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WEDNESDAY AFTERNOON

October 28, 1953

(The members and guests of the Massachusetts Medical Society convened for luncheon and a Symposium on Occupational Diseases of the Lungs sponsored in cooperation with the Institute of Industrial Medicine of the New York University Postgraduate Medical School in the Oval Room, The Sheraton Plaza, Boston, Massachusetts, at 12 o'clock noon, Dr. Earl Chapman presiding.)

DR. EARL CHAPMAN: Members of the Symposium on Occupational Diseases of the Lungs, it is indeed a heartening pleasure to see such a splendid gathering of physicians and others interested persons from all disciplines of life come here to attend this meeting and hear what I think is one of the most unique and exceptional groups on one platform qualified to speak on this disease pattern.

You will notice the group is sponsored by the Massachusetts Medical Society in cooperation with the Institute of Industrial Medicine of the New York University Postgraduate Medical School. Now, like any other meeting of this kind, it couldn't possibly have come about without the driving force and energy of one man behind it. He happens to be the Medical Director of Johns-Mandeville Company. Of course, there they know these diseases have occurred in their people, and they

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are out to give their workmen the best possible help.

It gives me great pleasure to point out Dr. Kenneth Smith and ask him to rise.

(Applause.)

We are also indebted to another man, Dr. Frederic Hagler, our President -- right over there.

(Applause.)

We will now have the Chairman of the Committee on Industrial Health. When Dr. Smith had this idea of getting this unique gathering together, he did everything possible to push it along and bring all the forces together within the society in the sectional aids.

This person is Dr. Henry Marble -- at the end of the head table here.

(Applause.)

We are interested in the welfare of the worker, and we are interested in industrial production. I think that explains this whole gathering.

As our guest speaker we have a man who is peculiarly qualified, I believe, to be our guest speaker. He is a man who for many years has been identified with labor and industry. He is, in fact, a member of the Asbestos Workers Union in this region, so he knows the attitude of the laboring man in regard to this problem of pulmonary disease.

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For some 35 years he was Secretary of the Building and Construction Trade Council and was able to carry through his experiance and knowledge of the worker's attitude toward these problems. Now, at the moment, he is Commissioner of Labor and Industry in the Commonwealth of Massachusetts.

It is a great pleasure to bring before you the Honorable Ernest Johnson.

(Applause.)

HONORABLE ERNEST JOHNSON: Mr. Chairman, head-table guests, ladies and gentlemen, I, too, add my congratulations to you for this splendid gathering here today which demonstrates the interest that you have in occupational diseases, particularly as they relate to lung diseases.

I have the privilege of representing the Commonwealth of Massachusetts as the Commissioner of Labor and Industry as a recent appointee of the present Governor -- last February. But I have been no stranger to the department. In my 35 years as a labor representative I have been in and out of the department -- serving on legislative committees, serving on research committees -- striving to better the laws and rules and regulations as they relate to labor. It is with a great deal of pleasure that I accepted the appointment of His Excellency, the Governor, to be the Commissioner of Labor and Industry in Massachusetts.

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It is noteworthy, however, to know that Massachusetts has been a pioneer. Massachusetts was the first State in the Union -- I might cover a great deal of territory by saying "the world" -- in that our Legislature first recognized the need of a special commission devoted to investigation and recommendations for legislative action on the problems confronting the workers, and in 1866 the Legislature enacted legislation which brought into existence what was then known as a Division of Statistics which has grown in proportion and has been used as a pattern in many other States in the Union.

We have seven departments in the Department of Labor and Industry. The Department of Industrial Safety and the Department of Occupational Hygiene I have placed at the top of the list because their work is closely related to the subject which you are discussing today. In addition we have the Division of Statistics, Concilliation and Arbitration, Necessities of Life, Minimum Wage and Apprenticeship Training. We have a total of 276 employees. We have 1,500 statutory laws and regulations to administer and enforce.

The Commissioner with the advice of an assistant Commissioner, who is a woman -- and is a woman by provision of the law -- and three associate Commissioners have the authority to appoint committees on which employers and employees shall be represented to make investigations and recommend rules and

regulations for the prevention of accidents and the protection of health.

The Department of Industrial Safety, which is the largest of our seven departments was created by an Act of Legislation under authority of Chapter 350 of the Act of 1919. This division enforces the laws embodied in Chapter 159 and 15, regulations embodied in bulletins of rules and regulations adopted by the department, which deal mainly with the prevention of industrial accidents in occupational diseases in plants and the building and construction trades.

Under adminstrative direction and supervision of the Director, the division has two supervising inspectors and 43 field inspectors. This constitutes the inspectional forces of the division. They carry on regular and systematic investigations of all phases as relates to safety and health.

We have a flexibility in that unlike other States -- and I note that other States are following the Massachusetts pattern -- we do not have to go to the Legislature for the adoption of a regulation or a code in our setting up of a rule or regulation governing certain occupational hazards that may occur in industry. The Legislature permits the department to call these tri-partite boards in many instances composed of employee and employer representatives and representatives of the public to make an investigation and study and recommend

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the adoption of rules and regulations as they relate to that specific industry. That creates a flexibility in that with changing conditions it is possible for us to call together a group and change the existing code or regulation to meet the conditions that may prevail in that industry at the time without resorting to legislative enactment and the long processes involved.

Our Division of Occupational Hygiene was established by law in 1934. It pleases me to note that the first director of that division is here today. This division operates under Chapter 331 of the General Laws. Section 11 (a) of that Chapter provides that there shall be employed in the Division of Occupational Hygiene persons having special knowledge of the causes and prevention of occupational diseases.

Now, I will note that the Division of Occupational Hygiene is an investigatory body to make recommendations to the Division of Industrial Safety, the Enforcement Division. They investigate the conditions of occupation in reference to hazards to health and determine the degree of such hazard and evaluate the methods of control to assist in the preparation of rules and regulations for the prevention of occupational accidents and diseases -- Dr. Hevey Elkins being the Director and is present here today. He has 13 persons on his staff.

The bulletins now in effect relating to occupational

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hazards, particularly occupational diseases, are the rules and regulations governing the use of anthrax, benzol, carbon tetrachloride, and other substances hazardous to health.

Compressed air work, prevention of accidents in construction operations, use of the common drinking cup, and several others we how have under study, including rule s and regulations governing the prevention of accidents in the linoleum and plastics industry.

I came across a law that I never knew existed until I became Commissioner, and I find that it has not been used to any extent. That is Chapter 149, Section 11, and I quote:

"The department may require every physician treating a patient whom he believes to be suffering from any ailment or disease contracted as a result of the nature, circumstances or condition of the patient's employment to report such information relating thereto as it may require within such time as it may fix, and it may issue a list of such diseases which shall be regularly reported upon by physicians, and may add to or change such list at any time. Copies of all such reports and all statistics and data compiled therefrom shall be kept by it, and shall be furnished on request to the Department of Industrial Accidents and to the Department of Public Health."

"No such report shall be subject to summons nor

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shall its contents be made public."

That, as I said, has not been generally used.

I think we are all aware that the need of an educational programa is apparent for the prevention of all types of accidents, particularly as relating to the use of materials that may endanger the life or health of persons engaged in a particular industry. I note that the Safety Council has as its slogan: "Safety is everybody's business." It is our business, and we are an impartial agency of the State, a neutral agency between labor and industry to enforce such rules and regulations.

We know that production costs are a large factor in the operation of a business, and one of those factors is, of course, injuries and department loss of time occasioned thereby which in many cases could be prevented if proper precautions had been taken.

An ounce of prevention is worth a pound of cure is an old axiom, and it is just as true today as the day it was coined. While organized labor has spearheaded and insisted on adequate safety regulations on the statute books of the Federal Government and State Government, nevertheless, other organizations in industry have not joined in this purpose of maintaining an educational program in that respect.

I am aware of the fact that large companies are

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conscious of their responsibility in regard to these cost factors. The small operator does not have facilities available. He lacks the technical supervision and advice that the large companies have. He does not join safety counsils. He is not interested in the over-all picture. He makes no effort to learn the cost factor occasioned by recurring accidents and hazardous use of materials. He believes safety precautions are an excess cost that he does not care to carry. It is our duty, generally, to supervise, inspect and educate the operators of such plants, particularly because it is in the public interest to pay attention to the causes and effects of such conditions.

It is interesting to note that there were no attempts to establish rules or regulations in Massachusetts until after the enactment of the Workmen's Compensation Law. In other words, we were thinking of paying out dollars for the injuries that occurred in industry, but we were not thinking of the need of preventive rules and regulations enforced and administered by a State agency. We are rapidly overcoming that inertia.

The latest statistics that we have been able to gather in Massachusetts show that on an annual basis the total reported injuries were 250,175, of which 53,462 were injuries in which at least one full day was lost. There were

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193 deaths from industrial accidents, 36 produced permanent total disability, 954 partial disability, and 52,279 temporary total disability.

All of the injuries accounted for a loss of 3,636,702 working days, of which 1,158,000 were charged to fatal accidents; 216,000 days to permanent total disability; 598,003 days to permanent partial disabilities; and 1,673,699 charged to temporary total disabilities.

We think the accident rate is too high in certain of our industries in Massachusetts. The very industry that I represented for 35 years leads the list with a percentage of 16.4 of the total number of accidents.

It may interest you to learn that I was astounded to learn that the largest number of injuries ocqurred in the age group 25 to 29 in the total of 53,462. We find in that age bracket a total of 6,780 cases in which accidents of either a temperary or total disability have occurred.

I constantly endeavor to bring forward the need of an educational program. It is the business of the total group engaged in industry to bring down this terrific toll that we have in industry in lost time occasioned because of accidents in industry.

I am very happy to attend this meeting here today. As your Chairman said, I'm an asbestos worker by trade. We

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found, in the last few years particularly, we have had four deaths occasioned which were diagnosed as asbestosis, and this caused some consternation in the minds of some of the men who are now asking: Are we now engaged in an occupation which has an occupational hazard in the form of a disease called asbestosis? The old timers never heard of it. Now, it may be that many of these cases were treated in other years and diagnosed as other than asbestosis and treated as pneumonia or some other lung disease and never diagnosed as asbestosis.

A remarkable thing in connection with this is the fact that many of our Unions now have health and welfare funds, and we have the means now to gather statistics and make a compilation of the causes of sickness and death and whether or not it is related to the particular occupation in which the man is engaged.

We processed these four deaths through the Department of Industrial Safety and have called upon the Division of Occupational Hygiene to make a study and report and found that one of the cases which involved the diagnosis of asbestosis was prosecuted in the courts. The defense claim was there was no liability. The claim was that outside conditions caused the death of the man. And we refrained from doing anything until the court case has been settled. When it has been settled, we intend to move forward and find out whether

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the employment of men engaged in the application of heat and frost insulating materials -- in which asbestos plays a large part -- is hazardous. We will call upon the services of the Department of Public Health and Occupational Hygiene to assist us in that work.

So I shall be an interested observer here today in listening to the various papers that have to do with this particular subject.

Thank you.

(Applause.)

CHAIRMAN CHAPMAN: The keynote of Mr. Johnson's talk was that it is not the business of any one person but the business of the total segment of the industry. I rather like that phrase.

Our next speaker, as you can see from your program, is Chairman of the Institute of Industrial Medicine, New York University, Post-Graduate Medical School, which, if you look at the top of the program, you will see is the co-sponsoring group. He, with Mr. Kenneth Smith, was instrumental in bringing this meeting about.

To most of you he needs no introduction. Somebody whispered in my ear, "He was the father of industrial medicine," but in cases of paternity there is sometimes doubt.

(Laughter.)

I am going to call him the Dean of Industrial Medicine in America, a man outstanding in his field. It is a great pleasure to bring before you, Dr. A. J. Lanza.

(Applause.)

DR. A. J. LANZA: Mr. Chairman, Mr. Commissioner, members of the conference, on behalf of my college of Industrial Medicine may I express my appreciation of the opportunity to participate in this rather unique meeting that we have here this afternoon.

May I also express my admiration for the work Dr. Marble and his associates in the Massachusetts Medical Association have done in the turnout that we see here today.

Industrial medicine is not static. Research goes on, and clinical knowledge progresses so that all of us continue to learn more and more. That being the case, it doesn't behoove any of us to be too dogmatic. We must learn to adapt out thinking to the changes in our knowledge and, if necessary, to change our minds.

When I was a boy they used to have a saying: It was only a wooden Indian and a certain kind of fool who wouldn't change his mind."

(Laughter.)

And occasionally we have to make up our minds that we have to change our minds.

