made from a little hand vacuum sweeper. The present beryllium standard is based on a total or gross sample.

Now the standard for asbestos specifically states how you shall collect the sample; that is, be using a 37 millimeter diameter membrane filter with a pore size of 8/10 micrometers. This is the unit. (slide presented) Even with two of them on, a worker out in an asbestos plant can drive a fork lift truck while he is being sampled. They don't really interfere with the work. The only time we've had to take them off of people is where maintenance people had to crawl under equipment.

This is the personal sampler. (slide presented) The filter is a 10 millimeter nylon cyclone which gives you a respirable sample for evaluating silica dust. (This is the rig that was developed by us in NIOSH.) Here's a picture of the impinger that I talked about. (slide presented) This is the original of what they called a strip impinger. This is a new sampler that's still being evaluated. This is a vertical elutriator cotton dust sampler. One of the real problems of collecting samples for cotton dust is the fact that the lint would build up and be a perfect filter itself. After the lint built up, nothing collects on the filter. That's not a personal type of sampler, that's a pipe that you hang in the plant to collect an area sample.

I will close with a brief reminder of the range of violations. For cotton dust there is no regulation as yet. They don't consider anything as even a de minimis. A de minimis violation by definition is something for which there is a regulation on the books, but if you violated it, it isn't really hurting anybody's health, so it is a de minimis violation. "Non-serious" is anything from 1/10 to 4/10 of a milligram per cubic meter in excess of the limit. "Serious" is anything greater than 4/10 milligram per cubic meter and, there is "imminent danger"--a level

such that a worker might be in danger of dying immediately. A high concentration of carbon monoxide might kill a worker; so it has an imminent danger level. In asbestos, a sample is collected on a filter, a piece is cut out of the filter, mounted on a slide, examined microscopically at between 400 and 450X magnification and the number of fibers counted. For lead, they have two categories, "non-serious" and "serious"; non-serious being 0.2 to 0.6 and serious being anything greater than 0.6, and these are all for 8 hour periods every work shift.

INDUSTRIAL HYGIENE DEMONSTRATION OF ENVIRONMENTAL MEASUREMENT TECHNIQUES

MEASUREMENT OF GASES AND VAPORS
Julian B. Olishifski, P.E.

Julian B. Olishifski, P.E.: Most of you physicians are not going to go out and sample for toxic materials yourselves. You are primarily concerned with relating the effects of exposure of an individual with the measurements that are made, the medical monitoring of the individual, the patient's work and health history, and the findings that you obtain from your examination. You would certainly like to be able to relate these measurements to the man's actual physical condition.

The question then may be what is the purpose of the sample? Is it to see whether or not the process is under control, or is it to analyze the environment where the individual is working? If the latter, are you in compliance with OSHA regulations? If there have been complaints of airborne concentrations of gases and vapors by the men working in certain areas, measurements will enable one to judge the complaint properly; i.e., whether or not the gas is within allowable limits for maintaining good health and do the clinical symptoms relate to a known occupational disease?

Air sampling methods may identify and quantify what the man is actually exposed to, but an air sample is not necessarily a true and accurate exposure or an index of the man's exposure. This is why in the ACGIH TLV's, or the OSHA compliance list, some compounds are designated "skin." If there is a possibility for skin exposure - bear in mind that skin is an important route of entry (an example that comes to mind is the organophosphate insecticide, Parathion) the air sample alone would not be sufficient. You could be misled if you just used environmental measurements. This point is brought out quite well in Lynch's book, "Biological Monitoring for Industrial Chemical Exposure Control," a very good reference book. The book is broken down into three main types of monitoring: urine, blood and breath. There are lists and tables describing which materials are best suited for each method of analysis.

For this discussion, my remarks will be confined to air sampling. There are two broad classifications of air sampling methods. There are direct reading instruments, and there are devices which collect the sample for subsequent instrumental or chemical analysis. Direct reading devices such as the colorimetric indicators depend upon color change of a column or deposited film of chemical reagent while electronic devices provide a digital reading on the meter. There are hot-wire instruments, for example, that measure hydrocarbons, CO, and so forth; while mercury detectors depend upon ultraviolet light absorption. Gas chromatographs are used for a variety of organic compounds.

