Conference on Pellagra : held under the auspices of the State Board of Health of South Carolina at the State Hospital for the Insane, October 29th, 1908.

from J. W. Babcock

CONFERENCE ON

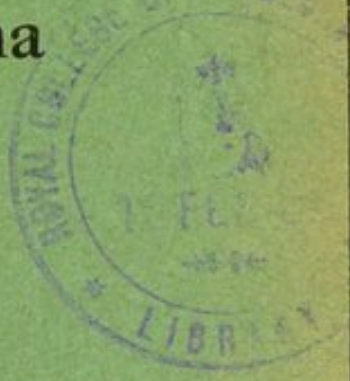
Pellagra

Held Under the Auspices of the

State Board of Health

of South Carolina

At the



State Hospital for the Insane

October 29th, 1908

Columbia, S. C.
THE STATE CO., PRINTERS
1909

PROGRAMME

OF THE

Conference on Pellagra

HELD AT

The State Hospital for The Insane

IN COLUMBIA, S. C., OCTOBER 29, 1908

UNDER THE AUSPICES OF

The South Carolina State Board of Health

Invocation—The Rev. S. M. Smith, D. D.

Welcome to Visitors—Gov. Ansel on Behalf of the State, and Dr. W. W. Ray on Behalf of the Board of Regents of the Asylum.

"Remarks Upon Pellagra as Seen in Italy"—Senator Tillman.

Opening Address—Passed Assistant Surgeon C. H. Lavinder, Public Health and Marine Hospital Service of the United States.

Clinic, Examination of Cases in the Hospital and Cases Presented—By Dr. Neuffer of Abbeville, Dr. Frontis of Ridge Spring and Others.

Discussion on Causation of Pellagra

(1) "Theories of Its Etiology"—N. M. Moore, M. D., of Augusta.

(2) "Some Problems in the Study of Its Etiology"—Edward J. Wood, M. D., of Wilmington, N. C.

(3) "The Maize Theory of the Italians"—J. J. Watson, M. D., of Columbia.

(4) "The Roumanian Theory"—J. L. Thompson, M. D., State Hospital, Columbia.

(5) "The Trypanosome Theory"—J. H. Taylor, M. D., Columbia.

(6) "Personal Experience With Some Cases of Mental and Nervous Diseases Showing the Pellagra Syndrome"—I. M. Taylor, M. D., Superintendent Broadoaks Sanitarium, Morganton, N. C.

(7) "Observations on Pellagra in This Country With Special References to Pellagrous Insanity"—John M. McCampbell, M. D., Superintendent State Hospital for Insane, Morganton, N. C.

(8) "Pellagra in Chester County"—H. E. McConnell, M. D., Chester, S. C.

(9) "The Supposed Relationship of Damaged Grain to Epizootic Cerebro-Spinal Meningitis of Horses"—M. Ray Powers, D. V. S., Clemson College.

(10) "The Pathology of Pellagra"—H. H. Griffin, M. D., State Hospital, Columbia.

(11) "Eye Symptoms of Pellagra"—E. M. Whaley, M. D., of Columbia.

(12) "The Diagnosis and Treatment of Pellagra and Pellagrous Insanity"—J. W. Babcock, M. D., Superintendent State Hospital, Columbia.