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Now, this matter of occupational diseases of the lungs is not a simple one. It is very complicated and like all chronic diseases presents a number of variables the effect of which we have not always learned how to evaluate. We have learned a good deal abbut silicosis. We have learned something about asbestosis. Those are the two main forms that cause disability and sometimes death. At the same time we must always remember that in spite of the enormous amount of research that has been done in most of the civilized countries of the world on the subject of silicosis -- almost enough to fill an ordinary library -- we still do not know what is the nature of the action of the silica particle upon pulmonary tissues. We don't know. Now do we know why it is that the silicotic is more susceptible than the normal individual to tuberculosis.

Likewise, for instance, in asbestosis, we do not know what is the nature of the action of the asbestos particle upon the pulmonary tissue. The late Dr. Gardner propounded the theory that chatrary to the action of silica the action of asbestos was largely mechanical. That may be so. On the other hand, it's only fair to state that there is a considerable body of opinion that doesn't go along with that. Consequently, the matter of diagnosis is not always too easy and calls for all of the skill that we can muster with respect to getting a careful occupational history as well as the clinical and ....

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evidence that may be available. It is incumbent upon us to be very careful and very precise in our use of terms. All too commonly people are apt to use these various terms signifying different types of pneumoconiosis as interchangeable. And that is not so. These different types of penumoconiosis like silicosis and asbestosis and siderosis are entirely different diseases; and if there is any sloppiness in our use of terms, we only confuse ourselves and makes the matter of diagnosis and the just treatment of the individual worker who may be claiming compensation very difficult.

While it is true that we have a fair knowledge of the action of the silica particles and asbestos particles, as far as the pathology is concerned,the effect upon the pulmonary tissue, we must always remember that these tests do not always exist under factery or manufacturing conditions as pure homogeneous particles because there are often dust particles among them as pointed out years ago in researches at Saranac these other dusts present along with silica, other substances, modify the action of the silica particles.

So we have to approach this whole subject with a considerable degree of humility because there is so very much concerning these diseases about which we know very little. Our only hope here lies in the progress in the type of work that you will hear described later in the afternoon by Dr. Wright

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The pathologists and physiologists are the only ones that hold the key that may unlock the door to further knowledge of what really goes on in the lungs of people who breathe the various kinds of dust that we find in our working places.

So far, our estimation of this disability consists mostly of rule-of-thumb methods, and if they can be combined with a certain amount of common sense, well, we hope than that the standards of dealing with claimants for disability may be of a moderately high order.

Again let me emphasize that there is a great deal of work to do. There is a great deal of knowledge that we do not have. You will hear much more on that from our distinguished speakers this afternoon.

With that word, I thank you.

(Applause.)

CHAIRMAN CHAPMAN: You have heard from one man who has been termed by designation, and the next man is actually a Dean. He is listed on the program, however, as being the President of the Medical College of South Carolina. The President of the College is the Dean. He, also, is a pioneer because in the middle 1920's he did the first studies in this country on asbestosis.

Now, you will see that he comes from South Carolina.

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I was down there a few years ago and learned some of my American history that there were more battles fought in the American Revolution in South Carolina than in any other State in the Union, and I submit, gentlemen, that there are more battles fought for the health of the worker in the Commonwealth of Massachusetts than in any other State.

It gives me great pleasure to bring to you Dr. Kenneth M. Lynch.

(Applause.)

DR. KENNETH M. LYNCH: Dr. Chapman, Commissioner Johnson, members of the Industrial Congress here gathered, I couldn't pass up the opportunity of this kind to give further acknowledgment to the man he called the Dean of Industrial Medicine. He wanted to call him the father of industrial medicine, and then, as we sometimes do in questions of paternity, we back away and choose another title because of the possible paternity question, which reminds me of a story that I heard yesterday at the meeting of the Medical Deans that is now going on. Dr. Chapman gave me the opportunity to repeat this. He said that a certain head of a medical school who was having a party -- one of these meetings in the category of medical people -- and he had a small daughter who was cautioned that she mustn't do too much talking about who and what her father was. So, later, as was natural, one of the guests spotted this

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little girl and said, "You certainly are a cute little girl. What is your name?" And she said, "My name is Mary Jones, and my mother is Mrs. Jones over there; but I'm not supposed to say anything about my father."

(Laughter.)

I would like to speak further of the relationships -- you might call them comparisons or differences -- between the two sections of the country, one of which I have the honor of representing at this moment that I am extremely honored that at this gathering I have the opportunity of merging my particular interest in the subject that brings us here.

While the asbestos industry may date its beginnings back into antiquity and while the condition now known as asbestosis may, therefore, have occurred a long time ago -- I once made some sort of an investigation of that matter and found that you could trace the asbestos industry back to a matter of some 2,000 years, so the condition known now as asbestosis may have also occurred in these very early times. Actually, the industry, as we know it now, is very new, and the knowledge of the health hazard which is associated with it is still newer. In fact, the term "asbestosis" was not fully acceptable for use as recently as 30 years ago, while up to about that time any thought of ill effects from the inhalation by reason of occupation of asbestos dust was likely to be placed

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in the category of silicosis.

Parenthetically, perhaps I may be permitted to express an opinion here that before we have finished with the study of the class of pulmonary disease attending exposure to dusts in industry -- as already indicated by Dr. Lanza -- the whole chapter will have to be revised and rewritten.

As you already are aware, it is within the time of experience of some of us who are still dabbling around with the study of certain unsolved problems in the subject that there has been established this distinct variety of pneumoconiosis. During the same period, and probably largely because of disclosure of the hazard, there have been improvements by the industry to protect workers from such exposure. And that is particularly emphasized by the fact of this meeting here today. So that atmospheric dust conditions in asbestos mills are greatly different from the conditions of 30 or more years ago.

Even though such improvements have been made and although our knowledge of the course of the disease has come largely from the study of workers whose employment went back into the very dusty conditions of earlier times, it should not be assumed that thereby the hazard has been eliminated. Harmful asbestosis apparently is still occurring. Only a few days ago I had the opportunity of learning of two clinical cases of

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well-advanced asbestosis whose employment in the industry and exposure to the dust did not occur back more than ten or twelve years.

Asbestosis is the product of the inhalation of asbestos dust in sufficient amount and over a sufficient period of time -- you will note the very broad qualification which I give to that -- but under working conditions -- that is "conditions" -- as they occur either now or formerly, there is no uniformity in degree of disease among the workers, even from what may appear to be similar exposure. Apparently some individuals in a group working in the same environment will be severely affected while others will be less affected, and some will escape harmful effects altogether -- and this for reasons unknown.

Furthermore, the time required for similar quantitative exposures to produce like grades of asbestosis apparently varies with individuals. Now, whether or not that is an exact statement, I do not know. It apparently varies with individuals.

Another factor of question is that while in our efforts to establish protection of the health of industrial workers we have set up certain standards concerning atmospheric dust concentrations, these are of rather general nature and not specific and may have given a false sense of security.

The very dusty atmospheres surrounding the workers

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in this as well as in certain other industries under former conditions would certainly be more harmful than the relatively clean environment to be found in a present-day careful operation -- and I mean just that: "clean" -- but we may need to establish something more than merely the counting and measuring practices now in vogue if we shall have dependable safety controls.

The particulate matter in asbestos plant dust is composed of broken mineral fibers of crystalline silicates. When inhaled, most of this material is expelled, but the quantity of fragments of lengths up to one hundred or more microns that may reach the terminal respiratory units is remarkable.

As compared with the material deposited in the lung in silicosis and in certain other conditions of occupational connection, this material apparently exerts physical rather than chemical influence. Dr. Lanza referred to Dr. Gardner's experimental work where he at first thought that it was the small particles that did it -- that were harmful -- and he later changed his mind, which is a good sign, too, and concluded or believed that it was the larger fragments that did the damage. At least, it now appears that free silica in the production of silicosis becomes at least in a crude sense chemically poisonous to the tissues in which it is deposited, and asbestos crystal fragments may traumatize living tissues by their

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physical qualities -- their sharpness. Hence the present belief that it is the larger particles that do the damage here, as contrasted with the harmful effects of the smaller particles in silicosis.

I am using something of a comparison between silicosis and asbestosis all the way through this discussion.

It is not unfitting to that conception -- and that is one of my objectives here, in so far as I can go -- that, possibly by a protective mechanism the asbestos fragment remaining in the lung becomes enveloped by foreign body giant cells and covered by a smooth coating, apparently of a colloidal nature and of tissue fluid or cell origin -- but, again, one of the unknowns of the subject. This process transforms the naked asbestos crystal into a golden or brownish object of a variety or architectural shapes, called the asbestosis body. This characteristic object is formed in the alveolus of the lung. Incidentally, I would like to give a different complication by saying that by "asbestosis body" means it came from the lung of a person who had inhaled asbestos dust. It may be found in the sputum of a subject of asbestosis, although in even advanced cases that is not necessarily so, but it may serve as a confirming diagnostic procedure.

I want to show three slides to illustrate this object.

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(Slide shown.)

This is a picture of the sheaf of asbestosis bodies as you may find them in the sputum in the concentrate of the sputum. They are not colored here, of course, because this photograph is not colored.

The next slide shows them in the alveolus of the lung, and they are shown merely for that purpose here and without any further discussion of this lung. The foreign bodies show their smooth quality. This shows the difference in their forms and size -- some of them long, and some small and round.

The next shows them in the ..... draining ....

Where they may occur they may be found in relatively small numbers. It is only the smaller sizes that have an opportunity to reach the draining lymph nodes, and it is interesting where they are deposited in these lymph nodes in smaller sizes and smaller numbers they appear to create -- as far as our observation -- to cause no fibrous tissue reaction, which may mean another point in the conception that it is the traumatic effect of l. that does the damage in the lung.

Now, if I may have the lights again.

This circumstance -- that is, the limitations of physical opportunity for entry into and transportation by the draining lymphatics -- seems to explain another characteristic

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difference between silicosis and asbestosis. The small free silica particles of silicosis are readily carried from the alveoli by phagocyts and deposited in lymphoid depots along the drainage system, thus setting up focal reaction and, finally, characteristic nodular fibrosis, whereas asbestos fragments stay mainly where they land, in the alveoli of the lungs -- certainly the bigger ones do -- particularly in the vestibular area of the lobules, so arousing a more uniformly distributed reaction and ordinarily non-nodular or diffuse fibrosis and, therefore, constituting a fundamental difference in the way in which the two diseases develop.

Just what composes the first part of the reaction on the part of lung cels to asbestos fragments deposited in lung alveoli is only abject to deduction at the present. If we assume the primary effect to be from physical injury -- that is, traumatism or pricking of the walls and linings of the alveolar linings and septa, we may, likewise, assume fluid accumulation in the alveoli of the lungs. In fact, if you cast back to your earlier times in the study of inflammation you remember that a humoral participation is a fundamental part of the reaction in the inflammatory process. This may constitute the first or acute reaction, and it happens to fit with the physical evidence of increase of lung fluid that clinicians say they find in asbestos workers before there is

any particular evidence of fibrosis, and it is also consistent with the early "ground glass" changes in roentgenographic lung fields. You will hear about that from more authoritative sources.

As the pathologic conditions progresses -- ordinarily through years of continued exposure -- and while alveolar fluid increase may continue to accompany the giant cell and colloidal envelopment of the fragments of asbestos crystals as long as fresh deposition continues, permanent change in the form of fibrosis takes over and proceeds. Just what the stimulating factor in that occurrence maybe, whether from continued traumatizing by fresh material, with or without influence by the older coated asbestosis bodies, the result is essentially the same as will occur from any relatively innocuous but irritating presence of solid foreign material located within living tissues. The occurrence and advancement of this chronic fibrosis, with perhaps final lung crippling and then circulatory obstruction, may best be told here by some illustration of other lantern slides that I will now show.

May I have the next slide, please.

(Slide shown.)

CHAIRMAN CHAPMAN: Will the owners of those heads casting the silhouettes be so kind as to move a little?

DR. LYNCH: This first slide is one of a moderate

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grade, in which you see the asbestosis bodies in alveoli and moderate alveolar wall fibrosis.

The next slide is a further advanced degree or grade of the same thing in which a larger number of alveoli are involved in deposits. You don't see the objects very well in this low power, but you do see the gross deposits.

This one is still further advanced. This was from a fatal cases of asbestosis, in which the alveoli in this territory are practically wiped out.

The remainder of it has been transformed into scar tissue that replaced whatever lung tissue was in that zone.

The next slide brings into question whether or not a nodular form of fibrosis is entirely or exactly limited to silicosis as constrasted to asbestosis. This is from a not-fatal case of asbestosis in which we encountered some nodular distribution. Fibrous changes here, localized collections of old scar tissue, not the rounded snowball nodules that are so characteristic of silicosis in the advanced stage but certainly in nodular forms -- that, too, without any knowledge that there was any free silica involved in this exposure. There may be some in the final stage between the two conditions.