Let's talk a little about colorimetric indicators. This group can be divided into three main types. One type involves liquid reagents using impingers and air titrations. For example, if you're working with hydrocholoric acid pickling operation and you would like to know about the man's exposure to HCl mist, you could do an air titration. A solution of phenolphthalein-

containing sodium hydroxide is placed in an impinger. The acid-containing air is pumped through the liquid, and eventually we see the pink color fade; at this point, knowing the rate of through-put and sampling time, it is a simple matter to determine from the concentration of neutralized base the level of acid in air in terms of milligrams per cubic meter. A second type of indicator consists of chemically treated paper; arsine is detected this way. In the third type, the reagent and substrate are contained in a tube, otherwise known as a detector tube. Recently, there has been a lot of work to develop tubes for an array of toxicants. These have been both a boon and a hindrance to the industrial hygienist. A boon in a sense that you get a direct, on-the-spot indication of the "ball park" figure. Detector tubes properly have an error factor of ±25 percent. The tubes are usually filled with silica gel that has been impregnated with a suitable colorimetric reagent. As we draw in air plus contaminant, the contaminant reacts with the reagent to produce the color change. Depending upon the particular design, we either compare the change in color against a standards chart, or else measure the length of the stain against a chart in order to relate the parameter of length to the concentration of contaminant.

There are a number of variables, however, to keep in mind. For example, the rate of flow through the tube has to be keyed to the rate of reaction of the gas with the reagent; if the gas flow is too rapid, there will be insufficient time for complete conversion to take place, leading to a lower than possible reading. The manufacturers have tried to take this source of variability into account; they and NIOSH have tried to standarize the procedure. In addition, the detector tube units (tube and pump) are now certified by NIOSH for accuracy and reliability.

Some of the other problems with detector tubes deal with interferences. Some of the older tubes that test for carbon monoxide are particularly inadequate. Carbon monoxide, as you know, is a strong reducing agent; and if, for example, we use these tubes in an atmosphere that contains ozone or some other strong oxidant, in addition to CO, the color change due to CO, tends to be bleached out by the oxidant gas. And, the final reading will be misleading. So make certain that the tube you are going to use will not be adversely affected by other gases in the environment. The manufacturer usually supplies information in this regard with particular tubes that you purchase. One of the interfering gases for carbon monoxide is hydrogen in amount of 2500 ppm; and there are atmospheres where we do have high levels of hydrogen. Hydrogen will also cause a color change, so the tube in a sense could be considered as non-specific. And lastly, when you consider using detector tubes, do not disregard shelf-life. Look at the date, which is usually stamped on the outside of the container; and, if possible, keep the tubes stored in a cool place, preferably a refrigerator. As we all know from beginning chemistry, the rate of a chemical reaction is temperature dependent; thus the dash or trunk of an automobile, or some other hot location will hasten the degradation of the reagent and shorten the shelf-life.

Another class of instruments your safety professional will use out in the plant is the hot-wire instrument. First, make certain that the operator knows how to operate the device. And, if the batteries aren't well charged, you may not get a reading. These instruments should be calibrated periodically the same as the detector tubes or other instruments. Practically all manufacturers involved in this field furnish calibration kits (aerosol containers with rubber septums filled with known concentrations of test gases) that can be used to determine whether or not the instrument is functioning properly. Above all, the directions and precautions that accompany the instrument should be understood by the individual who is going to do the tests.

Other available portable instruments include mercury detectors which can be used to determine the level of mercury vapor. These are very useful. If you're going to use these in a chlorine plant where there are strong electromagnetic fields, be careful; these fields may interfere with the operation of the equipment.

Some of the newer pieces of equipment coming out are based on gas chromatography. In these, you inject by syringe a sample into a heated chromatographic column where selective sorption takes place as a function of the compound in question, the temperature of the column, and the adsorbent in the column. These devices are especially good for organic compounds, since most compounds will produce a distinctive pattern that will relate back to the concentration of that compound. There are various types of detectors available for incorporation in this instrument. It is

important to remember to be certain that the instrument is calibrated either before or during operation and that you know definitely what is being analyzed.

