baths do not injure the patient, then it seems to me that such patients who cannot take sun treatment, direct radiation from the sun, would be improved if given air baths continuously.

DR. SAMUEL H. WATSON, Tucson, Ariz.: In answer to the question about air baths, I will say that it has never been proved that the real effects that we get from the direct rays of the sun are actually due to the direct rays of the sun. It may be that the air bath is more important than the direct rays of the sun, and for some time I have been advising all of my patients to take air baths. They get enough reflective rays of the sun, so that they get a great deal of benefit and this method never does harm.

ASPERGILLOSIS OF THE LUNGS AND ITS ASSOCIATION WITH TUBERCULOSIS *

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Aspergillosis of the lungs is a disease caused by an exceedingly common mold, *Aspergillus fumigatus*. It is a disease which clinically and pathologically so closely resembles tuberculosis that it usually escapes recognition and is diagnosed as tuberculosis. This mistake may even be made at necropsies; for the ulcerations of the bronchi, the pneumonic and emphysematous areas, the cavities and pleurisies are much the same in one disease as in the other.

There is a common belief that this mold is purely saprophytic; that it can live only on preexisting lesions in the lungs; that it cannot attack living tissues because it has no primary pathogenic powers. To show its pathogenic properties, nine rabbits were inoculated intravenously with varying doses of spores, from a pure culture of *Aspergillus fumigatus* furnished by Dr. Thom of Washington.

In March, 1926, the pathologist of the Flagler Hospital, St. Augustine, Fla., inoculated three rabbits. One rabbit died in twenty, one in twenty-four and one in forty hours. The lungs were solid as in lobar pneumonia; the spleen, liver and kidneys were greatly congested; the mycelium was found growing in the lungs, liver, spleen and kidneys, and a pure culture of the mold was obtained from them.

March 24, three rabbits were inoculated at the Johns Hopkins pathologic laboratory. One rabbit died in five days. Gross lesions in the lungs were not conspicuous, but the liver and kidneys looked like miliary tuberculosis. Two weeks later the second rabbit was killed. The lungs, liver, spleen and kidneys presented the typical appearance of miliary tuberculosis. The third rabbit remained apparently well. April 1, three rabbits were inoculated at the Hygienic Laboratory, Washington. April 12 they were killed. The livers, kidneys and spleens of all the rabbits presented the typical appearance of miliary tuberculosis; the lungs of the second and third rabbits were solid, as in lobar pneumonia; those of the first rabbit were studded with tubercles as large as a grain of rice, and were typically tuberculous in appearance. In all the livers, spleens, lungs and kidneys, tubercles were abundant under the microscope.

In human beings there are two general types of aspergillosis of the lungs, just as there are in tuberculosis, the wet and the dry; of the wet, or parenchymatous, type, there are varieties corresponding to the location of the attack and the tissues chiefly involved. When the mucous membranes of the bronchi are attacked, they may become almost black from congestion; ulcerations may be eaten out, and patches of membranes formed. Pathologically and clinically, this is the bronchitic type of pulmonary aspergillosis. An emphysematous type was shown in the case of a farmer, aged 21, who began suffering from crises of dyspnea while apparently perfectly well.[1] Four months later he died in a crisis of suffocation. The lungs were highly emphysematous and completely covered the heart. The spores of the mold had been carried down into the sacs of the alveoli, where they had begun their development and then spread on up into the smaller bronchi, eating them out as they went, until the lungs were riddled with microscopic cavities.

A child, aged 2½ years, died of pneumonia twenty-seven days after the illness began.[2] The lungs were like sieves, they were so filled with tiny cavities connected with the bronchi, while posteriorly there was a large cavity directly connected with a large bronchus.

In another case of supposed pneumonia, five centers of growth of *Aspergillus fumigatus* had spread out into circular patches from 2 to 5 cm. in diameter, and these were surrounded by areas of hepatization and compensatory emphysema which gave the appearance of chronic pneumonia.[3]

A woman, aged 71, died in a crisis of suffocation.[4] The pulmonary artery was distended to 5 cm. and stuffed with a growth of *Aspergillus*, and with clots and thrombi. The growth had crept up to the root of the left lung and followed the descending vessels down to their termination, plugging and obstructing them and causing necrosis of their terminal areas. The mycelial filaments had everywhere plunged through the walls of the blood vessels into the surrounding tissues, causing necrosis and cavity formations. Areas of surrounding hepatization and emphysema confirmed the diagnosis of chronic pneumonia, which was accepted until the growth of *Aspergillus fumigatus* was found in the blood vessels.

In another case,[5] a soft, spongy, purulent mass as large as an apple was found in the upper anterior part of the lung. The vessels leading to this area were obstructed by a growth of *Aspergillus fumigatus*, which had formed plugs, clots and thrombi.