The next slide. The gross picture of a fatal case. The lungs are kept in position. And you can see that they remain fixed in full distention. The pleural surface is smooth

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and clean. This was not in error. It shows some evidence of local thickening here and there, but there is obvious emphysema.

The next slide is a section of the lung -- also from an advanced case of asbestosis in which ..... shows. Sometimes you have a completely obliterated pleural sac with cartilagenous thickening, and sometimes you do not. That depends on something which is secondary, not an essential part of the disease of the lungsitself. You willnnotice a marbling appearance. It appears that way when it is fresh, and is grossly honeycombed; it's a coarse-textured lung. You don't find very large emphysemas but you find wide-spread emphysema.

Then there are two more slides which I will show very quickly illustrating the same split lung section -- I mean the same type of split lung section of differenttcases uniformly marbled rather than scarred and distorted lung that you sometimes see in silicosis but with a gross honeycombed texture, loss of elasticity, and the lung remains in fully distended state.

The next one is merely the same illustration in a different case.

Now, may I have the lights on again for a moment.

It appears that the condition here illustrated is one of irreversible nature, and I see no reason to expect that the stage of fibrosis reached may become materially reduced. Rather, after an advanced general fibrosis has been produced,

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and even though there be no further exposure, the mechanical handicap of the lungs, by fixation, emphysema, and vascular interference should tend to automatic continuance of chronic lung disease stage in that lung. However, I want to quickly qualify that by saying that there is some evidence that removal of the subject from further exposure to asbestos dust does tend to halt the disease.

Here, again, there is a difference with silicosis. With some reluctance to accept the evidence on this important point on the part of a morphologist in analyzing such a matter and with some reservation as to whether -- at least in the advanced stage -- it can be said that it may have a sound basis or, at least, that a basis for it may be set up for such a contention.

If the effect of asbestos dust is traumatic in nature and only the naked crystals are harmful, then as the fragments become enveloped in the foreign body, giant cells, and colloidal coating, they should become incapable of further harm of that sort. The disease would, therefore, tend to be halted at that point, whereas in silicosis the material already deposited apparently continues to be effective indefinitely and exert its influence, whatever the nature of that may be.

At least, from more than one occurrence in our own experience, it may be said that a person may work in asbestos

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dust sufficiently to acquire considerable lung deposits of asbestosis bodies -- which deposits may be found there many years after the employment has ceased -- but having caused no appreciable fibrosis.

Now, that to me is a very important thing from the standpoint of the practical questions involved and is very important to the individual concerned and to the industry. In other words, a shifting of occupation may have more importance in this condition than in silicosis.

There is apparently another important difference with silicosis that Dr. Lanza has mentioned, and I will not pursue that in detail -- that is the apparent reported increase of susceptibility to tuberculosis along with silicosis whereas there have been no such reports in connection with asbestosis.

I want to merely mention the matter of the occurrence of carcinoma of the lung in asbestosis. In 49 cases of asbestosis we have encountered 4 associated with carcinoma of the lung -- that is, the disease from which the patient died -- an incidence quite out of proportion in comparison with our ordinary incidence of carcinoma of the lung in the autopsy series.

There have been several similar reports from widely scattered locations in this country and elsewhere of the occurrence of carcinoma in association with advanced asbestosis in a higher ratio than was to be expected ordinarily, and I

want to show you an unreported case of ours to illustrate that question and leave it as a question strictly, but one of sufficient importance that we must pay further attention to it.

The last slide, please.

That is a lung in which you see the general state very much like those shown before, and advanced condition of asbestosis and an advanced carcinoma at the root of the lung in connection with the bronchials.

And the next slide is the microscopic characteristic of this carcinoma, typical bronchogenital.

And the next one shows the asbestosis bodies in connection with the carcinoma cells themselves. The final slide shows the same thing. It's not supposed to have any significance here whatsoever. I don't mean to imply that the presence of asbestosis bodies in ..... epiphelial cells caused them to become malignant, but merely to illustrate this occurrence and, therefore, the arousing of whatever suspicions may be justified.

Thank you very much.

(Applause.)

CHAIRMAN CHAPMAN: Now, we have heard the pathology of the disease, which is, of course, the root or mainstay of the study of the disease, and these reports date back to the

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very first in the country.

Now we are going to turn to the present-day study with the Radiological Aspects of Silicosis and Asbestosis. Our next speaker is a young man trained at John Hopkins who thought he would go up to Saranc for a short time. And the short time, he tells me, has gone on five years because he became so intrigued with the work he is doing there.

Dr. Leonard J. Bristol comes from the Trudeau Sanitorium and will speak to us on the rRadiological Aspects of Silicoais and Asbestosis.

DR. LEONARD J. BRISTOL: Dr. Chapman, Commissioner Johnson, ladies and gentlemen of the symposium, a complete radiological study of the chest should include a fluoroscopic and roentgenographic examination. Both these procedures lend aid in evaluating the naatomy of the chest, some of the physiological aspects of respiration, and the presence or absence of abnormal shadows. It is imperative that one be aware of the variations seen in the healthy chest before an attempt at accurate interpretation is undertaken. The lack of understanding of such variations, and roentgenograms of poor diagnostic quality, are frequent causes of incorrect diagnosis.

(Slide 1)

The normal lung structure is produced by the pulmonary artery and its branches, and these are visible from the

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hilum to the periphery. These branches divide and subdivide, forming finer and finer ramifications. This is a reporduction of a normal excised inflated lung, with the pulmonary artery injected with a contrast medium; namely, lipiodol. It is an excellent demonstration of how the massive contrast-filled vessels stand out sharply against the air-containing alveoli and bronchi.

(Slide 2)

This is another example of the same, showing how the normal lung structure seen on a chest X-ray is produced by the pulmonary vessels. In the conventional projection -- the postero-anterior view -- the branches of the pulmonary artery overlap each other and present a definite networklike pattern. This lung was injected with barium, and the vessels are not as sharply outlined as in the previous demonstration, but it will give us some idea of the massive numbers of blood vessels within a normal lung. In viewing a single film there is much overlapping, and unless we are aware of what these white shadows represent, when they are linear or sometimes nodular in character we might incorrectly diagnose them as indicating a lesion.

In all instances it is mandatory to evaluate the roentgen changes in relation to the clinical history, physical findings, and laboratory data. Far too often this aspect of roentgenologic diagnosis is overlooked, and the outcome may

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be most embarrassing.

Silicosis is the result of the interaction of free crystalline silica and lung tissue. Silica has a specific effect, causing proliferation of connective tissue and the formation of parenchymal silicotic nodules. Microscopically the nodules are seen to be composed of hyaline collagen fibers and are evenly distributed throughout the lungs.

The diagnosis of this condition rests primarily on two criteria:

(1) An adequate history of exposure to free crystalline silica.

(2) The roentgenologic demonstration of discrete generalized nodulation in simple silicosis, and of conglomerate areas of increased density in the complicated forms.

A useful method of classifying this disease from the roentgenological point of view is by subdividing it into three stages, according to the number and size of the nodules.

A. Simple Silicosis.

(Slide 3.)

Stage I - first degree silicosis. The noddles are barely visible, and are associated with the preservation and at times exaggeration of the linear markings.

State II - second degree silicosis. The nodules are from 2 to 3 mm. in diameter, and are of sufficient size to

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obscure the linear markings to a great extent.

Stage III - third degree silicosis. The nodules are more than 3 mm. in diameter. Small areas of coalescence may be seen.

B. Silicosis with Conglomeration.

(Slide 4.)

Probably the result of the interaction of an infectious organism, not necessarily the tubercle bacillus. It is conceivable that there is no relationship between the silicotic nodule and the infectious element.

C. Silicosis with Tuberculosis

This may occur as the result of infection superimposed upon a progressive and still active silicosis, or of such infection superimposed upon an old and already stabilized silicotic process.

Diffuse obstructive emphysema is known to be one of the complications of silicosis. It is seldom clinically manifested in cases of simple discrete nodular silicosis, but conglomerate silicosis is almost always complicated by it in some degree. I do not mean to imply that chronic pulmonary emphysema is never detected in an individual who has discrete nodular silicosis, but to raise a question as to its underlying cause when the two are associated. Is the emphysema directly related to the pulmonary deposition of quartz, or is it an entirely

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independent process?

Individuals with diffuse obstructive emphysema have a reduced M.B.C. and an increased R.A. It is felt that important knowledge concerning the actual residual air total volume ratio can be obtained from an adequate chest fluoroscopy and from films made in full inspiration and full expiration. It would appear logical that the area enclosed within the silhouette of the thoracid cage at full inspiration should bear a relationship to the total volume of air within the lung, and that the silhouette of the thoracic cage at full expiration should bear a relationship to residual air. A short time ago, Dr. George Wright made a study of 234 dases, which showed a good correlation of R.A/T.V. with area of radiologic expiration / area of radiologic inspiration, the coefficient correlation being 0.772 with a S.E. of 0.02.

(Slide 5.)

Therefore, it seems permissible to say that when diffuse obstructive emphysema is a complication of the silicotic process, a chest fluoroscopy and a chest film in full expiration will furnish information as to the presence of pulmonary functional impairment. The moderate and severe forms should not be difficult to identify, but it is not easy to be definite about the individuals with slight impairment. I know of no way in which we can determine accurately the degree of diffuse

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obstructive emphysema other than by employment of pulmonary functional studies.

(Slide 6.)

This is an iron-ore miner whose X-ray showed discrete generalized nodulation in 1938. It is classified as simple silicosis, Stage I.

(Slide 7.)

Here is the same man twelve years later. There has been no change in the degree of discrete generalized nodulation, but in the interval there has developed a slight degree of coalescence of the nodules in the right outer second interspace. In 1950, when this film was taken, he had no respiratory complaints, and was working.

(Slide 8.) Here generalized discrete nodulation is present throughout both lungs. These increased densities reflect the unit pathological lesion of silicosis - a nodule of hyaline fibrous tissue. This man is still working and has no respiratory symptoms.

(Slide 9.)

This man had Stage II simple silicosis in 1933, without symptoms. The X-ray changes are characterized by discrete generalized nodulation and definite enlargement of the lung roots.

(Slide 10.)

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This is the same man in 1950 - 17 years later. There has been no progression of the X-ray changes, and he is working without complaints.

(Slide 11.) This is another example of simple silicosis. Discrete generalized nodulation is present throughout both lungs. Both hilar regions are accentuated.

(Slides 12 through 16.)

Asbestosis is a form of pneumoconiosis resulting from prolonged inhalation of asbestos dust. It is a chronic disease, with diffuse pulmonary fibrosis which takes years to develop. As in other occupational diseases of the lung, the chest roentgenogram is the cornerstone in establishing the diagnosis. Of course, it does without saying that an adequate history of exposure to asbestos dust is also a requisite criterion.

It would be wise, before proceeding to the roentgenological classification of asbestosis, to caution about making statements as to pulmonary functional impairment - "disability" - from the X-ray. An expiration film or chest fluoroscopy is not of much help in these patients, because their lungs usually empty very well. When functional impairment is present, it is usually a problem of a "tight lung," in contradistinction to the diffuse obstructive emphysema seen in complicated forms of silicosis. Physiological study of such cases usually

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reveals a decreased M.B.C., a small T.V., and no increase in the residual air. Dr. George Wright has performed complete physiological functional studies on many men from the asbestos industry. When we correlated his findings with the X-ray, it was found that some individuals with slight roentgenological changes had definite functional impairment. This, as you will remember, was not the case in simple discrete nodular silicosis.

As in silicosis, the roentgenolocial changes in the lungs of individuals with asbestosis can be divided into varyingly severe stages of development.

Asbestosis - Stage I (slight):

There is present an extremely fine network of increased densities at both bases, radiating from the cardio-phrenic angles toward the costo-phrenic sulci. There may or may not be a superimposed granular pattern. The middle thirds of the lung fields may also be slightly affected. The portions of lung involved have a hazy appearance.

Asbestosis - Stage II (moderate):

The roentgenologic picture is more dense. There is a beginning decrease in the clarity of the cardiac silhouette. The densities extend to the periphery of the middle and lower thirds of both lung fields. These parenchymal changes are sometimes referred to as having a "ground-glass" appearance.

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The lung roots are generally accentuated.

Asbestosis - Stage III (severe):

Considerable shadowing of the middle and lower thirds of both lung fields is noted, which can extend to the upper thirds. The outlines of the cardiac silhouette are from difficult to impossible to differentiate against the densities within the lung parenchyma -- the so-called "shaggy heart."