Another broad field that we should be interested in is the manner of collecting samples for subsequent laboratory analysis. This can be broken down into two main categories, static and dynamic. In the static method, we evacuate a container in the lab, send it out to the actual point of operation where the man is being exposed, open a petcock, fill the container, close the petcock, and take the sample back to the lab for analysis. If the container is large enough, and depending upon the rate of sampling, we may conceivably be able to sample for a 1 to 2 hour period. Either a rigid container or a plastic bag will do. In the dynamic method, we use an air mover at the site to draw air through some sort of collector device, such as an impinger or fritted disk bubbler. Again, there would be either a color change, or the material that we're interested in would be absorbed or collected in the liquid. The contaminant and liquid are then brought back to the lab for evaluation. One of the things to be concerned is the matter of collection efficiency. In other words, what percent of the intended sample are we collecting? This is more a problem for the chemist, but the physician certainly could ask this question before making an interpretation. In most cases, efficiency is at least 80 to 90 percent; whatever it may be, the important thing is to know what it is. It's better to have a number even if not perfect than to have no number--in a sense, this is the box in which we sometimes find ourselves.

The use of charcoal tubes for sample collection is increasingly popular. They are glass tubes about 6 millimeters in diameter and roughly 5 inches long. Depending upon the rate of air flow, we can actually use a small monitor, or a small air mover, mounted on a man's belt with a tube leading to the man's breathing zone. This would give us a good indication of the man's respirable exposure. Back in the lab, the contaminant, having been absorbed on the charcoal, is stripped off by suitable means for subsequent analysis.

We said before that it is always best to collect the sample at the man's breathing zone if we wish to have the most representative view of his respirable exposure. But what are some of the other problems involved? If we take an integrated sample, is the rate of contaminant release at a steady rate, or does it have peaks and valleys? In a typical welding operation for example, how long does it take for a man to use up a welding rod--perhaps 2.5 minutes? Thus, the concentration of fumes will rise to a peak during this 2.5 minutes and then drop off when he stops welding and begins stripping. If he uses another rod, we will see another series of peaks and valleys. To the integrated sampling device, however, we're getting an average concentration over the entire sampling period, with no hint of the extent of the peaks or valleys. The results of the analysis of the average may well be below the Threshold Limit Value, and presumably the man should not be in danger. However, the man may, nevertheless, be complaining of irritation. What you should be concerned about then is the matter of the peaks, because he is complaining about what may be as little as a 1/0 of a second surge. The surge is what he gets in his nose, causes his eyes to water, or whatever the problem may be. So there are pitfalls and drawbacks of this.

Let me finish with a discussion of continuous monitoring. With all this concern we have about vinyl chloride, we certainly would want to go to continuous monitoring. The monitor could be rack-mounted, installed in one location with sample lines going out to the points to be monitored. At least one particular application of this type of monitoring has been in areas where explosive concentrations are likely.

In order for a compliance officer to issue an alleged violation of the OSHA regulations, he must relate the exposure to an individual. He cannot just say that in a particular area of the plant the levels exceeded the standards. He must be able to say that there was at least one individual in the area who was overexposed. Preferably then, we must sample at the individual's breathing zone because this sample actually relates more to what the man is exposed to than the overall environmental air. We should also measure for peaks so that we have some idea as to what those excursions are and how often they occurred. Despite all the problems and uncertainty in our present sampling methods, they are the best that we have available to us at the present time. Perhaps in the future, more reliance will be placed on biological monitoring; this may provide improved answers, because in a sense the man himself is the one who can best tell us what his exposure is. The total dose that the man has absorbed is, after all, what determines the effects.

INDUSTRIAL HYGIENE DEMONSTRATION OF ENVIRONMENTAL MEASUREMENT TECHNIQUES

MEASUREMENT OF PHYSICAL AGENTS
C. Lyle Cheever, M.S.

My discussion will be on the measurement of some of the physical factors relative to assessment of occupational health hazards. It will include measurements of (1) ionizing radiation, (2) microwave radiation, (3) noise, and (4) heat stress. Some of the problems in obtaining accurate measurements of these factors will be pointed out.