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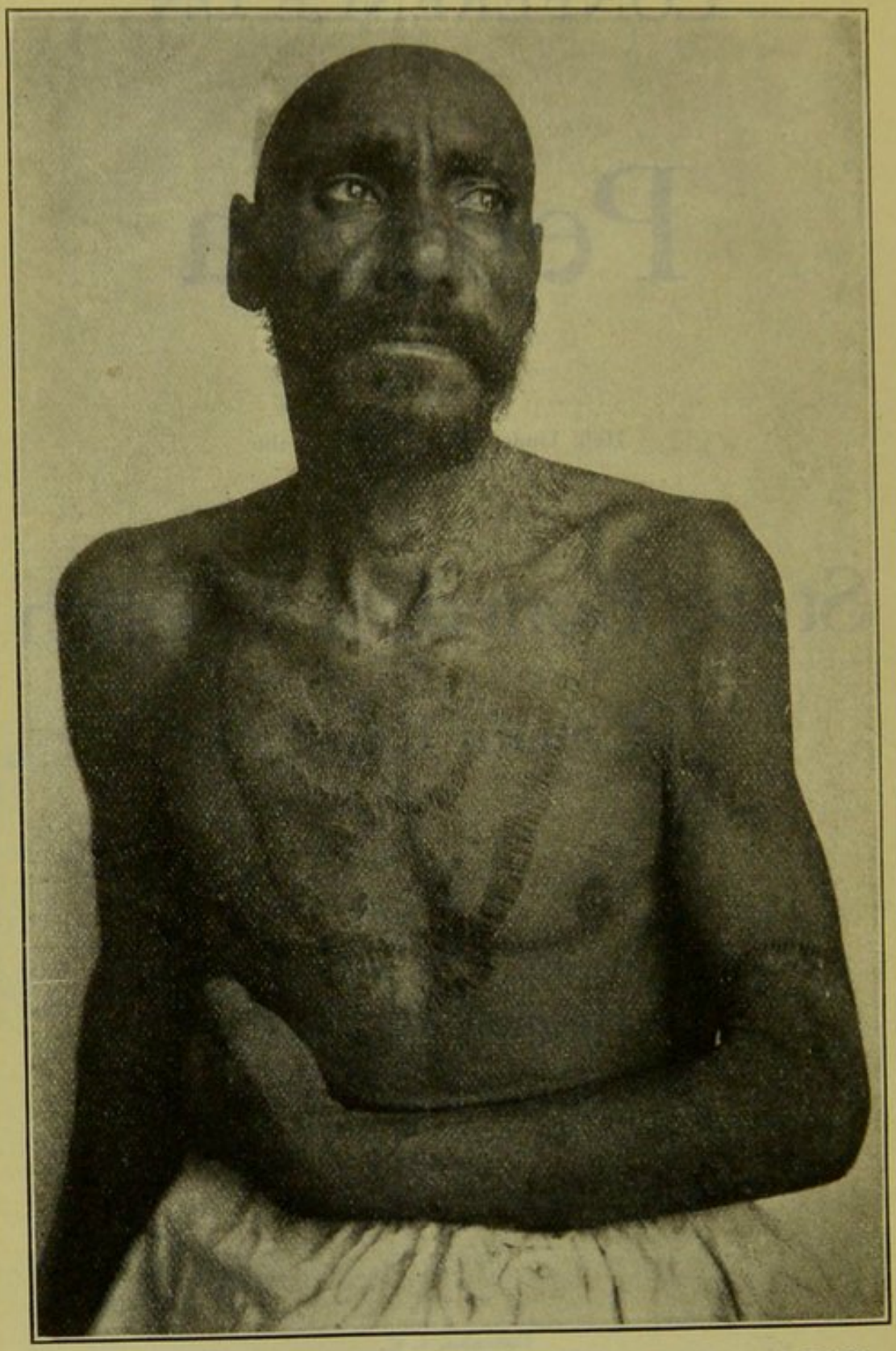

CASE OF PELLAGRA, CAIRO, EGYPT. COURTESY OF DR. F. M. SANDWITH, LONDON.

Conference on Pellagra.

INTRODUCTION.

In the report of 1907 of this board a preliminary report was included, made by the medical officers of the State Hospital for the Insane, upon the suspected presence of pellagra in South Carolina.

Subsequent experience at the State Hospital, and among a number of the members of the medical profession throughout the State, gradually confirmed the tentative views expressed in the preliminary report. Several calls were received by this office for consultation in cases of suspected pellagra. Upon inquiry, I learned that other cases had been seen in consultation by other physicians; notably Drs. Babcock and Watson, who had made a special study of the disease as it is observed in Italy, and identified the Italian form of pellagra with that existing in our State.

By the early fall sufficient data were at hand to justify the board in contemplating a thorough investigation of the pellagra problem.

At the regular quarterly meeting of the executive committee of the State Board of Health, on October 15th, by request Dr. Babcock was present, and informed the committee that a sufficient number of cases were then in the State Hospital to furnish material for an interesting clinic on pellagra. It was left to Dr. Babcock and the secretary to arrange for the meeting, should they find it feasible in the limited time. After due deliberation it was decided to take advantage of the clinical material at the State Hospital as a basis for a conference, to which invitations were extended to physicians in South Carolina, North Carolina, Georgia and other Southern States. Additional reasons for holding such a meeting were found in the fact that two important medical meetings were to be held in Columbia during Fair Week. The committee, therefore, arranged a definite program, and the conference was called for October 29th, 1908.