It is not expecially important if we have some difference of opinion as to which X-ray stage of asbestosis an individual has. The important thing is to be certain of our diagnosis, and of the presence or absence of pulmonary functional impairment as the result of the pulmonary deposition of asbestos dust. This is most difficult in cases which demonstrate only a very slight departure from the usually accepted normal chest roentgenogram. These cases are classified as doubtful or uncertain. Usually stereoscopic films of good diagnostic quality are most helpful in such situations. At any rate, it would seem mandatory that these individuals be closely followed by means of serial chest X-rays. Fortunately, this problem does not confront us too often.

Now I should like to demonstrate by lantern slides some various manifestations of pulmonary asbestosis, along with a brief occupational history of each case and a statement as to the presence or absence of breathlessness.

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(Slide 17.)

The lung fields are clear. Blunting of both costo-phrenic angles is seen. It is conceivable that these changes are the result of a previous pleural reaction. The individual is 63 years of age, and has worked 36 years as a bagger and 7 years as a millwright. He has no complaints and is still working. The point to be made here is the 43 years of rather extensive exposure, without clinical or roentgenological evidence of pulmonary asbestosis.

(Slide 18.)

There is a fine network-like pattern of increased density in the region of both cardio-phrenic angles, with associated slight granularity extending toward the costo-phrenic sulci. A definite haziness is present in the middle thirds of both lungs. Accoring to our classification, these changes are compatible with a diagnosis of asbestosis, Stage I. The man is 76 years of age, is still working and has no complaints. His occupational history includes 37 years as a millwright and 9 years of very slight exposure. After 46 years of exposure to asbestos, this individual does have typical X-ray changes as a result of pulmonary deposition of dust, but he has no respiratory symptoms whatsoever.

(Slide 19.)

This is a 61-year-old man who is dyspneic, and as a

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result of this has been compensated. The chest roentgenogram demonstrates the classical findings of asbestosis, Stage I. Definite cluding is present as both bases, characterised by slight mottling interspersed with linear bands of increased density which extend into both costo-phrenic angles. In these latter regions there is blunting due to a pleural reaction. The area of increased density in the second right anterior interspace is presumably the result of a stable tuberculous lesion. The man has worked for 20 years in the testing room and 25 years as a millwright. For all intents and purposes this individual probably had an exposure comparable to that of the man in the previous case. The point is that, while both came within the same roentgenological classification, one had no respiratory complaints while the other was dyspneic.

(Slide 20.)

Here is a 46-year-old man with an occupational history of 11 years as a bagger, 2 years as a millwright, and 11 years of slight exposure. He has no complaints and is still enjoying gainful occupation. The X-ray changes are less marked than in the two previous cases, but this must of necessity be classified as asbestosis, Stage I. Along the right cardiac border there is an almost homogeneous density, extending from the inferior aspect of the hilum to the cardio-phrenic angle. Fine, irregular densities radiate out from it to the costo-

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phrenic sulcus, which is blunted. Linear areas of increased density are visualized at the left base, extending from the left cardiac border to the costo-phrenic angle, which, as on the opposite side, is blunted as a result of pleural reaction. The middle and upper thirds of both lungs are clear. This type of change is considered to represent rather early X-ray manifestations of pulmonary asbestosis.

(Slide 21.)

An 86-year-old man who has worked for 34 years as a millwright. As you can see, he has considerably more roentgenological change within his lungs than the previous cases. The outline of the cardiac silhouette is not sharply defined. The densities have extended to the peripheral portions of the lower and mid-lung fields, and even to the upper thirds. The character of the changes resembles a "ground-glass" appearance. The roentgenological classification is asbestosis, Stage II, and this man was compensated, not on this alone, but because of his extreme shortness of breath.

(Slide 22.) Another example of Stage II asbestosis. The densities involve the middle and lower thirds of both lung fields. The over-all character is that of a "ground-glass" appearance; however, nodular densities are present in both costo-phrenic angles. The left costo-phrenic sulcus is blunted. The cardiac borders are not clearly delineated. This man is

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60 years of age and has worked for 20 years as a bagger and 6 years in the mill. He had extreme shortness of breath and has been compensated.

(Slide 23.)

This individual worked for 21 years as a bagger, 10 years as a shipper, and 13 years in an area of slight exposure. The changes noted in the X-ray are sufficient to classify this as a case of Stage II asbestosis. There is marked pleural thickening in the right hemithorax, extending from the costophrenic sulcus to the apex. The right diaphragm is irregular in outline as a result of this general pleural involvement. The characteristic densities are scattered through the lung fields, being most pronounced at the right base. This man had slight to moderate exertional dyspneia.

(Slide 24.)

This man was employed by the asbestos industry for 37 years. 24 of those years he spent as a bagger and shipper. The typical "ground-glass" appearance is noted throughout both middle and lower lung fields. Also seen is bilateral apical pleural thickening. The cardiac borders are not clearly defined. The transverse diameter of the heart is increased above the normal. While I did not have an opportunity to fluoroscope the individual or to review oblique films of his chest, I should think that most of the enlargement was due to a dilated

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right ventricle. The man is said to have died two years after this film was taken -- as a result of cor pulmonale. He worked up to two months of his death, and his only complaint prior to that time was of slight dyspnea.

(Slide 25.)

Here is a typical case of Stage III asbestosis. The left border of the heart is "shaggy" in outline. The shadowing extends throughout both lung fields and is more dense than in the two previous cases. This man retired in 1949, after working for 24 years as a millwright. At that time he had very slight exertional dyspnea. He now exercises each day by doing odd jobs around his home and by walking about one mile per day. His X-ray has shown no change since 1946.

(Slide 26.)

The left border of this heart also presents a shaggy appearance, and the parenchymal shadowing is heavy throughout. The individual is 56 years old and has been employed for 26 years as a millwright and 2 years as a bagger. He is still working and has no complaints.

This paper does not represent a complete discussion of the problem of pulmonary asbestosis and silicosis. It was my intention only to demonstrate various roentgenological manifestations of the lung abnormalities incident to these conditions. However, I should be remiss if I did not at least

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mention the fact that these fibrotic diseases may give rise to cor pulmonale. The right ventricular enlargement is best demonstrated in oblique views of the chest and by fluoroscopy. It is possible to have a considerable degree of such enlargement without gross alteration of the cardiac silhouette on the postero-anterior projection.

Conclusions.

1. Silicosis and asbestos result from the inhalation of sufficient quantities of free crystalline silica and asbestos.

2. When pulmonary reaction occurs as the result of deposition of either dust, a characteristic though different roentgenological pattern is manifested in the chest X-ray.

3. An X-ray classification of both conditions is employed according to the extent of the characteristic chadows.

4. Simple silicosis is usually not accompanied by any pulmonary functional impairment, whereas the complicated forms almost always demonstrate some departure from the normal.

5. The functional impairment in silicosis is due to diffuse obstructive emphysema. A chest fluoroscopy and or chest roentgenograms taken in full expiration aid in evaluating the presence or absence of this complicatior

6. Not all cases of asbestosis have a breathing problem. When it is present, it is due to a "tight lung." Expiration films are not of much value in determining the

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presence or absence of respiratory impairment in these cases, as there is no increase in residual air.

7. Unlike silicosis, we found some individuals with slight X-ray changes indicating asbestosis, with definite functional impairment present.

Thank you.

CHAIRMAN CHAPMAN: I am sure you will all agree with me that is a most instructive paper.

The man who has been asked to discuss this paper is a Professor of Medicine at the Harvard Medical School -- a Professor of Radiology, certainly not a Professor of Medicine as all you internes here will agree with me he is a man who believes in his own techniques and for years has tried to have the stethoscope thrown out of the practice of medicine.

Dr. Merril Sosman.

(Applause.)

DR. MERRIL SOSMAN: Dr. Chapman, Commissioner Johnson, ladies and gentlemen, our problem in the general hospital trying to make a diagnosis of silicosis is quite different from the problem just presented. In fact, with any group of patients who are being studied and roentgenographed in the sanitorium or in the survey of any particular industry, in most of those situations the patient is comparatively well and you have the one problem of deciding does this patient

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silicosis or asbestosis and in what stages. Our patients are sick. They come in with shortness of breath, cough, fever, loss of weight; and we have to try to determine the cause of that individual's variation from normal; and from all the shadows on the lung that we may find, it is surprising how many different diseases and conditions can imitate one another.

At our institution we had 8,000 patients with X-rays last year of their chests, and there were only three that we could definitely decide had anything to do with pneumononiosis, occupational pneumonitis you may call it. We see many pulmonary shadows you see in these early stages of silicosis and asbestosis without any other symptoms or signs, and right there I'd like to take up the battle again with Dr. Chapman. We fought this out many times.

Dr. Briston says in evaluating the X-ray film or X-ray study you must also consider the history, the laboratory examination, and the physical examination, and I submit to you, gentlemen, that as a physician practicing radiology, not as a technician, that I do not have a single patient in which the physical examination changed the diagnosis made from the history of from the X-ray findings of a combination of the three. In other words, it's a waste of time listening to your lungs.

(Laughter.)

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You can go ahead. It impresses the patient.

Here are the problems we come up against. First and foremost, carcinoma of the lung. We can get a carcinoma of the lung that will duplicate the conglomerate you have seen and no history of silicosis at all. We can get the ..... lesions from elsewhere in the body that will duplicate the second stage, the nodular stage. We can get the lymphatic permeation coming up from the lymphatics, and this will duplicate the effect you get with asbestosis .....

(Prepared paper read.)

(Applause.)

CHAIRMAN CHAPMAN: We are doing fairly well on time, and it might be well to ask if one of the men would like to ask Dr. Briston a question or two and see if he can give us an answer.

Are there any questions from the floor?

If there are no questions, we will proceed. I might say after the next paper we will have a formal break of about fifteen minutes and then wind up with the final three papers on the program. We will get going with the next paper now, and as I look over this audience I see several of those eager faces characterizing men in research. I am sure this speaker will have a particularly interesting paper for that group because he has done these studies of pulmonary deficiencies as

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mentioned by Dr. Bristol, and he will talk to us on the Functional Abnormalities of Industrial Pulmonary Fibrosis.

Dr. Wright did a good deal of this work at the Saranac laboratories and only recently he has gone to Cleveland where he is now Director of Medical Research at the St. Luke's Hospital in Cleveland.

It gives me great pleasure to bring before you Dr. George W. Wright.

(Applause.)

DR. GEORGE W. WRIGHT: Mr. Chairman, ladies and gentlemen, I have been asked to discuss the functional abnormalities that occur in certain of the industrial pulmonary diseases.

I suppose the idea behind this is to impress you with some of the diagnostic difficulties and, also, to give you such help as I may in regard to that ever vexing problem of estimating disability. Time certainly will not permit us to go into all of the industrial pulmonary diseases, and I propose to talk only about silicosis and asbestosis; and I do that primarily for purposes of comparison.

If I may have the first slide. I'm going to prepare a little background so that those of you who do not have a medical background can get something from the 15 or 20 minutes that I'm going to speak.

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What you have before you is a crude and graphic representation of the essential features of a pulmonary apparatus. The bellows you see represented is a concertina. The bellows are made up of the thoracic wall in the diaphragm and muscles that actuate these various walls and then depressing the chest air is moved into and out of this space inside the chest, and the air goes in through a system of tubes. And these tubes have much the appearance of an inverted tree bare of its leaves. There are literally thousands and thousands of tiny little tubes, and then on the end of these tubes there are actually things similar to leaves, which we call alveoli. And suspended inside this air space is a vascular network made up of tiny blood vessels. I don't know exactly how to describe that to you other than to say that it looks more like a can of fishing worms than anything else except that there is a little air space between all portions of this can of fishing worms.

I have represented this as a tube conducting blood that appears here and then divides up into a system of parallel channels and ultimately collecting here. The blood flows into the air space, divides up, and spreads out over this enormous surface area, estimated as 7 square meters, and then collects again and comes out here. Of course, it is pumped into this air space by way of one chamber of the heart and collects back

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again in the other part of the heart and pumped out through the body.

I think this drawing is very useful because it lets me point very quickly to the vulnerable areas in the system. Certainly one of the vulnerable areas has to do with this bellows. Anything that will interfere with the mechanics whereby this chest can be enlarged and compressed will certainly interfere with the business of getting air into and out of the lung. And anything that interferes with the passage of air down through that system of tubes will interfere with the ease one experiences in getting air into and out of the lungs.

Then, once the air is in the lung and distributed around it must go across the tiny membrane that separates the blood from the air space. In other words, here you have a most remarkable air space which has this great mass of capillaries inside it but the blood doesn't get out into the air space itself. It is kept in this system of tubes, and the thing that contains it is a very thin wall, and the blood can diffuse across that membrane very rapidly in both directions.