In giving a brief presentation, there is always the tendency to oversimplify. Therefore, it should be pointed out that the state of the art isn't completely satisfactory for these measurements, and some complex problems exist. In certain cases good practical survey instrumentation is yet to be developed. There is a need for survey-type instruments to measure ultraviolet radiation and for measurement of intermediate energy neutron radiation.

<center>MEASUREMENTS OF IONIZING RADIATION</center>

The U.S. Senate Committee on Commerce hearings regarding the "Radiation Control for Health and Safety Act of 1967," brought out that there were some 200,000-plus diagnostic medical and dental X-ray machines in the U.S. and that more than half the population received an X-ray in a given year. This, plus, the growing use of ionizing radiation producing devices and isotopes in industry shows the widespread exposure to certain amounts of man-made ionizing radiation. What. of the nature of this radiation?

It may be high energy (short wavelength) electromagnetic radiation (X-rays or gamma rays) or it may be particle radiation such as alpha, beta, or neutron particles. At any rate, it is either directly or, as in the case of slow neutrons, indirectly capable of producing ionization of atoms or molecules, and this is what we measure.

There are three modes of formation of ion pairs by X-ray or gamma radiation. The incident photon imparts all of its energy in removing an electron from its orbit in the photoelectric effect. In the Compton effect, a portion of the incident photon energy is given up in removing an orbital electron. At certain energies, the photon may annihilate itself forming a positron and electron. Most of the ionization which occurs results from the released secondary charged particles.

How do we measure the degree of exposure to ionizing radiation? The absorbed dose, or the energy imparted to matter, is a function of the ionization produced.

The radiation survey instruments are designed to operate in one of the three different useful voltage regions. In the ion chamber region, the voltage gradient is sufficient to collect at the electrodes essentially all of the ions produced by the radiation while it is below the voltage threshold for production of secondary ionization. This voltage region is used for Ion Chamber instruments and is the principal region in which absorbed dose is measured.

The proportional region is used for alpha radiation measurements, and it has a voltage gradient sufficient to produce secondary ionization. The read out is in counts per minute, and it operates in the voltage region where the amount of secondary and total ionization is proportional to the ionization produced by the measured radiation. Since alpha particles produce more ionization than beta or gamma radiation, the counting threshold can be set to detect only alpha radiation.

The Geiger-Muller (GM) voltage region is the highest useful voltage region. In this region, secondary ionization saturation is produced for all incident ionizing events. The GM detector is used as a sensitive counter for beta and gamma radiation. A gas filled probe which is self-quenching is utilized and read out is generally both by meter scale indication of counts per minute and by aural signal through earphones.

Ion chamber instruments often utilize air as the counting medium. The voltage bias across the electrodes is typically supplied by four 22.5-volt batteries in series.

In a scintillation detector, the ionization produced by an incident photon results in a flash of light from a phosphor (typically sodium iodide for photons, plastics for beta radiation, and zinc sulphide for alpha radiation.) There is subsequent amplification of the signal by a photomultiplier tube; the readout is in counts per minute or sometimes disintegrations per minute is indicated for an assumed counting geometry.

In addition to measurement of X-ray or gamma ray photons and alpha and beta particles, there is a need for measuring neutron particles in certain cases such as at nuclear reactor facilities or from portable neutron sources.

Measurement of fast neutrons can be made by measuring the ionization produced by recoil protons from a hydrogenous material liner in the detector probe liner. Slow neutrons can be measured by neutron capture in boron 10 and measurement of the ionization produced by prompt alpha decay of boron 11.

What is the accuracy of these radiation measurements? That depends on the conditions and the techniques employed. There is no pat answer. Ideally, the surveys would be made under representative conditions or so as to assess the greatest hazard potential. Where scanning with the GM survey meter shows a reading of 10 percent or more of the exposure guidelines limit, then a dose rate reading should be obtained with an ion chamber instrument. The instruments should be properly selected for the particular radiation hazard, and they should be properly calibrated and maintained. The calibration accuracy is in general ± 10 to 20 percent and linearity is generally ± 10 to 15 percent of full scale. Battery checks and radioactive check sources should be used routinely to show that the instruments are functioning. What are some of the problems involved in measuring radiation hazards?

