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tion should be a tri-part members of this group, with Doctor Eiller and Doctor Peterson to formulate a definition under the aegis of individuals, specifically concerned with the use of terms. Then I place that responsibility upon Doctor Seward Eiller, Doctor Peterson, hoping they can arrange this committee.

(Adjournment of morning session 12:20 P.M.)

SUBJECT: THE INHALATION OF CERTAIN INDUSTRIAL SUBSTANCES

Chairman: Theodore F. Hatch — Monday, Sept. 22. 2:00 - 5:30 P. M.

Brief Reviews

Silica	Thomas M. Durkan
Asbestos	Arthur J. Vorwald, M. D.
Beryllium	Harriet L. Hardy, M. D.
Bauxite	C. G. Shaver, M.D. and Donald Solandt, M. D.

Discussion.

New Synethetic Silica — Philip C. Pratt, M. D.

The Fate of Inhaled Particulates — Merril Eisenbud

Discussion, led by — Theodore F. Hatch.

BY DOCTOR VORWALD:

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It's not easy to cover the topic of asbestos and asbestosis in the short time which we must, and I shall only try, and shall try to be very brief.

As you perhaps know, the disease asbestosis was first described about 1900 in England and since that time, there have been many, many reports concerning asbestos and notably those reports by Doctor Merewether in England, Doctor Lanza and McConnell and Fennel of the United - of the Metropolitan Life Insurance Company; by Doctor Lynch and others, and again by Doctor Lanza in the Monograph, published by the Oxford University Medical Publications, entitled "Silicosis and Asbestosis".

And, following these reports, it was thought that

BY MR. DURKAN:

Mr. Chairman, Members of the Symposium:

(Doctor Durkan read his prepared paper, which is on file at the Laboratory).

BY DOCTOR HATCH:

In view of the fact that our several subjects in this topic are expressed in several papers, it would be best to perhaps limit discussion and comments to the end of the presentation, so I call next on Doctor Vorwald who will duscuss the findings in reference to asbestos.

BY DOCTOR VORWALD:

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And, following these reports, it was thought that

we knew all about asbestosis, that there was nothing more that could be known. However, I don't believe that that view is substantiated and, certainly, all of us recognize that there are still many, many problems concerning the disease asbestosis, problems which are very complex and problems which must still be considered.

Therefore, in my remarks, I certainly do not wish to appear dogmatic in any way. I will merely try to point out some of the things which are established and seem to be established, and other things which appear to be valid in accordance with our present knowledge.

You all know what asbestos is. Of course, the name is not really one that refers to a definite specific mineral, but it rather is a term which is applied to a variety of several different substances occurring as - in the fibrous form, and one could mention crysotile and various other types of asbestos in accordance with a commercial term which has been given to them.

Now, the total number of persons engaged in the asbestos industry probably does not exceed 1,500. The number of such, at least in this country. The numbers of such workers inhaling asbestos dusts is not definitely known. However, industrial hygiene surveys and the frequency of pulmonary changes in such workers would indicate that only a few of the total number of men exposed to sufficient

quantities of asbestos fibers develop asbestosis. Thus, the condition differs considerably from the condition resulting from the present deposition of free crystalline silica. Certainly, it may go differently, the magnitude of the problem in one is different than in the other.

Now, it would appear that industrial environments that exist today necessitate generally ten or more years of exposure to asbestos dust in concentrations exceeding one million fibers longer than ten microns per cubic foot of air. It appears that way. This is basically subject to criticism and we know of other levels of permissible concentration that have been established for asbestos, but from our studies and our interpretation, and I repeat, it appears that industrial environments which exist today necessitate generally ten or more years of exposure to asbestos dust in concentrations exceeding one million fibers longer than ten microns per cubic foot of air.

Now, of course, exceptions to this occur. In subjects exposed to concentrations above that tentative permissible limit, and I recall to your mind, the statement brought out by Doctor Fletcher this morning, namely, the concentration. After all, if the concentration is high, well, then we find within the pulmonary tissue, many changes; so, too, with asbestos.