And as the blood comes in through this tube, coming back from the body itself, it is very poor in oxygen and rich in carbon dioxide. Inside this very thin surface oxygen is diffused from the air in the lung and diffused into the blood; and carbon dioxide, which is in excess in the blood, is diffused

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out into the air space of the lung.

Now, as oxygen is diffused in and carbon dioxide diffused out, the air space changes its character and, therefore, being like the air inside a crowded room, becomes full of smoke and for comfort you have to ventilate it, in the same way the air spaces must be ventilated in order to provide that atmosphere rich in oxygen and poor in carbon dioxide. So a vulnerable point in this membrane is anything that tends to thicken the membrane or make the distance the gases must travel greater and thus interfere with the diffusion of those gases across the membrane.

Another vulnerable point is the actual number of capillaries. If one were fortunate enough to be born with an enourmous excess of this particular commodity -- and we can suffer a tremendous loss of them -- if one removes enough of those capillaries then the heart will have difficulty in pumping blood in sufficient quantity to permit one to carry on.

That, in essence, is the make-up of the apparatus that we call the respiratory system. This is in the control of a higly integrated apparatus in the brain, but I shan't go into that at this time.

I would like to show you two diseases which interfere with this apparatus in a very definite and distinct way. And the reason I show you these two is that they represent the

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the changes that occur in our industrial pulmonary diseases.

The next slide. First, I would like to show you a pure example of an individual who has an obstruction in his system of air ways -- the pipes that lead into the air-containing chamber.

Here are three films of the same individual, and I show you these because they will explain some of the later films. The film on your left is a film taken in full inspiration, with the lungs as full as one could make them; and the film on the right is in full expiration, with the lungs as empty as one could make them.

The black area is the air-containing part of the lung, and the difference is size of the black area represents the degree by which the lung can be changed or the volume of the breath of the lung; and anything which interferes with the expansion of the lung will lead to trouble; and anything that interferes with emptying the lung will lead to an unusually large film on expiration. That is only common sense.

The next slide.

Now, I put this in, also, just in case any of you were confused.

(Laughter.)

This square box or long box is the same thing as the bellows, and here is the blood flowing around in a circle

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coming back, and the gas goes across from the air of the lung by diffusion; and I show that because we can actually measure the pressure of oxygen in the air space and the pressure of oxygen in the blood and thereby determine the head of pressure that governs the movement of gas from the air in the lung into the blood of the lung. And if you know that head of pressure, you have a pretty clear measurement of the ease with which the gas crosses the membrane. Interference with the passage of gas leads to pressures. In other words, the oxygen does not get into the blood as readily as it should.

Here is a man who had no industrial exposure at all. He is a dentist. Although I understand some dental technicians have been known to develop silicosis, this man has had no industrial exposure, but he does have extreme shortness of breath, and he has the disease we .....

This is a film in full inspiration. This is on the left, and full expiration on your right, showing you the tremendous impairment to emptying of the lung present in this particular man. Measurements that have been made in various laboratories have shown quite clearly this impairment in emptying of the lung occurs primarily in those little tubes I spoke to you about which carry air into and out of the lungs. In other words, there is an obstruction to the air flow, particularly in the phase of trying to empty the lung --

therefore, this disease has the name: diffuse obstruction emphysema.

That slide will show you to a very marked degree to which respiratory functions are impaired. First of all, the maximum breathing capacity, which in this man should be 124 liters. In other words, 124 quarts of air in a big man, and might be, for example, below 30. In other words, there is a marked impairment of the power of his breathing. And this impairment of his power of breathing pump is primarily responsible for his great difficulty in carrying out physical work and to some extent responsible for the severe shortness of breath.

Now, in addition to that marked impairment as a pump, you will notice, also, that the volume of air in his lungs when he has exhaled as far as he can exhale is still almost 6 quarts of air or 6 liters and should be only 1½, so there is a marked increase in the residual air.

These two phenomena, a reduction in breathing power and marked increase in residual air are almost pathopneumo ... diffuse obstructive emphysema; and from a clinical point of view if one doesn't have this and doesn't have a diminution of breathing capacity, it is most unsafe to venture a diagnosis of diffuse obstructive emphysema.

Now, the histologist undoubtedly can find areas of

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obstructive emphysema in individuals who have normal physiological data, but when the areas become extensive enough to interfere with the man's ability to get around and do physical work then these measurements will be distinctly abnormal.

You will also note in this particular man there is a marked impairment in the ease in which oxygen can get into and out of the blood. The hemoglobin is unsaturated and that his carbon dioxide content is higher than it should be.

The next slide.

This is a classical example of diffuse obstructive emphysema, and this is of nonindustrial origin. And the underlying pathology --- This is the exact opposite. Here is a man also completely incapable of physical work because of severe shortness of breath, but this man has an impairment of the passage of gas across the membrane that separates the air from the blood in the lungs. This is an industrial pulmonary disease, and those of you in the Boston area have a better acquaintance than you would like to have, I'm sure.

This is a man who has been exposed to beryllium, and the autopsy confirmed the diagnosis. You note the diffuse abnormality of the film on your left in full inspiration. Here is a film in full expiration. And those of you up close can see this man actually still retains a good ability to empty his lungs.

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The next slide shows you his breathing power is not impaired at all. He has normal breathing power. He has perfectly normal residuary air and no evidence of retention of air in the lung during expiration. The only thing present is that it is smaller than normal. He has a restricted lung; a tight lung; a lung that cannot be filled to the extent that it should be.

The next slide.

This man has a much more severe alteration of the diffusion of gas across the membrane than the man with emphysema. The other man had a head of pressure across the membrane of only .... This man had a head of pressure of 36, showing definite impairment.

The next slide shows blood gases obtained during exercise. Here is a tremendous increase in the head of pressure across the alveola membrane.

The next slide.

Now, this man has extreme shortness of breath but not due to a loss of breathing power. He has absolutely normal ability to breathe so far as getting air into the lung and out of the lung, but his response to breathing is tremendously exaggerated as is shown by the fact on doing a simple level walk 2½ miles he was breathing 76 liters, and most of us breathe 25 liters -- that being shown by comparing the amount

of

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of air breathed and the oxygen .....

This figure is three times as high as it should be, as it would be at about 18,000 altitude. He becpmes short of breath doing easy pieces of work because he breathes a much greater volume of air per minute. In other words, a load is cast upon his breathing apparatus. I show this not so much because it is an industrial pulmonary disease, but because it represents the second way in which the breathing apparatus fairly commonly becomes altered.

Now, may I show you very quickly several cases of silicosis and asbestosis.

The next slide.

This is a man with diffuse nodular silicosis. That is what I thought when I went back to look at it. It is very ..... for some reason I picked out one case of ciderosis. This is a film of a man with ciderosis. This data I brought with me is of the patient who really does have silicosis, but this is a mistake in making out my slides. This is a man with ciderosis, but it looks like silicosis.

The next slide.

This is a 49-year-old man. Now, I'll just call attention to the salient features. There is no impairment of breathing power. The ..... is slightly elevated, possibly abnormal for this man, but still falling within the limits of

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normal.

The next slide.

There is no evidence of impairment of passage of oxygen across the membrane that separates the blood from the gas in the lung. Normal arterial saturation and normal head of pressure for an exercising man.

The next slide.

Breathing response in terms of ..... is entirely normal, 26.4. Here we have a man with no measurable abnormality and who has no clinical complaints whatsoever. Now, if Dr. Lynch had brought some slides to show other silicotics, you would easily understand why this man shows no abnormality. As he mentioned industrial silicosis is a focal phenomena and in between the areas of nodulation there are great areas of perfectly normal lung tissue, so that although there are abnormal areas of tissue in the lung, in the aggregate they don't compose enough of the lung to make their abnormality known by a functional measurements.

The next slide.

This is a slightly different story. This is a man with a conglomerate silicosis at full inspiration and full expiration, showing impairment of emptying and a smaller lung than it should be.

The next slide.

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This shows a marked diminution of breathing power as compared to what it should be for a normal man, a definite elevation of residual air, no change in the total. In other words, here we have two findings that go very well with the diagnosis of obstructive emphysema, and that man was totally disabled so far as doing manual labor.

The next slide. Now, in spite of this he had impairment of passage of oxygen.

The next slide.

Contrasted to that man who showed definite evidence of diffuse obstructive emphysema is this individual who has conglomerate silicosis. You see the two large butterfly-shaped masses, but on the expiration film you see the lower portion of the chest can be emptied quite well.

The next slide.

This man is still working at actual physical labor, and you see that his maximum breathing capacity is lower than it should be. It is nowhere near as low as the previous individual, and there is no evidence whatsoever of increase in residual air. So that you can have conglomerate silicosis and still have no seriously disabling functional disturbance so that the diagnosis of conglomerate silicosis is not synonymous with total disability. The individual with conglomerate silicosis is very apt to have very severe impairment but it is

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not an absolute fact.

The next slide. We passed that. I'll take the next slide.

The degree of interference with breathing responses is negligible.

The next slide.

Now, I'd like to show you several examples of asbestos exposure. We have in the last few years studied approximately 75 men who had various degrees of exposure to asbestos fiber, and this is a classical case, now proven by autopsy, of asbestosis. Here you see the film in full inspiration and full expiration. You will note that this individual has perfectly normal ability to empty his lungs. There is no evidence here whatsoever of anything that looks like the severe grade of emphysema I showed in the very first case.

The next slide.

He has normal ability to pump air into and out of his lungs and no evidence whatsoever of an increase in residual air, so that although the histologists report emphysema is characteristic of asbestosis, that certainly was not present in this man from a clinical point of view. Now, that doesn't mean that there were no areas that the histologist would identify as emphysema. I am sure there were. As a matter of fact, there were in this particular man, but the areas were minor in extent

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and not large enough in the aggregate to be measureable; and from a clinical point of view -- certainly in this particular man -- I think we have ample evidence to show in the group as a whole from the clinical point of view diffuse obstructive emphysema is not characteristic of asbestosis. Much more characteristic is the finding you will see in the next slide, where you see that at rest this man has very marked arterial saturation. In other words, there is an impairment of the passage of gas across the alveolar membrane and an increase in the head of pressure. This individual from a blood gas diffusion point of view is like the pulmonary ....

The next slide.

The response to exercise is of the same category, a ventilation equivalent much higher than it should be, so that, in essence, the man with asbestosis of a severe enough grade has a generalized diffusion problem which I think one should be prepared for; and in view of Dr. Lynch's description, instead of being focal with large areas of lung tissue in between the asbestosis, the lesions are apt to be diffuse and practically all of the lung tissue is involved so that the blood must go through areas where there is an impediment for the passage of oxygen across the membrane.

The next slide.

I show you several other cases because they exemplify

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certain questions that might be raised.

Is it always true that the man with X-ray evidence of asbestosis must also have functional abnormalities? This is a case in point. This man, although you can see in the film what we consider to be typical findings with roentgenogram of asbestosis -- this is full inspiration and full expiration -- you will note very good emptying.

The next slide.

This man we consider asbestotic who had a high grade of intensive exposure as indicated by that figure of 366 .... nothing here to indicate diffuse obstructive emphysema.

The next slide.

The arterial saturation and exercise entirely within normal limits.

The next slide.

In response to exercise within normal limits. Here we have a man with definite asbestosis, but no functional impairment.

The next slide.

This is another individual I put in to consider the question of whether or not years of exposure of necessity lead to the development of asbestosis. Here's his full inspiration and full expiration film. These slides are terribly overexposed, and the films are much better to look at. There was no

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evidence here of asbestosis.

The next slide.

In spite of a very intense and prolonged exposure as dated by the figure of 504, this man had nothing in the X-ray that is abnormal. Normal breathing power. No increase in residual air.

Next slide.

There is a perfectly normal saturation during exercise and no elevation of pressure across the membrane.

The next slide.

Abnormal response to exercise. Here we have an individual with years of exposure but no evidence whatsoever of asbestosis.

The next slide shows you a very different picture, a man with exposure whom we thought didn't show clearly -- at least on the X-ray -- any evidence that we could interpret as asbestosis. This is full inspiration and full expiration. And this was to our surprise.

The next slide.

We found that in a certain number of these individuals although they had normal breathing power which we expected even in the people with asbestosis.

The next slide.

Here's a man with no evidence in the film of asbestosis

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that was clear-cut but with a very difinite degree of impairment of the passage of oxygen across the membrane as evidenced by a reduced arterial saturation and elevated .... and this fits in with what Dr. Lynch told you earlier in the physiological changes long before wide-spread fibrosis develops. He has seen some evidence of a diffuse accumulation of fluids in the alveolar suffaces.