In none of these cases of supposed pneumonias had the diagnosis of aspergillosis of the chronic pneumonic type been made during life.

In the pleuritic type, the spores are carried to the periphery of the lung, become deposited there, and attack the pleural surfaces, causing congestion, thickening, toughening and sometimes bands of adhesions, conditions very familiar to those attempting the production of an artificial pneumothorax.

In this pleuritic type of aspergillosis, pain is the first, and often the only symptom. In 1923, a woman, aged 40, had a severe attack of pleurisy of the right base, with such severe pain that hepatic colic was diagnosed.[6]

* Read before the Section on Pathology and Physiology at the Seventy-Seventh Annual Session of the American Medical Association, Dallas, Texas, April, 1926.

1. Arkle and Hinds: Pneumomycosis, Tr. Path. Soc. London, May 9, 1896.
2. Wheaton: Tuberculosis with Aspergillus Niger, Tr. Path. Soc. London, May 20, 1890.
3. Weichselbaum: Eine Beobachtung von Pneumomycosis aspergillina, Wien. med. Wchnschr., 1878, No. 49, p. 1289.
4. Macaigne and Nigaud: Aspergillose primitive du poumon avec artérite pulmonaire oblitérante, Bull. et mém. Soc. méd. d. hôp. de Paris, Feb. 11, 1926, p. 183.
5. Kohn: Ein Fall von Pneumomycosis aspergillina, Deutsch. med. Wchnschr., 1893, no. 50, p. 1332.
6. Miéres, J. F.: Caso de seudo-tuberculosis pulmonar causada por el Aspergillus fumigatus, Semana méd. 1: 370 (Feb. 12) 1925.

After a year of suffering, an operation for gallstones was followed by a very painful attack of pleurisy. Two months later she had an attack of acute bronchitis with high fever and bloody sputum. In 1924, she had a violent hemorrhage. The sputum had always been negative to tubercle bacilli, but the diagnosis of tuberculosis was made because the clinical, physical and roentgenographic observations supported it. She improved in the mountains until another violent hemorrhage came on; then guinea-pigs were inoculated and proved to be negative to tuberculosis, while the sputum was positive to *Aspergillus,* so she was put on iodides and made a good recovery.

Another case of acute pleurisy followed by acute bronchitis in a woman, aged 40, was reported by Castrillón.[7] It began with a violent attack of pleurisy, with chills and fever. Fifteen days later she began to cough continuously and to suffer from crises of dyspnea. The bronchitis extended from the largest to the smallest bronchi, and the noisy, wet râles drowned out all other sounds. The sputum was negative to tubercle bacilli but positive to *Aspergillus,* so she was put on iodides and made a rapid recovery.

The asthmatic type is rather characteristic of aspergillosis of the lungs. In one of my cases a celebrated specialist could obtain only a test reaction with dust. Was this because the dust contained spores of *Aspergillus?* The sputum was positive to *Aspergillus.*

The insidiousness and the latency of the interstitial type of aspergillosis of the lungs makes the course of the disease very like the same type of tuberculosis. It occurs when the spores pass through the walls of the alveoli and begin their growth within the interstitial tissues, very much in the same way as do the bacilli of Koch. Hildebrandt[8] describes the defensive gathering of the tissue cells, the lymphocytes, leukocytes and macrophages, to build an imprisoning wall around the invaders and shut them in by tubercle formation. Just as it may happen in tuberculosis, these tubercles may remain discrete or become conglomerated; may vary in size from invisibility to nodules as large as a nut, and may become sclerosed by connective tissue infiltrations. The future course of these invasions, their clinical, physical and roentgenographic manifestations, are so similar to those of tuberculosis that it may not be possible to make a differential diagnosis without inoculations and cultures. Tuberculin is of no diagnostic value whatever because aspergillosis gives precisely the same reaction as tuberculosis.

There are cases of aspergillosis on record which subsequently became tuberculous, and Rénon says that this is a dangerous feature of the disease. While it is not generally believed that the development of tuberculosis is in any way associated with a disease so rare that it is a curiosity, how do we know that this disease is so infrequent? Because we do not recognize it is no reasonable proof that it does not exist. Suppose we should make cultures from the sputum of every case of tuberculosis, how many cultures would be positive to *Aspergillus?* I am confident that the percentage would be higher than we could believe. During the last three years I have had ten cases of positive cultures which were sent to me as cases of tuberculosis. If ten cases of aspergillosis are found in three years in an obscure village on top of the Blue Ridge Mountains, how many cases would be found if routine cultures were made from the sputum of every case of tuberculosis? Such a search would inform us as to the frequency of the disease, its possible danger as a cause predisposing to the development of tuberculosis, and its influence on recovery. Here is a disease that is capable of causing pleurisy, acute and chronic; bronchitis, acute and chronic; pneumonias, acute and chronic; emphysema; bronchiectasis; atelectasis; sclerotic fibrosis; tubercles; cavities; endarteritis; thrombosis; infarcts; hemorrhages; asthma. Would it be strange if such a disease should seriously complicate or even inhibit recovery in a case of tuberculosis?