Alpha radiation may be shielded, or self-absorbed and not be measured. Since alpha particles produce a high density of ionization and have a range of only a few centimeters in air, the detector probe window must be very thin, and the probe window may be an aluminized mylar film with a density of only about 1 mg/cm (2). Damage to the windows can produce leakage of the counting gas from alpha detectors and GM probes and light leakage into scintillation detectors.

The counting geometry must be considered. Small beams of radiation will not expose the entire detector chamber volume and a multiplier should be applied to indicate the radiation intensity

of the beam. Beta particles will backscatter so that the counting geometry of a check source mounted on stainless steel with 30 percent backscatter is considerably less than that from the same source mounted on platinum producing 70 percent backscatter. Neutron measurements are difficult, especially at intermediate energies where determination of the energy spectrum is important. Stray high level RF or magnetic fields may interfere with meter measurements. Also, high humidity can cause insulation breakdown, and temperature extremes may cause battery irregularities.

The overall accuracy of measurements depends on how well the instrumentation is calibrated and maintained, and on the selection, techniques, and interpretation used in applying them. Hopefully, a correct statement of the measurement accuracy will be made. The measurement of mixed fields of radiation and of neutrons of intermediate energy are looked on as the more difficult problem areas.

MICROWAVE RADIATION MEASUREMENTS

Microwave radiation applications have been increasing rapidly, most notably by the growth of the microwave oven industry for food preparation. The frequency range of this electromagnetic radiation is given as 100 MHz to 100 GHz for the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Values. In general, the microwave ovens operate at 2,450 MHz which has a wave-length of about 12 cm. Also, some diathermy equipment operates at this frequency. These electromagnetic waves act on polar molecules such as body water and apply rapid repetitive forces to align the molecular charges with the microwave field. The heating of tissue that results is the principal effect dictating exposure limits. The ACGIH Threshold Limit Value is 10 mw/cm^2 for exposure periods of less than 6 minutes, with a maximum exposure of 25 mw/cm^2.

The microwaves may be measured by means of instruments using antenna pickups and sensors such as thermocouple for generation of current which is amplified and read out on a meter scale as mw/cm^2 power density. The microwaves may be reflected by a shield such as a screen or metal plate. Measurements should be made periodically for leakage of radiation at the door periphery and at other penetrations into a microwave oven cavity.

Microwave power meters suitable for measurement of microwave leakage in the near field, such as at oven doors, must have small antenna probes to avoid excessive interaction and changes in the microwave field. Also, with large antennas phase cancellation of microwave energy can occur in the near field. The instruments that are used to monitor leakage from microwave ovens have very small crossed dipole antennas. These instruments may be calibrated for measurement of specific frequencies such as 2,450 MHz radiation. Antenna probes can be purchased to give meter full scale readings such as 2 and 20 mw/cm^2 or 10 and 100 mw/cm^2. The meter can be sent to the manufacturer for recalibration in an anechoic chamber. A standard gain horn is used in the calibration procedure, and it is comparable to a standard gain horn utilized by the National Bureau of Standards (NBS) for generation of microwave radiation. The response accuracy of a properly calibrated meter of this type is stated as ±0.5 dB at the 2,450 MHz frequency.

MEASUREMENTS OF NOISE LEVELS

Instruments for measurement of noise levels such as the sound level meter and the noise dosimeter simply transduce the mechanical vibration energy or progated periodic oscillations of air pressure into electrical energy for approriate readout. The sound level meter may utilize a condenser type microphone where movement of a diaphragm changes electrical capacitance or a piezoelectric-type microphone where the sound pressure stresses a crystal material and thereby generates a voltage signal. By subsequent amplification and frequency weighting of the electrical signals, an appropriate readout in decibels is indicated on the meter scale.

The relative response differs at high frequencies for random, perpendicular and parallel incidence of the sound waves to the microphone. Note that for perpendicular incidence

appreciable variance from true decibel readings occurs at the higher frequencies. The meter orientation can be changed during a survey, if it is suspected as a potential error.

The A scale frequency weighting in the Sound Level Meter is used for a simplified correlation to the hearing loss hazard. At lower frequencies appreciably more sound energy can be tolerated. The ACGIH Threshold Limit Value for 8-hour exposure to noise is 85 dBA.