The next slide. I'll pass on very quickly to the next one.

We found abnormalities in this group of 75 men, and heading the list is a lowering of the arterial CO_2. In other words, evidence of impairment of diffusion of gas across the alveolar membrane and increase of ..... In other words, the two commonest abnormalities in asbestos exposure had to do with the passage of gas across the alveolar membrane, and in a sense they are pulmonary granuloma and very clearly contrasted to the diffuse obstructive emphysema so typical of disabling silicosis. The only thing that had anything to do whatsoever with something other than gas diffusion is this reduction in total volume. In other words, these people show a somewhat tight or restricted lung.

Now, from a diggnostic point of view, this presents a very confusing picture because it suggests at least and needs confirmation -- it suggests at least that the earliest

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that the earliest evidence clinically of asbestosis will probably be found in a study of the arterial blood gases during moderate to heavy exercise and the roentgenographic changes will come somewhat later.

The next slide.

I just want to show for one moment the meaning of this in terms of ability of a man to work. In other words, it is one thing to say that we can find abnormality of the pulmonary function. That does not of necessity mean that every individual has loss of power for exercise work. You must remember that great civilizations have been built at altitudes where everybody had arterial saturation and that many of our cardiacs have carried on a very useful life with a moderate degree of arterial saturation,and we tried to get at this not just in relation to asbestosis but other things by exercising people to their maximum to the greatest ability they have for physical work on the treadmill and the measurement of their physical work and peak oxygen intake; and here a group carried their peak oxygen ability to do physical work; and this next slide you see this grid which describes the distribution of that population.

The next slide shows you a group of pathological subjects. There are two or three very pathological ones in that area, but pathological on the good side. This is a group

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of our very fine ..... who have a phenomenal ability for oxygen intake. Here's a grid of normal people; and there are some cases of asbestosis superimposed on that; and you will see there is one above average; and they tend to cluster in the range of lowered ability, but they still -- many of them are within normal figures.

The next slide will show in a little different way -- This probably won't be visible to all of you. Here is a group of people. All of them except those with open circles have had exposure to asbestosis fiber. Those that are blue are people with arterial saturation definitely, and those in red have none. And you will see that they tend to all fall in together, so that although in the diagnostic point of view the finding of arterial saturation has relatively little significance for predicting the degree to which an individual will be disabled.

In conclusion I would just like to reiterate I believe there is a distinct difference between the silicotic and the asbestotic in this way -- two particular ways: The silicotic may have very advanced evidence in the roentgenograph of his disease without any physiological abnormality.....

Thank you.

CHAIRMAN CHAPMAN: We have with us to discuss this paper, Dr. James Whittenberger. I'm sorry I can't regale you

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with any personal eccentricities of Dr. Whittenberg because I didn't meet him before the meeting so that I could tell you things about him.

Here he is. Dr. James Whittenberger, Department of Physiology, Harvard School of Public Health.

(Applause.)

DR. JAMES WHITTENBERGER: I'm sorry I wasn't more considerate to our Chairman. I didn't realize the opportunity I was depriving him of.

Mr. Chairman, ladies and gentlemen, I was pleased to hear Dr. Lanza's remark. He made a number of remarks I was going to make. I hasten to say many of us in the field of pulmonary physiology don't understand all of what Dr. Wright was talking about, but I personally agree with the statement Dr. Lanza made that in many ways we are in a primitive state as far as our information about industrial pulmonary diseases are concerned.

I was reading the other day an essay by a British doctor written in 1796 in which he discussed the effect on the health of people of one of the large industrial cities in England -- the effect of the smoke which heavily contaminated the atmosphere. He said it was well established that elderly asthmatics, especially in the fall of the year, when the smoke was inclined to be most severe, suffered severely and often

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died as a result of this exposure. Even today, we are very much concerned with the problem, and I am not sure we understand it very much better than he did at that time.

I think, having been shown the large array of pulmonary function tests which Dr. Wright used very skilfully in his study of industrial pulmonary disease, we should ask ourselves the question of the significance of some of these tests. I am not by any means against doing these tests. I think that is the only way we will learn about these conditions and perhaps learn how little we know about them, but the pulmonary system -- the respiratory system in spite of the simplified diagram Dr. Wright presented is an extremely complex system. The inner relationships of ventilation of the lungs and the diffusion in the lungs and circulation throughout the lungs are so complex it is often extremely difficult to say what is due to impairment of the circulation and what is due to the impairment of diffusion.

As a doctor seeing patients with pulmonary disease which may be related to occupational exposures, I am sure that you would like to know what tests are really feasible outside of the larger hospitals and university centers. I think that the vital capacity, especially if it can be timed by the capacity is a measurement which is definitely worthwhile. I think the maximum breathing capacity, which is a really simple test,

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is very worthwhile.

There is a point here about normal standards. If we use the standards in the literature, it is often difficult to tell what patient is normal or not normal. Whereas if we had a vital capacity for maximum breathing capacity on the patient himself at some prior time, when presumably his lungs were normal, we would have a much better idea whether disease has affected this individual or not.

I don't know how feasible it is in industrial medical practice to get this type of measurement, but I personally think it would be very worthwhile.

A good many of the tests Dr. Wright described depend a lot upon the cooperation of the patient. It is true that arterial saturation during exercise changes independently of any willingness on the part of the patient, but the degree to which he is willing to push himself is very important. The industrial capacity, the NBC, depends obviously upon the voluntary cooperation, and the extent to which we can be objective in our measurements of the patient is certainly desireable.

I would like to take this opportunity to agree with Dr. Sosman about the value of the physical examination. I don't know how many of you have read about the tests that were carried on at the Cardiff Pneumoconiosis Research Station in Wales a year or two ago, but some of the men in this outstanding

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research station were dissatis fied with what they could tell about a patient's physical examination, and they invited a number of Professor Christie's experts to come down from London and examine the same patients. These patients had emphysema or a mass of pulmonary fibrosis resulting from pneumoconiosis occurring in coal workers.

Since time is very short, I will only say that the doctors were dismayed by how often they disagreed on physical signs even in patients with advanced pulmonary disease. There were discrepancies. They just didn't agree at all. So that not only should a doctor thrown away his stethoscope, but he may as well not bother to percuss or inspect the patient.

(Laughter.)

I should not close on such a pessimistic note, but I would like to say that many researchers in this field believe there is hope in the development of subjective tests of the type which meaure the mechanical properties of the lung, the elastic properties, the resistance to breathing, to what extent the lung which may appear to be normal or abnormal by X-ray does it differ from normal in terms of its stiffness. These studies are relatively in their infancy. They have not been to any great extent applied in patients with industrial pulmonary disease. So I will close by saying we need humility in our study of pulmonary disease, but still we should continue

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to keep trying.

Thank you.

(Applause.)

CHAIRMAN CHAPMAN: That slight crunching noise you heard was Richard Cabot and Lannek rolling over in their graves.

We will now take a five-minute recess.

(Recess taken.)

CHAIRMAN CHAPMAN: While we are waiting I am going to bring up a short question-and-answer period. Dr. Lanza is here, and we'd like to have him answer these questions.

Dr. McLaughlin, of the U. S. Navy, has come forward with two questions, and I think the person best able to answer these questions is Dr. Lanza.

First, he'd like to know about the maximum and minimum sizes of particles causing the difficulty in asbestosis.

DR. LANZA: Generally speaking it has been accepted that the particiles of silica are three microns or smaller. As to the minimum range, I don't think anybody can answer that as yet. That is one of the things that still remains to be determined, the possibility of harm by submicroscopic particles.

As for asbestos, all I can say that particles have been reported found in the lungs as long as 200 micrometers. Again, when we get down to minimum particles, I don't think we know the answer.

That is not a very satisfactory reply, but it's the best I can give you.

CHAIRMAN CHAPMAN: The second question is: Are there substances other than silica, iron, asbestos, and others identified as causing pneumoconiosis, particularly rayon, cotton fiber, hemp fiber or chippings from paint?

DR. LANZA: As far as I know, organic particles do not cause pulmonary fibrosis which is associated with pneumoconiosis.

Does that answer your question?

CHAIRMAN CHAPMAN: Dr. McLaughlin, does that answer your question?

DR. MC LAUGHLIN: How about paint, which is not an organic substance?

DR. LANZA: I never heard of that, but I don't think that paint particles are troublesome. If that is so, I never heard of it.

CHAIRMAN CHAPMAN: I know that men in the Navy spend a lot of time chipping paint.

Do you think, Dr. McLaughlin, that you have seen men with some sort of pneumoconiosis from inhaling these paint chips?

DR. MC LAUGHLIN: Yes, I have seen acute episodes of a form of pneumonitis following exposure.

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DR. LANZA: Well, now, pneumonitis -- pneumoconiosis implies a formation of fibrous tissue; pneumonitis is a chemical reaction -- an inflammatory reaction of the lungs, and I don't think it can be classified as pneumoconiosis. I'd rather have one of my pathological friends answer that question. That is a separate subject.

CHAIRMAN CHAPMAN: Thank you very much, Dr. Lanza.

Now, I have an important announcement to make. Those of you making notes may welcome some relief. Dr. Phillip Bricker said these papers will probably be published in the archives of Industrial Hygiene and Industrial Medicine of which he is the editor, subject, perhaps to some editorial revision. That is the understanding and agreement. I thought you would like to know.

We'll get on with our meeting, and I think you will agree this meeting has sort of a broad national character to it in having a man from the West and the South and men from many areas coming before us.

Now we are going across the border into Canada and have a paper on Some Clinical Observations of Asbestosis in Mine and Mill Workers, by Dr. Paul Cartier, Thetford Industrial Clinic, Thetford Mines, Quebec. I don't think I have to remind you that the Thetford Mines produce 80 per cent of all the asbestos that is used in the United States.

It gives me great pleasure to present to you Dr. Paul Cartier.

(Applause.)

DR. PAUL CARTIER: Mr. Chairman, ladies and gentlemen, in the presence of a group of qualified pathologists, physio-pathologists and radiologists who have at their disposal concrete and objective means of studying a disease, I am reluctant to present the usual clinical description of asbestosis because, in my opinion, even the best clinical description remains a matter of subjective and personal impression and as such can be considered controversial; and it is for this same reason that I am bring to your attention a series of remarks and comments inspired by my work, which consists in the medical supervision of some 4,000 asbestos workers.

From 1945 to 1953 the annual medical and radiological examination of the asbestos workers along with the histological study of 58 autopsy cases permitted the detection of 128 cases of asbestosis, 40 of those are already dead -- and have been autopsied -- and 88 are still living.

The first slide.

TABLE I

THETFORD MINES SURVEY 1945 - 1953

Distribution of 128 cases of asbestosis by age groups and by intensity.

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	AGE GROUPS					
	35-49	50-59	60-64	65-70	70 & over	TOTAL
Minimal Asbestosis	20	24	8	11	9	72
Moderate Asbestosis	2	11	13	5	4	35
Advanced Asbestosis	0	5	10	3	3	21
TOTAL	22	40	31	19	16	128

The first table gives the age group distribution and the classification by degree of asbestosis of those 128 cases. You may see that there are 72 cases of minimal, 35 cases of moderate and 21 cases of advanced asbestosis. The age distribution indicates that 16 cases are 70 years and over -- one case of far advanced asbestosis is 84 years of age and still living -- and 66 cases are 60 years and over.

First, it seems indicated to explain how the diagnosis of asbestosis has been made for these 128 cases. The answer is the following: 121 cases have been diagnosed by radiography; 33 of these 121 cases have been confirmed by the pathological study of the lungs, so we can say that 33 cases have been diagnosed by radiological and histological interpretation. The remaining 7 cases have been diagnosied by the pathological study only, having been missed on the reading of the standard chest films, but it is important to add that these 7 cases

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are cases of minimal asbestosis. Without going into a too long discussion of the effectiveness or the superiority of the radiography over the histopathology or vice-versa, for making a diagnosis of asbestosis, I would like to add the following remarks.

We have to admit that the radiological interpretation might fail to detect cases of minimal asbestosis, but from personal experience and from repeated contacts with Gardner, Sampson, Robert, Bristol, and Vorwald, I can say that no cases of asbestosis of clinical importance have been diagnosed by the pathologist without having been detected anteriorly by the radiologists. I do not know if a similar statement is true for employees of the Asbestos Textile Industry, where apparently the X-ray pattern of asbestosis is fainter than the one found in the Asbestos Mining Industry.

A second comment is about the wide discrepancy in the appreciation of the degree of asbestosis by different pathologists. In a few instances, cases which look like minimal asbestosis to one pathologist have been classified as advanced asbestosis by another pathologist. With the increasing number of autopsies for old employees, with the recent use of the lung biopsy and the lung resection in asbestos workers, we may assume that there will be more instances where pathologists will differ in opinion between themselves and,

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also, will not agree with the radiologist in the estimation of the amount of asbestotic fibrosis present. This lack of agreement is very confusing in any medical study, but still more confusing before a Compensation Board.