7. Castrillón, B. A., and Borsani, E. P.: Micosis aspergilar, Semana méd. **1**: 924 (April 23) 1925.

8. Hildebrandt: Experimentelle Untersuchungen über des Eindringen pathogener Microorganismen von den Luftwegen und der Lungen, Dissertation, Königsberg, 1888.

SUMMARY

We are thoroughly ignorant of the frequency of aspergillosis both as a primary and as a secondary disease.

We have no idea how much aspergillosis of the lungs predisposes to tuberculosis in human beings or in cows.

We do not know how much it impedes or even inhibits recovery in cases of tuberculosis.

We have no idea whatever as to its association with acute respiratory diseases.

We know that it affects cows much as it does human beings.

We know that it gives the same reaction to tuberculin that tuberculosis does.

Should we apply this knowledge to the study of the tuberculosis of dairy herds?

In order to gain adequate information as to the frequency and importance of this disease, should not a systematic research study be made:

1. By determining the percentage of aspergillosis cases among the tuberculosis cases in the large tuberculosis sanatoriums.
2. By determining the percentage of cases of aspergillosis in the lungs at necropsies.
3. By determining the percentage of cases of aspergillosis in cases of respiratory diseases.
4. By applying these principles to dairy herds.

ABSTRACT OF DISCUSSION

DR. HENRY C. SWEANY, Chicago: I am greatly interested in this report on aspergillosis. It seems to me that the similarity of this type of disease to tuberculosis is not unusual. Nature has very few methods of reacting to different types of foreign protein and different types of foreign bodies. When there is a foreign body of the nature of tubercle bacilli or aspergillosis fungus, the same type of cell comes out and causes the reaction that is so characteristic, and perhaps this is the reason it is so difficult to differentiate between aspergillosis and tuberculosis. In our sanatorium in Chicago we have performed about 175 necropsies in chronic tuberculosis cases. Grossly and miroscopically we have not been able to find anything resembling aspergillosis. The question comes to my mind whether the climatic conditions or locality do not have a great deal to do with it. We know of the work of Castellani, who states that this type of fungus growth is far more prevalent in certain regions than it is in others. As to the predisposition to tuberculosis, there is no doubt that any condition of this nature would predispose to tuberculosis. Anything that will lower the vitality of the human body and bring it down below par is bound to allow the tubercle bacilli to become active. Perhaps in our sanatorium we have overlooked them. Perhaps there are many cases in which the two coexist. Were these parasites producing pathologic changes in rabbits from pathogenic or wild aspergillosis fungi found on bread and so forth?

DR. KENNETH M. LYNCH, Dallas, Texas: As Dr. Lapham indicated, *Aspergillus fumigatus* is a ubiquitous organism,

and this fact must be considered fully in interpreting a report of this kind. I want to encourage Dr. Lapham to further effort toward finding the tubercle bacillus. I can readily see how the sputum of many persons may contain *Aspergillus fumigatus,* possibly without relation to disease, possibly as secondary or accidental factors. I may be old fashioned in my ideas about aspergillus infections, and my respect for this organism has been increased by some recent experiences with it in scalp lesions. Even here, however, it probably was inoculated by scratching. I have had no personal experience with aspergillosis of the lung, but I should expect *Aspergillus fumigatus* to be commonly found in open tuberculosis of the lung, and I think we must be positive about the absence of tuberculosis before saying that such conditions are aspergillosis.