The tolderance limits specified by ANSI for type 2 sound level meters are ±0.5 dB over the frequency range 63 to 2,000 Hz and ±1dB over the range 22.4 to 11,200 Hz. However, the measurement techniques and interpretation may produce greater inaccuracy than the ±dB meter tolerance limits. If the noise is at random incidence in the reverberant or far field, it may be relatively simple to obain reproducible noise readings. However, in certain instances such as with direct field noise, the orientation of the observer with respect to the survey instrument can have a marked effect on the reading. An attempt should be made to avoid shielding (low reading) or reflection of the sound back to the instrument (high reading) by the observer's body. In certain high levels of noise, microphonics by air-induced fibration of the survey instrument can produce false readings. This can be checked by removing the microphone and replacing it with equivalent impedance and noting the instrument reading with the microphone removed. The sound level meter microphone responds to wind pressure, and therefore, a wind screen over the microphone and a check on wind effects on readings is required where there is appreciable air movement. A battery check and calibration of the sound level meter should be made just prior to and just subsequent to each usage. With proper calibration adjustment, the meter reading should check with ±dB at each of the calibration test frequencies. For impact noise, i.e. noise peaks occurring at intervals greater than 1 second, special measurement techniques are required. A limit of 140 dB peak impact sound pressure level has been set by ACGIH.

Noise dosimeters are now available for personal monitoring of noise exposure. One type of noise dosimeter has a small memory capsule which stores the electrical energy transduced by the microphone response. The capsule has silver plated walls and a gold plated central electrode. The current which is generated in proportion to the sound pressure levels causes transport of silver through an electrolyte to the central gold electrode. The readout unit measures the current necessary to deplate the silver from the central electrode. When the silver is deplated, the resistance increases and the readout then indicates the integrated noise exposure.

"In "An Evaluation of Personal Noise Dosimeters," by Confer et al, published in the AIHA JOURNAL it was recommended that noise dosimeters be used to pinpoint potential problem areas and to give an index of the hazard but that additional measurements need to be obtained to determine the real exposure risk. It has been found that appreciable difference in readings may result from various orientations of the wearer of dosimeters with respect to the sound source. Dosimeters should have a remote microphone which clips to the lapel since directional response effects were more pronounced with the microphone housed in the dosimeter case.

MEASUREMENT OF HEAT STRESS

The NIOSH criteria document on hot environments and the ACGIH proposed Threshold Limit Values for heat stress give information on measurement of the Wet Bulf Globe Temperature (WBFT). These measurements obviate the need for air velocity measurements which are requisite measurements in other heat stress calculations. This makes the WBGT somewhat easier to apply. The WBGT serves as a useful index of heat stress, although all of the various commonly applied indices have some shortcomings.

A Globe Thermometer, dry bulb thermometer, and wet bulb thermometer suitably arranged are needed for WBGT measurements. The dry bulb thermometer shall be shielded from radiant surfaces in the environment without restricting airflow. The wick on the wet bulb shall extend an additional bulb length up the stem and shall be kept wet with distilled water for at least 30 minutes before the reading is taken. The globe shall be a 6-inch-diameter, hollow copper sphere with the exterior painted flat black. The globe, with the thermometer bulb at its center, shall be exposed for at least 25 minutes before its reading is recorded.

The WBGT is then determined as follows:

With solar load . . . . WBGT+0.7WB+0.2GT+0.3GT
Without solar load. . . . . WBGT+0.7WB+0.3GT

The indicated ACGIH proposed Threshold Limit Value of WBGT is then taken from curves of various work-rest cycles with WBGT plotted against the rate of work (or metabolism) in BTU/hr. The movement of workers from one location to another is one of the drawbacks in obtaining representative heat exposure measurements.

CONCLUSION

The measurements of some of the physical factors relating to health hazards of the work environment have been discussed to show both their utility and shortcomings.

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NAME	PAGE
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Chief, Cardiovascular Service United Airlines Washington, D.C.	
Irving R. Tabershaw, M.D. .	V-1
President, Tabershaw/Cooper Associates Rockville, Maryland	

NAME	PAGE
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A-2