CONFERENCE ON PELLAGRA.

Thursday, October 29, 1908.

The conference was called to order at 3:30 p. m., in the amusement hall of the State Hospital for the Insane, by Dr. Robt. Wilson, Jr., Chairman of the South Carolina State Board of Health. After

the invocation by the Rev. S. M. Smith, D. D., addresses of welcome were delivered by Governor Ansel on behalf of the State, and by Dr. W. W. Ray on behalf of the Board of Regents of the State Hospital for the Insane. Senator Tillman being introduced by the chairman, described the visit he had made with Dr. Babcock to study pellagra at the Provincial Asylum at Mombello, near Milan, Italy.

Then followed the address by Surgeon C. H. Lavinder, of the United States Public Health and Marine Hospital Service. Dr. Lavinder explained that his presence was due in part to an invitation from the committee, but also because of the interest his Service was taking in pellagra. His address was informal and reviewed the opinions he had expressed in his recent monograph*, published by the government.

A clinic was then given on the several patients of the State Hospital, and cases presented by Dr. Neuffer, of Abbeville, Dr. Frontis, of Ridge Spring, Dr. Lancaster, of Columbia, and Dr. Bailey, of Clinton. The following program was then carried out:

DISCUSSION ON CAUSATION OF PELLAGRA.

(1) "Theories of Its Etiology"—N. M. Moore, M. D., of Augusta.
(2) "Some Problems in the Study of Its Etiology"—Edward J. Wood.
(3) "The Maize Theory of the Italians"—J. J. Watson, M. D., of Columbia.
(4) "The Roumanian Theory"—J. L. Thompson, M. D., State Hospital, Columbia.
(5) "The Trypanosome Theory"—J. H. Taylor, M. D., Columbia.
(6) "A Theory as to the Cause of the Recent Appearance in this Country of Pellagra"—John McCampbell, M. D., Superintendent State Hospital for Insane, Morganton, N. C.
(7) "Personal Experience with Some Cases of Mental and Nervous Diseases Showing the Pellagra Syndrome"—I. M. Taylor, M. D., Superintendent Broadoaks Sanitarium, Morganton, N. C.
(8) "Pellagra in Chester County"—H. E. McConnell, M. D., Chester, S. C.

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*Pellagra. A Précis. By Passed Assistant Surgeon C. H. Lavinder, Treasury Department, Public Health and Marine-Hospital Service of the United States. Prepared by direction of the Surgeon-General. Washington Government Printing Office. 1908.

(9) "Report of Three Cases of Pellagra in One Family"—Dr. D. B. Frontis, Ridge Spring, S. C.

(10) "Four Cases of Pellagra"—G. A. Neuffer, M. D., Abbeville, S. C.

(11) "A Case of Pellagra"—Dr. R. A. Lancaster, of Columbia.

(12) "The Supposed Relationship of Damaged Grain to Epizootic Cerebro-Spinal Meningitis of Horses"—M. Ray Powers, D. V. S. Clemson College.

(13) "The Pathology of Pellagra"—H. H. Griffin, M. D., State Hospital, Columbia.

(14) "Eye Symptoms of Pellagra"—E. M. Whaley, M. D., of Columbia.

(15) "The Diagnosis and Treatment of Pellagra and Pellagrous Insanity"—J. W. Babcock, M. D., Superintendent State Hospital, Columbia.

As these papers are given in the order in which they were read, comment is unnecessary. At the conclusion of the papers, remarks were made by Dr. I. W. Faison, of Charlotte, N. C., and Dr. J. P. Monroe, of Davidson, N. C. During the meeting a motion regarding the publication of the transaction was made and carried, and another motion that a committee of nine be appointed by the chairman, including himself, to consider the organization of a society for the study of tropical diseases.