Now, an occasional case has come to us for study

with manifest pulmonary changes and profound symptoms, whose occupational history discloses an exposure of less than five years, but I again hasten to add in those instances, to an extremely high atmospheric concentration of long fiber asbestos. In view of the experimental studies, the pulmonary deposition of long fibers of asbestos seems to be the agent responsible for changes in the lung which are identified as asbestosis.

Rather than to cite all the experimental evidence, our evidence here at the Saranac Laboratory, and those by Smith, Woodin and King in England, have failed to show evident pulmonary change with fibers less than ten microns in length in our instance, and less than 2.5 microns in the instance of King and his workers, co-workers in England, and that, Whereas long asbestos fibers, in our experience, caused well-marked para-bronchiolar and interstitial resemblance, the condition, as we see it in human subjects exposed to asbestos.

Now, as you all know, the pulmonary changes due to the asbestos fiber occur initially as collars of fibrous tissue about the respiratory bronchials, and the progression of that fibrosis occurs and it is characterized by extention into the adjoining alveolar walls, which distorts those walls and distorts the formed alveolar space, giving rise often to what we identify as anatomical emphysema and

also to clinical manifestations of respiratory difficulty due, perhaps, to that emphysema and perhaps due to a diffusion phenomena. I shall not dwell upon that, because that is going to be a subject for discussion by Doctor Gregoire on Thursday.

The point to be made, however, is this, that asbestos manifests itself in an entirely different way than does asbestosis manifest itself in an entirely different way than does silicosis. Asbestosis is a diffuse pulmonary involvement, giving rise to the ground glass appearance in the lower lung, often involving the lower lung, manifested Roentgenographically. One condition, in our belief, is accompanied in many instances by respiratory difficulty, whereas the other condition, simple nodular silicosis, is often free of respiratory difficulty.

The action of inhaling asbestos fibers, I think the concensus of opinion today is, that it is a mechanical action, that the fiber being flexible, is inhaled into the repiratory bronchials and because of its flexibility, it can be taken up by a cell and it can be carried into the alveolar wall where it is deposited and produces its damage. Our experimental evidence seems to prove that since fibers which are brittle and just as long, glass wool fibers, which can be broken very readily, fail to produce this type of reaction which we see characterizes the pulmonary response

to the asbestos fiber. Thus we believe that the flexibility of the fiber is an important factor in thecapacity of the asbestos fiber to produce damage in the lung.

There are some, however, who believe that the chemical actions, that asbestos produces its effect by chemical action. Supposing that the minerological coating of mineral substances is removed, leaving bare the asbestos fiber or spicule, allowing silica - silicic acid to be liberated and thus producing damage. I think that that needs proof. I don't - I do not believe that silicic acid produces the damage. We have been unable to reproduce the experimental effect, or reproduce it experimentally.

The asbestos bodies -- sometimes I like to refer to it as commonly called, body - asbestosis bodies, as the asbestos body. Why? Because these bodies appear anywhere in the lung. They often lie free in the alveolar space. The body is coated with a layer of substance which gives a positive reaction to iron, which we and others believe is derived from tissue fluids. I like to look upon the asbestos body as a - a body which has been removed or is incapable of producing damage. This has been cited by other workers, notably, Colus, Lynch and others, and I like to refer to it as the asbestos body rather than the asbestosis body, because the presence of this body does not mean that asbestotic fibrosis is present in the lung.

We have seen asbestosis, asbestos bodies appear in the sputum, without evident change in the pulmonary tissue. Therefore, the asbestos body in the sputum is not diagnostic for the disease asbestosis. It merely means that the individual has inhaled an asbestos fiber and that the body has reacted thereto by coating it with iron.