DR. ALDO CASTELLANI, New Orleans: For some years I have been working at various bronchomycoses. There are two types of aspergillus mycosis, the same as any other mycosis. There is the primary type and the secondary type. The aspergilli found in cases of tuberculosis are, as a rule, secondary invaders; but, as Dr. Lapham has pointed out, there is no doubt whatever that there is a primary aspergillus bronchomycosis. Several cases have been put on record by Veedal and Brownee, myself and others. The first paper I think called attention to the fact that this condition may be common in certain districts and certain countries. I quite agree with him. The condition is common in certain parts of France and certain parts of Italy. It is common among a certain class of workers, the so-called pigeon breeders. I do not see how the pigeon breeder fattens his or her pigeons. The pigeon breeder will fill his mouth with various grains. Then he insufflates the grains into the throat of the pigeon. The grains contain a large number of spores of fungi, principally aspergilli, but the aspergillus spores are the commonest of all. These spores for a time invade the pharynx, larynx, bronchi and lungs. The bronchopulmonary type of disease varies. I have seen several of these cases, and clinically they certainly resemble tuberculosis. In bad cases the patient brings up mucopurulent sputum with occasionally some blood. At times a true mycosis takes place. In both cases percussion will reveal all the symptoms of ulcerative tuberculosis, and even roentgen-ray examination will not help much to differentiate. These cases have a very bad prognosis. The moment the diagnosis of broncho-aspergillus mycosis is made, the only thing to do is make the patient, if he is a pigeon breeder, give up his work and make him have a change, a change of climate or a change of air. In cases of medium gravity, occasionally potassium iodide gives very good results, but in bad cases it is no good.

DR. MARY E. LAPHAM, Highlands, N. C.: The diagnosis of secondary aspergillosis is suggested by the appearance of membranes, but primary aspergillosis is apt to be overlooked because membranes are not always formed and mycelium may not be found in the tissues, and the spores remain unsuspected. The tubercles are so similar both macroscopically and microscopically to those of Koch's bacillus that the diagnosis of tuberculosis is made. The cultures were furnished by Dr. Thom of the Department of Agriculture, Washington, D. C. A positive culture from the sputum does not prove that there is aspergillosis of the lungs, because these spores are probably more or less constantly present in the air and therefore in the lungs. Agglutination tests may be more convincing. As Dr. Lynch says, the aspergillus lesions of the scalp might be due to scratching, because aspergillus spores are often found beneath the nails and often cause inflammatory conditions. Since aspergillus spores are often present in the air, a loss of immunity might at any moment lead to an attack of aspergillosis. Studies of this disease might prove that it is far more common than we suppose and that it is to be feared as a common predisposing cause of tuberculosis. It would be highly desirable to know how many cases of tuberculosis develop on aspergillosis. When a case of tuberculosis is complicated by aspergillosis, recovery seems to be rendered more difficult and more unstable because there is a decided tendency to relapses. If I had a simple infection of my lungs by tubercle bacilli I should not be as anxious about my recovery as I would if it were complicated by aspergillus processes. Good recoveries are made in primary cases of aspergillus, but the tuberculous cases complicated by aspergillosis have not done well. Dr. Castellani mentioned the use of iodine. Unfortunately, the sympathetic nervous system is often too irritable to permit its use. In these cases, sodium cacodylate may give good results.

ROENTGENOTHERAPY OF EXUDATIVE IRITIS *

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AND
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Our experience in the roentgen-ray therapy of iritis of the exudative type dates back about seven years. The first observation made was in a case of iritis supposedly of traumatic origin, and roentgen-ray exposure was made to determine the presence of a foreign body. There was a large fibrinous exudate in the anterior chamber, and it was noted that rapid disappearance of this spongy material followed roentgen-ray exposure. This happened without the administration of internal treatment, there being used here only mydriatics to keep the ciliary body at rest and to dilate the pupil fully. However, in the light of continued experience, similar clinical pictures, by the use of similar light, stimulating doses of roentgen rays, administered at the direction of one of us (H. N. B.), not by one laboratory but by several different laboratories, all resulting in the same marked amelioration of symptoms with prompt, eventually complete resolution of the lesion, it is felt that there is here a definite contribution to be made to both ophthalmology and roentgenology.

The cases of iritis that have been treated were of the exudative type, variously classified in the different textbooks as spongy iritis and croupous iritis. The causation in the majority of cases has been syphilitic.

The patients have shown the usual signs of severe iritis, with deep or pericorneal injection and narrowed, fixed pupils. The markings of the iris have been indistinct, and the characteristic feature has been the formation of an exudate in the anterior chamber. When the pupil has been dilated with a mydriatic, this exudate has been seen on the anterior capsule of the lens, in the pupillary space. The severity of these cases does not seem to be as marked as in those of the plastic type, as the pupil is more ready to respond to mydriatics and there is less tendency to the formation of posterior synechiae.

The latest patient who has been treated had a late postoperative infection, following cataract extraction. There was a hypopyon of several millimeters, with a yellowish exudate in the pupillary space and a threatened infection of the vitreous, especially at the height of the disease. The iridocyclitis was so severe and the consequent deposit on the posterior wall of the cornea so heavy that the iris was seen only with difficulty.

The usual therapeutic agents were employed in this case: continuous hot applications, mercury inunctions, leeches to the temple, milk injections, intravenous administration of a 1 per cent solution of mercurochrome-220 soluble, all without avail, and finally the

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