For practical purposes, it is of great importance that a solid radiological classification of the cases of asbestosis, correlated with the histo-pathological findings be recognized and accepted by all those concerned with this problem of asbestosis, because the chest film must be considered as an essential criterium in making a diagnosis of asbestosis and, notwithstanding the limitations of the chest films in some exceptional minimal cases, this tool remains more objective and more adequate than any other presumptive criteria.

The second table, please.

TABLE II

THETFORD MINES SURVEY 1945 - 1953

Causes of death in a series of 40 cases of asbestosis

	Minimal Asbestosis	Moderate Asbestosis	Advanced Asbestosis	
Evolutive tuberculosis	5	7	0	12
Coronary thrombosis	1	4	0	5
Cardio-vascular diseases	3	[illegible]	3	10
Cor pulmonale	0	[illegible]	3	4
Bronchogenic carcinoma	3	[illegible]	2	6
Broncho-pneumonia & bronchiectasis	2	0	0	2
Other cause	1	0	0	1
TOTAL	15	17	8	

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The second table shows the 7 main causes of death for the 40 cases of asbestosis who have been autopsied, 12 cases have died from evolutive tuberculosis, 5 from coronary thrombosis, 10 from cardio-vascular diseases, 4 from cor pulmonale, 6 from bronchogenic carcinoma, 2 from broncho-pneumonia and bronchiectasis, and the last case from cancer of the brain.

In reading this table the first question which comes to our mind is: To what extent this enumeration of causes of death is different from a similar enumeration for a comparable group of employees from another industry. Unfortunately, I do not know if there is a marked difference, and I did not have the opportunity to discuss this table with other doctors nor to compare this table with a similar one which may exist in the medical literature; nevertheless, briefly, I want to review these causes of death, trying to investigate the part played by asbestos in the death.

It is true that 12 cases, or 30 per cent, of the deaths are caused by an evolutive tuberculosis; and this incidence may seem too high, but a more complete statistical analysis has revealed that the factor of asbestosis in the whole group of asbestos workers in the Thetford Mines does not influence the incidence and the evolution of tuberculosis. Therefore, I think we can accept that there is no causal

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relationship between asbestosis and tuberculosis.

Considering the second cause of death, the Coronary Thrombosis, it is difficult to explain how a minimal or a moderate asbestosis could contribute to the formation of a thrombosis in the coronary circulation; and, consequently, I am inclined to estimate these 4 deaths as not related to the factor asbestosis.

The third cause is the Cardio-vascular diseases. It is not so simple to say how many of those employees have died from their cardio-vascular disease and how many have died from their asbestosis.

The fact that most of the employees who have died from cardio-vascular diseases are 60 and over and the fact that the statistics mention that 65 per cent to 70 per cent of the population of the same age are dying also from cardio-vascular should be taken into consideration before arriving at a conclusion.

In a few borderline cases in which it was difficult to appreciate clearly the role played by the asbestosis and the role played by the cardio-vascular pathology, the different cardiologists consulted were of the opinion that unless the asbestotic fibrosis is extensive enough to produce pathological or clinical signs of the right heart failure, it is impossible to tell with some certainty that the fibrosis has contributed

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appreciably to the death, especially if there are evident signs of advanced degenerative diseases.

In practice, this group of employees with cardio-vascular diseases and a minimal or moderate degree of asbestosis is becoming a serious problem as far as the compensation aspect is concerned. Without too much imagination and from the findings of an increasing number of autopsies in old employees, we can assume that there is a large unknown number of employees who have a minimal amount of asbestosis and who, most likely, will also present later on a cardio-vascular disease; and then, unless definite criteria are developed to estimate objectively the harm produced by the asbestosis and the harm produced by the fibrosis, the compensation boards might decide to reward compensations on the ground that in case of doubt the asbestosis can be considered as an aggravating factor of any cardiac lesion; and we have already had a few cases in Quebec compensated on that basis.

On purpose, I have separated from the cardio-vascular group the 4 cases of cor pulmonale because these 4 deaths seem quite evidently related to the presence of asbestosis, and there are, also, 2 other cases who although they have died from a bronchogenic carcinoma have also shown evident signs of right heart failure, and we can say that 6 cases of asbestosis have developed a cor pulmonale and have died from their asbes-

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tosis.

The following group of 6 bronchogenic carcinoma present a special interest. Without going into any discussion of this problem of possible relationship between asbestosis and pulmonary carcinoma, I just want to say that there are, also, 7 other cases of bronchogenic carcinoma among the employees who did not have any asbestosis and, moreover, a general statistical survey of all employees in the industry does not seem to indicate any statistical evidence of a causal relationship. Therefore, the part played by the asbestotic fibrosis in this group "5" remains questionable.

The two cases who died from broncho-pneumonia and bronchiectasis died most likely from this pathological processus rather than from their minimal asbestosis. The last case died from cancer of the brain.

In summary, in my personal opinion, 6 cases quite obviously died from asbestosis, and the other 34 cases developed a lesion which could very well be considered as the cause of death, although we do not know the role played by the asbetotic fibrosis.

If I may have the last slide, please.

TABLE III

THETFORD MINES SURVEY 1945 - 1953

Clinical status of 88 living cases of asbestosis.

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	Minimal Asbestosis	Moderate Asbestosis	Advanced Asbestosis
Good	36	4	3
Mild symptoms	11	3	2
Moderate symptoms	8	9	3
Severe symptoms	2	2	5
Total	57	18	13

This last table intends to give the clinical status of the 88 living cases of asbestosis.

In analysing this table, I have to admit that we cannot be too certain of the classification of the cases of asbestosis in minimal, moderate and advanced. Here the classification is based on a radiological appreciation and, likely, the histo-pathological classification might be different.

A second important reason why this table may look highly questionable is that it is quite impossible to evaluate clinically the respiratory function of a group of employees when a large proportion of those employees are 60 years of age and over and when they present at the same time other diseases able to impair the respiratory function to the same extent that asbestosis might do.

Obviously, at that age, there are many other factors other than asbestosis which could influence the cardio-respiratory function and, for example, these factors could explain very well

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why 10 cases of minimal asbestosis are presenting moderate and severe symptoms without any participation of the asbestosis present.

With all its limitations, this table seems to prove at least one thing that the clinical status of 36 minimal, 4 moderate and 3 advanced cases is good and that a total of 59 asbetotic employees are able to work without any discomfort -- I mean those in the first two groups.

For the remaining 29 cases, no good correlation between the degree of asbestosis and the clinical status has been found. There is a better correlation between the age of the employees and the clinical status showing the role played by the age.

This table may also demonstrate indirectly that too many similar tables giving clinical data such as cough, expectoration, and weight -- tables like the one I am just showing -- cannot prove too much.

To summarize, I believe that asbestosis is a serious disease in some instances but more often remains a disease which can be tolerated quite well for many years even without appreciable symptoms as long as another serious disease does not supervene to cause the death.

On the other hand, in practice, this disease may look more serious and cause important medico-legal problems if

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a too scientific medical concept or a too liveral social interpretation are accepted by the medico-legal professions, by the labor and compensation bodies. As a matter of fact, if the least amount or a minimal amount of asbestotic fibrosis is interpreted as asbestosis, "occupational disease," and more so if the compensation boards decide to apply the Aggravating Factor Clause, notwithstanding any effective dust control program, the problem will remain for many years to come unnecessarily serious.

The combined effort of pathologists, physio-pathologists radiologists, cardiologists, and clinicians is needed to orientate any research program and to bring answers to the many unknown aspects of asbestosis.

Thank you.

(Applause.)

CHAIRMAN CHAPMAN: We come now to the last scientific paper on the program, and this comes from Milwaukee, Wisconsin. Our speaker is there associated in medicine at Marquette University Mecical School and conducts a clinic for occupational diseases. Dr. Sander is known to most of you from his writings as an authority I am sure.

We will enjoy hearing from Dr. Sander.

(Applause.)

DR. O. A. SANDER: Mr. Chairman, ladies and gentlemen,

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I think you will all agree there is very little left for me to say. My subject is Asbestosis as Differentiated from Other Pneumoconioses. I decided I better include other diseases and conditions, too, because the X-ray differential diagnosis is fairly clear-cut and asbestosis is clear-cut and certainly quite different from what we say is silicosis, but I'm just going to throw that away and discuss some other things.

As you know, a diagnostitian is a scavenger. He picks up the factual data which others have produced. He absorbs this and comes up with an opinion. I have been observing a group of asbestos textile workers for about 10 years, and we have a good many in that group of approximately 30 who have had a diagnosis of asbestosis ten years ago; and of that group we have had 3 or 4 who progressed, but the rest remained stationary without any X-ray changes as far as we could detect. And when I say "we," I mean not only myself but a committee of industrial hygiene unit of the State of North Carolina with whom I review these films every year. We have been unable to detect progression in the majority of these individuals diagnosed 10 years ago.

What is the reason for that? Well, this little plant has done a remarkable job of dust control. We haven't mentioned dust control today. I think a few words on that might be in order.

Asbestos textile plants disperse asbestos fibers in a

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good many different operations, from the coming in of the raw material to the thread, and it's a very difficult job of clearing the air because ventilation costs a lot of money. But this little plant has invested a lot of money and found it paid off. It is worthwhile in that we have been able to stop the progression which was apparent before that and apparently no more progression than we would have had had those individuals been advised to leave the industry and get into some other industry.

We have had a little progression, as I said, and most of that has been where we knew there still was too much asbestos in the atmosphere. The count was by the U. S. Public Health Service and the ..... method was upwards of a hundred million particles per cubic foot down to a range of five and ten million now in most of the operations.

So much for the prevention. It can be done in the textile plants. And I believe that is the goal we are all looking for. When that goal is arrived at, you as physicians will no longer have to advise patients with definite cases of asbestosis to leave the industry. We are keeping them at work, and I believe rightly so. I believe our experience warrants our continuing them at work.

Let's mention a few things about differential diagnosis just to pick up a few of the points which have been

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made and reemphasized. One, I believe, has not been properly emphasized, and that is the technique of a chest film. I have been asked to evaluate chest films remarkably underexposed, taken with a milliampere tube and at a fifth of a second, and the details were blurred so that the perfectly normal chest can be made to look like a first stage asbestosis -- a perfectly clear-cut first stage asbestosis. The high diaphragm compresses the vascular trunk at the base, giving the shaggy heart appearance. The blurred vascular markings are typical of the first stage asbestosis.

Besides that, I want to certainly discourage the use of miniature survey films for evaluation of asbestosis. There are still too many being made. Proper evaluation cannot be made with a miniature film. The very best 14 by 17 technique with an exposure time of a twentieth of a second or less is indicated. Where we have a condition which presents a hazy pattern on the roentgenogram when there is pathology, how can you evaluate such a thing unless you know you have caught the vascular function in complete lack of motion and where your other outlines are perfectly sharp.

The second factor of differential diagnosis which has been a complicating one is the individual who is overweight. There you have a similar situation of a person in the expiratory phase and you have a high diaphragm. You have a

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heavy pectoral musculature, and you have a compression to the large vascular function at the base, giving the appearance of first stage asbestosis and sometimes even more advanced.

Another point on differential diagnosis is the individual with emphysema, and you know emphysema is a disease which many individuals have who have never had dust exposure as Dr. Wright pointed out.

Again the patient who presents himself to you with emphysema who has had some exposure to asbestos dust and add to that a little bit of bronchitis and you have what looks like a case of asbestosis because it is very easy to read it as a ground glass appearance and into a emphysema chest film. There will be another misdiagnosis because of improper evaluation, and it happens every day. We see it out our way, and I'm sure you see it out your way.

One more word about emphysema. There is no question but what an inspiratory film is insufficient to evaluate emphysema. You should always have an expiratory film to see whether the emphysema is obstructed. If the airways are open you should be much less concerned than if the airways are obstructed. If you have a good total volume apparent between an expiratory and inspiratory film, you have less cause to worry than if there is very marked obstruction.

We have noted a few coal miners with early changes

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of the linear type by the British called reticulation which they now discarded and are calling category one or two, which resembles asbestosis to a high degree, particularly when there is some emphysema, and those cases must be differentiated in detail.

Then there is that vast group of diseases which Dr. Sosman mentioned which must be differentiated. There isn't a single stage of asbestosis which cannot be simulated by something else from the very first suspicion of asbestosis all the way through to the massive conglomerate fibrosis in emphysema.