The following physicians were present during the whole or a portion of the conference:

Allan, Sarah C., Charleston, S. C.
Babcock, J. W., Columbia, S. C.
Baker, Mary R., Columbia, S. C.
Baker, S. C., Sumter, S. C.
Brailsford, H. M., Mullins, S. C.
Burdell, W. J., Lugoff, S. C.
Burman-Wildman, Matilda, Columbia, S. C.
Cheyne, Walter, Sumter, S. C.
Coleman, D. A., Blackstock, S. C.
Connor, P. M., North, S. C.
Dawson, J. L., Charleston, S. C.
DuRant, J. B., Lake City, S. C.
Dwight, F. M., Wedgefield, S. C.
Epting, R. B., Greenwood, S. C.
Faison, I. W., Charlotte, N. C.
Frontis, D. B., Ridge Spring, S. C.
Furman, Davis, Greenville, S. C.
Gibbes, Robt. H., Columbia, S. C.
Gregg, B. G., Florence, S. C.
Gregory, G. W., Jefferson, S. C.
Griffin, H. H., Columbia, S. C.
Guignard, Jane Bruce, Columbia, S. C.
Hall, H. T., Aiken, S. C.
Hamilton, J. H., Union, S. C.
Harmon, S. E., Columbia, S. C.
Hay, W. S., Allendale, S. C.
Irwin, J. R., Charlotte, N. C.

Jervey, J. W., Greenville, S. C.
Lancaster, R. A., Columbia, S. C.
Lancaster, S. T. D., Spartanburg, S. C.
Lavinder, C. H., United States Marine Hospital Service.
Lester, W. M., Columbia, S. C.
Lindsay, Samuel, Winnsboro, S. C.
McCampbell, Jno., Morganton, N. C.
McConnell, H. E., Chester, S. C.
Miller, J. R., Rock Hill, S. C.
Monroe, J. P., Davidson, N. C.
Moore, Fillmore, Aiken, S. C.
Moore, N. M., Augusta, Ga.
Mower, F. D., Newberry, S. C.
Nardin, W. H., Anderson, S. C.
Neil, G. P., Greenwood, S. C.
Neuffer, G. A., Abbeville, S. C.
Parker, M. L., Wedgefield, S. C.
Parrott, W. T., Kinston, N. C.
Pelham, W. E. Jr., Newberry, S. C.
Pixley, C. S., Winnsboro, S. C.
Pope, T. H., Newberry, S. C.
Pryor, S. W., Chester, S. C.
Ray, W. W., Congaree, S. C.
Redfern, A. M., Clemson College.
Rice, H. W., Columbia, S. C.
Roberts, G. F., Lexington, S. C.
Ross, J. K., Charlotte, N. C.
Shecut, L. C., Orangeburg, S. C.
Shipley, Anna C., Columbia, S. C.
Sosnowski, J. C., Charleston, S. C.
Taylor, I. M., Morganton, N. C.
Taylor, J. H., Columbia, S. C.
Taylor, J. T., Adams Run, S. C.
Thompson, J. L., Columbia, S. C.
Wannamaker, J. F., Vance, S. C.
Wannamaker, T. E., Cheraw, S. C.
Watson, J. J., Columbia, S. C.
Weston, Wm., Columbia, S. C.
Whaley, E. M., Columbia, S. C.
Williams, C. F., Columbia, S. C.
Wilson, G. deFoix, Spartanburg, S. C.
Wilson, Robt., Charleston, S. C.
Wyche, C. T., Prosperity, S. C.
Wyman, H. H., Aiken, S. C.
Young, Robt. S., Concord, N. C.

ETIOLOGY OF PELLAGRA.*

By Noel M. Moore, M. D., Augusta, Ga.

My experience in observing cases of pellagra has been so limited that I do not feel at liberty to discuss its prevalence in this country further than to say that I am convinced by the reports published during the past year from various Southern States that we have quite a serious problem to contend with. So much so that a consideration of its prevention at this time is of great importance.

Although empirical prophylactic measures have been successfully employed in the past against certain diseases, still a positive knowl-

*Printed in Journal of South Carolina Med. Association, November, 1908.

edge of the etiology of any disease renders these preventive measures much more certain and effectual. Considering the etiology of pellagra, we find that many different theories have been advanced. Toxic substances found in damaged maize, various fungi, and other organisms have been suggested as the causal agent, but farther investigations as to the actual etiological factor are certainly warranted. As Dr. Babcock has suggested, the association with Indian corn is too constant to be ignored; and yet, certain resemblances between pellagra and some of the protozoal diseases suggest, according to Sambon, the possibility of damaged maize bearing a somewhat similar relation to pellagra that stagnant water does to malaria. As to a causal relation existing between damaged maize and pellagra, Dr. C. H. Lavinder has said: "Students of pellagra seem generally to be in accord, but it must not be overlooked that this relation is by no means definitely understood."