I have seen asbestos bodies lying free in the alveolar space, without evident pulmonary change in the alveolar walls which form that space. I have seen asbestos bodies in the alveolar wall without evident fibrosis. So, therefore, I like to look upon the asbestos body as a body which is incapable of producing damage, that their presence means merely that the fiber has been deposited in the lung, and that - and that their presence in the sputum has no reference whatsoever to the degree or extent of fibrosis in the pulmonary area.

Asbestosis is productive of pulmonary disfunction and this, too, shall be a topic for discussion on Thursday. Suffice it to say for the present time that the symptomatology of asbestosis established as bestosis as dominated by phthismia and irritating cough. The complications of asbestos, inhalation of the asbestos dust, does not apparently alter significantly, the final outcome of tuberculosis, either experimentally or clinically.

I know that there are those of you who will take

exception to that statement. However, our clinic study of clinical patients, our study of experimental animals, infected with the tubercle bacillus, have failed to demonstrate that the asbestos fiber deposited in the lung increases the susceptibility of that tissue to infection by the tubercle bacillus, thus asbestosis is quite different then or from silicosis.

Inhalation of asbestos dust and carcinoma of the lung is also a subject for discussion on Wednesday and I shall not dwell upon that at this moment. There is often associated with asbestosis, well-established asbestosis, cardiac enlargement that seems to result from increased pulmonary tension.

Bronchiectasis, identified as the dry type, is frequently associated as a complication of asbestosis, and it is logical since the fibrosis of asbestosis gains about the respiratory bronchioles and about the bronchials, that there is a degree of fibrosis which interferes with the wall of the respiratory bronchiole and which will often cause a physiological disfunction, thus retention of secretions, thus also the appearance of bronchiectasis.

Experimental evidence with respect to asbestosis indicates, and also there is some clinical evidence, that on removal of the asbestotic from the - from the atmosphere, that there is appreciable clearing of the asbestosis of the

lesion in the lung, that, thus, it also differs from silicosis. Remove a man with silicosis from his - the atmosphere containing free crystalline silica, that silicosis will progress for a short period of time and then it will become stabilized and indeed, it may regress by reason of contraction of the fibrous tissue, but with the asbestotic, that individual, on removal from the dusty atmosphere, the disease in his lungs seems to stabilize very quickly, much more quickly than that does to the free crystalline silica particle.

As far as therapy is concerned, studies by the Saranac Laboratory and also by Smith, Woodin and King, experimental studies with aluminum hydroxide, colloidal aluminum hydroxide, showed no evident retention or retardation of the development of asbestosis in experimental animals. Thus, again, it differs from the reaction due to the free crystalline silica in the form of quartz.

Furthermore, this asbestosis, this aluminum hydroxide or aluminum, colloidal aluminum hydroxide did not prevent the development of the reaction to the asbestos fibers deposited in the lung. If anything, our experimental evidence, and that is also supported by the evidence from King in England and his co-workers, is that aluminum hydroxide augments the fibrosis due to the asbestos fiber.

Cortisone? I know of no experimental or clinical

study where Cortisone has been used as a possible therapeutic agent against the pulmonary reaction to asbestosis, to asbestotic fiber or for asbestosis. It seems to me, however, that Cortisone should be given a trial. We have considered that for silicosis, but by reason of the fact that the individual with free crystalline silica in his lung is more susceptible to tuberculosis, we have not favored the use of Cortisone which also increases the susceptibility of the lung to tuberculosis. But that does not appear to be the case in asbestosis.

We, therefore, consider giving Cortisone a trial in the asbestotic. Thank you.

BY DOCTOR HATCH:

I'm a little curious to know why, in settling the times for this afternoon, Doctor Vorwald has allotted more time for discussion of Beryllium. I thought Doctor Hardy could tell us all about it in five minutes, but instead of that, she is allotted a little longer time. I'll ask Doctor Hardy to talk on Beryllium.

BY DOCTOR HARDY:

Professor Hatch and Members of the Symposium: There has been in the past decade a truly impressive accumulation of American and foreign literature relating to the epidemiological, clinical and experimental aspects of exposure