A lung biopsy has been of some help in differentiating difficult cases if we assume that the individual presented to us has had a thorough evaluation -- by "thorough evaluation" I mean a detailed occupational history. I am reminded, for example, of a case presented to us about 6 or 7 years ago of a boy who was working in a foundry. He only worked there 4 or 5 years. He presented a rather hazy pattern on the chest film, and he had an adherent diaphragm on one side, no history of .... that we could get in the work at the foundry, which was not particularly dusty, just the ordinary foundry dust exposure, and it didn't look like silicosis, and we inquired into his past history and found he had actually been exposed to asbestos dust in his late teens working as a plumber's helper cutting the asbestos coverings for pipes in a small basement room in a

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confined area. When they cut a lot of pipe covering all day long, there is a considerable build-up of dust in an area where there is poor ventilation; and there have been a number of such cases noted in the past.

So it is important to evaluate in detail past occupation histories. Now, many exposures in the past have not been clear-cut. Many persons who have had asbestos dust exposures have also had silica dust exposure and other dust exposures.

I am reminded of a group of cases in a plant where there was considerable asbestos exposure. I was asked to evaluate some of the X-ray films and was presented with these films showing rather massive conglomerate fibrosis in the upper and lower lobe, and I asked the doctor who did the work for this plant -- He was really a surgeon; and he was a sweet old fellow who really didn't have too much interest in occupational diseases -- and I asked him whether the exposures were to anything besides asbestos. I assumed it was asbestos because it was an asbestos fabricating plant. And he said, "Well, they work in the cement plant -- the cement house." I asked, "Is there any exposure there?" He said, "I suppose cement dust exposure. I really haven't been out there for a good many years."

(Laughter.)

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And I said, "Could you find out what the other exposure was?" And he brought out a chart of other materials processed there in the cement plant, and there was a considerable amount of diatomaceous earth processed in this plant along with asbestos. So you will see occupational history involves a detailed analysis of what the exposure factors were in the past from the very first day of work, not just the immediate past exposure.

Biopsy is becoming a fairly definitive diagnostic procedure when all other efforts to make a clear-cut diagnosis fail. It is being done more and more. Just one word on that. If too small a slice of lung is obtained you may not get the lesion which will give you a diagnosis of asbestosis. For example, a pathologist can't make a diagnosis unless he finds the asbestos body. It's an invariable finding there, where there has been an exposure to asbestos dust, in the absence of asbestos bodies -- and you notice I say "asbestos bodies" -- it's a preferable term, where asbestosis is manifest asbestos bodies are found in the sputum of individuals exposed to asbestos dust where there has been no pathological change. So in the absence of pathology of asbestos bodies proper diagnosis can be made with a little snip of lung, and so that diagnosis will be proper a fairly good sized piece of lung should be removed for lung biopsy for study.

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Even pathologically a diagnosis is difficult, and I like to show the slides of a case of an individual who worked for a railroad in the repair shop who had considerable to do with the packing and unpacking of the packing around the locomotive boilers and pipes, and I recognized the exposure to asbestos dust. He became extremely short of breath starting over a period of two or three years. This was the chest film made within about six months before he died. He died of .... a high cyanosis before death. When you have high grade cyanosis you can be sure .... at post mortem.

We have these large air spaces with .....epithelium lining the .... we have fibrosis of the interspace et cetera. All normal structure of alveoli have disappeared. This whole lung was carefully dissected. Sections were made. No evidence of asbestosis bodies was found.

Next slide.

The high power film shows a considerable columnar epithelium. This was finally diagnosed by a number of outstanding pathologists in the middle west as .... with the fibrosis here due to long standing infection in these cysts and not a case of asbestosis.

On the other hand, there are still one or two pathologists who feel this is a case of asbestosis, and the controversy still exists on the question of whether or not asbestos bodies

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are essential in the pathological diagnosis and one which has not been completely settled. I'd like to have an answer on that from our friend the pathologist from South Carolina.

In conclussion I should like to say that to prevent the embarrassment of missing diagnosis with this disease it is essential to evaluate in detail all of the diagnostic factors, and I will agree a stethoscope hasn't helped too much. Occasionally we use it, I will confess, to determine whether or not we have expiratory wheeze which is significant of some obstruction in the airways and, of course, our expiratory film if it's there, but I believe the stethoscope still is of some help, certainly whether or not there are rales in the ..... which is fairly characteristic of fairly well advanced asbestos bodies.

I think it behooves all diagnostitians to carefully evaluate ever case and do everything necessary to establish the diagnosis before making a missing diagnosis and advising the patient to leave the industry in which he has chosed to pursue his livelihood. A very unfortunate situation arises when such a thing happens.

We have a case of ciderosis, for example, an individual who was advised to quit his welding trade. He has a ..... iron pigmentation, absolutely no impairment of the lung function yet he is so convinced that the welding fumes have ruined his

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lungs that he stopped driving a car. He said he could no longer drive his car, and he is doing nothing. It's an unfortunate psychological deal given him when he was advised that his lungs were full of welding fumes and dust, and I think it's a situation that can be improved by more critical disgnostic criteria and differential diagnosis.

Thank you.

(Applause.)

CHAIRMAN CHAPMAN) I will take the liberty here as Chairman to interject a few remarks and change the program just a little bit. We have a little time left.

I have admired the versatility of Dr. Sander. He has characterized and highlighted two important things. One is the fact that sometimes we have to don the peaked cap of Sherlock Holmes to make a proper diagnosis and, second, we often turn to our surgeons. I notice in this group there are at least two thoracic surgeons, and I'd like to call on one, Dr. John Streeter. In a way, I think Dr. John Streeter was the first man who ever operated on a patient with congenital heart disease in an attempt to cure him so maybe this is a pioneer field we are getting into now by opening up a chest and taking out a piece of lung to make a proper diagnosis. This sounds like radical procedure, and I'm sure 10 years ago a good many heads would have waggled on this procedure.

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It so happened on my visit to the Massachusetts General Hospital last summer we had a boy with a few shadows in the lung and some were nodular. We couldn't understand it. We reverted to biopsy. Perhaps Dr. Streeter would like to say something on the hazards of lung biopsy?

I am informed he has left the room.

Dr. Starkey, do you want to say a word?

DR. STARKEY: I am a poor substitute for Dr. Streeter, but I must say that more and more frequently we are now called upon to do lung biopsy for ..... of the lung which nobody can make a diagnosis of. Unfortunately, we have made more than one lung biopsy with ..... graniloma and the diagnosis was ..... graniloma without a definite ideological agent being singled out.

The technique is very simple. Mortality, certainly, is zero. It can be done very simply. One of the most available segments is the inferior lingular segment which one can approach by interspace insertion under the left breast. It is very simple as I said. It takes only 15 to 20 minutes and causes the patient very little difficulty. When one can arrive at a diagnosis by any other method, I think it is very helpful, and we are doing it more and more often. Occasionally we do not arrive at a diagnosis by other means and those do not help and a biopsy will give you the diagnosis and with practically no

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mortality at all.

CHAIRMAN CHAPMAN: Thank you very much, Dr. Starkey.

Your next speaker, according to this program, was to be Dr. Harriet Hardy. Unfortunately, an abrupt illness in her family prevented her from coming here this afternoon. I will call on Dr. Karl T. Benedict to dicuss the talk given by Dr. Sander.

(Applause.)

DR. KARL T. BENEDICT: Mr. Chairman, ladies and gentlemen, it is a privilege to substitute for Dr. Hardy -- she has helped me a great many times in my problems -- but a substitute is always a second rater. I am to discuss differential diagnosis, and as you will recall Dr. Sander threw away his paper, so I'm doubly in trouble.

I am not qualified by experience because I have had no experience with asbestosis. Also, my youth interferes with an adequate interpretation of many cases of pneumoconiosis. As you recall, some of them took 25 to 30 years to develop.

On the other hand, I have been doing industrial medicine for 15 years at one of the world's largest artificial abrasives manufacturers. Since 1940 we have had about 20,000 X-rays on some 5,000 exposed individuals. Prior to that time we did routine examination.

I agree with Dr. Sosman that the physical examination

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is not worth very much in these particular instances. As a matter of fact, Dr. Elliot reads most of our X-rays, so I'm just a figurehead. In fact, thanks to Dr. Phil Drinker, many of our dust problems are controlled at our abrasive plants, with our dust count running around 3 to 4 million. On the other hand, we have a great variety of dust exposure because of the artificial abrasives which we manufacture.

In our fusion by hydro-electric power to combine certain raw materials, we have the problem of fume exposure such as occurs in Schaeffer's disease. We have the problem of free silica, and the problem of the dust from the end product.

We are dealing with the processing of these products, and you may wonder then what type of dust occurs in this artificial abrasive product.

In the first place, you have heard the word, "carborundum" .....

(Applause.)

CHAIRMAN CHAPMAN: Well, I must admire the versatility of Dr. Benedict, a very excellent discussion.

I will now call for a change of pace. We are going to close now with a few remarks from the Hon. Eugene Giroux, Chairman of the Department of Industrial Accidents, Commonwealth of Massachusetts.

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Mr. Giroux.

(Applause.)

HON. EUGENE GIROUX: Mr. Chairman, ladies and gentlemen, I want to assure you I have spent a most profitable afternoon here, which confirms what I have so often said in public gatherings composed of medical men that the administration of the Workmen's Compensation is dependent in a great degree upon the medical profession. I think we can say that degree is greater now than it has been in the determination of liability and the facts in the cases that involved occupational diseases -- not only pulmonary diseases but also other diseases as a result of contact with toxic substances which affect, perhaps, the liver or the kidneys and is manifest, perhaps, in some blood condition.

In listening here, it occurred to me after we make a decision on evidence that is given to us in a case, say, involving pulmonary disease that those of you who are experts in this line must marvel sometimes at the brashness of the findings we make. However, mercifully so, or perhaps from a purely practical standpoint, the Supreme Court has decided -- and, after all, they have the last guess you might say -- that the findings that we made can be said to be warranted on the evidence that was presented to us and that the findings that we made shall stand.

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I might illustrate that by a matter I read recently in the Medical Journal which said that in compiling statistics with respect to workers who were afflicted with lung cancer that there was one common incidence found among many of them over a survey made in going over a period of some 15 years that they had this in common that they had been or were engaged in industry in which chromates were handled.

Now, assuming that we had a case that came before us for determination, and that was the issue, whether or not the lung cancer that the employee had was causally related in any way to his employment and we did have evidence from a medical expert that he felt that the industry that he had been engaged in was related to it, and we made a finding, they would say that our findings were justified in arriving at that conclusion that the lung cancer has some relationship at least with the industry in which the employee was engaged in.

You must remember that in occupational diseases that many times a liability is a creature of statute. Of course, the Workmen's Compensation Law is a statutory law to begin with, but I think it can be said that the founders of our Workmen's Compensation Law had in mind primarily establishing liability because of traumatic injuries, and then, of course, it became evident, particularly as different substances were being processed in industry generally and it came to be noticed

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that certain diseases were common among workers in various types of industry and silicosis was more or less common among those in the granite industry and things of that kind that it could be reasonably be assumed that the injury there could arise in the course of employment, and as a result the Legislature amended the Act and said that if it could be found that the disease that the employee had was inherent in the nature of the employment that could be considered to be an injury arising in the course of the employment.

Then there was a later finding we made that the incidence of the employees' injury may be aggravated or reactivated a latent or inactive condition of tuberculosis that the employee had, and that was predicated on the words, "reasonable evidence," and that could be considered to be an injury arising in the course of employment.

Now, some jurisdiction is of such an inflexible character that as new diseases are found and characterized by the medical profession, we have to amend the Act and include them specifically. For example, in recent years we have beryllium poisoning and we have to include these different diseases specifically by statute.

Another important aspect is determining liability for an occupational disease. I suppose it can be said that medical science is still in the pioneering stage in determining

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the causes and the relationship of the industry to some of these diseases, and we can look forward to new fields such as in the development of atomic energy as they predict they are going to do, and there will be a degree of exposure to radiation with a causal connection; and, who knows, there may be more fields as yet unheard of that human ingenuity will come in contact with in later years.

As I see it, the proper administration of the Workmen's Compensation Act will always be dependent in a great measure upon the medical profession; and the worker, too, is dependent upon you a good deal, not only in regard to treatment that he is going to get but many times whether or not he will be entitled to monetary benefits that the Workmen's Compensation Act provides -- this will depend a great deal on the research that the medical profession makes and the opinions that they give to those of us entrusted with the administration of the Workmen's Compensation Act.

Thank you very much.

(Applause.)

CHAIRMAN CHAPMAN: Thank you very much gentlemen. This has been a most interesting audience which always gives a boost to the speakers.

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