Of the various moulds suggested as the cause of pellagra, the common blue mould or penicilium crustaceum is the one most often considered as responsible for the disease. This fungus grows, under favorable conditions, on cheese, fruit, bread, and various grains, so it is difficult to understand why it should produce pellagra when grown on corn and not when grown on other foods.

Various toxic substances supposed to be formed in damaged maize by a fungoid growth have been assigned as the cause. Dr. R. H. Bellamy quotes Lombroso as claiming that a fatty oil found in damaged maize is the toxic principle. He is quoted as having produced symptoms, analogous to those of pellagra in man and other animals, by feeding them upon an extract obtained from damaged maize. It would be very interesting to know whether only some of the symptoms of pellagra were produced or whether the typical disease, with exacerbations recurring each spring, resulted. Of course, this statement coming from such an authority must have great weight; and yet there are certain facts difficult to reconcile with this theory. For instance, it is generally agreed that usually in the chronic cases improvement in the symptoms occurs during the fall and winter months, only to be followed by a relapse during the following spring. Why, then, if the cause is simply a toxaemia from a fatty oil, should it tend to this periodic reproduction? The other grain intoxications, although giving rise to severe nervous and gastro-intestinal symptoms, do not show this tendency.

The fact that pellagrous patients admitted to hospitals tend to have a recurrence of the trouble during the following spring, even though

maize has been excluded from their diet, would tend to negative a toxæmia as the sole cause of the condition.

It has been claimed by good authorities that pellagra only occurs in those who have worked in the fields, and never in those who have lived exclusively in cities, even though their diet consists partly of damaged maize. This observation, if proven correct, would have a most important bearing upon the subject, and in future reports of cases it would be well if this point in the history could be established.

These observations, accepted as facts by so eminent an authority as Sambon, have led him to suggest that pellagra may prove to be a protozoal disease, possibly contracted in the maize fields through the medium of some biting fly. Bearing this, at least plausible hypothesis, in mind, further studies should be made in an effort to solve this problem. I have recently heard several farmers make the statement that they experienced a very severe inflammation as the result of the exposed parts of their bodies coming in contact with the fodder while working in the corn field. From their statement, the local reaction seemed to be more severe than one would ordinarily expect from a mere abrasion. I am unable to state whether this experience is common among field laborers or not, but if so, it would suggest the possibility of the infection occurring in this way, primarily through these skin lesions.

It should be remembered that in some of the protozoal diseases, as in kala-azar, the organisms seldom appear in the peripheral circulation. As autopsy findings in cases of pellagra almost uniformly show some trouble with the cerebro-spinal system, and as the nervous symptoms are so prominent during the course of the disease, it would seem that examination of the cerebro-spinal fluid might offer an interesting field for further investigations.

The object of this paper is not to discourage prophylactic measures directed against the ingestion of damaged maize, which efforts I believe to be most desirable, but rather to encourage further study, so that if pellagra should be proven to be a protozoal disease, possibly even more effective preventive measures could be inaugurated.

PELLAGRA—SOME PROBLEMS IN THE STUDY OF ITS ETIOLOGY.*

By Edward J. Wood, M. D., Wilmington, N. C.

Too much has been taken for granted in the study of the etiology of pellagra. The problem before us today is all important and we cannot expect to make any progress until order is brought out of the present chaotic state of knowledge and theory. Why should we accept the corn theory so readily? I believe that the corn plays a part, but I have no right to such a belief. Dr. Lavinder since writing his excellent monograph has found a definite case of pellagra in New York, and he writes me that the patient acknowledges eating corn food only once or twice a week. I have now a case of pellagra in a German woman who seldom eats corn food. Think of the statement that amounts to this: "Psuedo-pellagra is pellagra occurring in an individual who does not eat Indian corn"! The whole thing is cloaked in as much mystery as was the etiology of malaria before the discovery of the plasmodium, and yet what medical student does not know thoroughly how to recognize the parasite that made Laveran famous? I acknowledge that pellagra and corn have been too universally associated to allow us to pass the question over lightly, but we have not sufficient evidence to condemn the corn. The greater portion of the corn consumed in Wilmington, North Carolina, comes from Virginia and Ohio. We have certainly had 30 cases of pellagra. I have carefully gathered three samples of what I had reason to suspect was bad corn meal. The report from Dr. Stevens, of the North Carolina Experiment Station, is:

No. I.

Penicilium	30 per cent.
Aspergillus	60 per cent.
Syncephalstrum	10 per cent.

No. II.

Aspergillus	70 per cent.
Penicilium	20 per cent.
Chaetomium	5 per cent.
Syncephalstrum	5 per cent.

No. III.

Mucor	5 per cent.
Penicilium	45 per cent.
Aspergillus	50 per cent.

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Dr. Stevens says that he would regard all of this meal as suitable for food of a good average quality. Each of these specimens was kept for three months by me in a damp place under conditions most unfavorable. If corn meal can be harmed by any outside influence this should be bad. Is it not possible that there is some specific organism that finds a suitable place for its history in damp corn meal? If so this organism must be resistant to high degrees of temperature, or else must be a wonderful toxin producer.

Two organisms have been isolated from corn. The first is a short bacillus producing an orange yellow growth on agar-agar, staining by Gram's method and forming no spores. The cultures are abundant and gelatinous. It does not produce gas. The organism is not pathogenic. The second bacillus forms spores. On agar-agar it produces faint round colonies which also grow on glycerine agar. It liquefies gelatine. This baccillus is not pathogenic to rabbits. It corresponds with the bacillus mesentericus, or to the bacillus maidis of Heider and Peltauf. This bacillus could be isolated from corn meal and corn bread. It occurred in the feces of pelligrins, but has also been found in the feces of individuals who did not have the disease.

Majocchi, in 1881, described the bacillus maidis as a very motile organism which he found in the blood of pellagrins in the beginning of the disease. Cuboni found this organism in diseased corn and claimed that it resisted boiling temperature. Peltauf and Heider claimed that the bacillus maidis developed very like the bacillus mesentericus or the potato bacillus, and that it was not always present in the feces of pellagrins. These bacilli were counted non-pathogenic, but an alcoholic extract of the corn meal containing them caused narcosis, paralysis, and death of mice. Babes and Sion produced symptoms resembling pellagra in various animals by the injection of this bacillus. Ballardini working with the sporisorium maidis was able to produce in man gastro-intestinal irritation, but his results were not conclusive.

To my mind Tizzoni's work is most important. He produced the disease experimentally with his organism which he isolated from the blood of a pellagrin. He claimed to have found the organism in the cerebro-spinal fluid and organs as well as in the blood. He also found it in two out of nine samples of corn furnished by the Ministry of Agriculture.

I think we will all agree with Sambon in considering that pellagra, both in its symptoms and pathology, is allied rather with syphilis

and trypanosomiasis, than with a fungus disease. The sooner we dispel the idea that pellagra belongs to the ergot group of diseases, the sooner will we clear up the question of its etiology, for surely except in that both diseases are intoxications, and that in both diseases the spinal cord suffers the brunt of the attack, there is litle in common.

I am making blood cultures from every case of pellagra within my reach, and I am also making cultures from corn meal. It is too soon to report results and it matters not how successful I may be I cannot do more than Tizzoni, for has he not satisfied all the postulates of Koch?

It has already been shown by Babes and Manicatide that the blood of pellagrins contains an antitoxin. It is probable that, whatever the germ may be, it is capable of causing the disease in most of the domesticated animals, therefore may we not hope that in a short while we will be able to secure an antitoxin with which to combat this great scourge? It remains for us to find the association, if any exists, between the etiological factor and the Indian corn. After that we may be in a position to know how to care for the corn. We cannot overlook the fact that in countries where the corn has been dried in ovens the disease has materially decreased.

In closing, I repeat, cannot the corn, whether it be diseased or sound, form a culture medium (which, of course, requires moisture) on which an organism which is the specific cause of the disease grows and develops? This view would in no wise conflict with Lombro's, nor would it make it hard to understand why heating the corn in the drying ovens has a tendency to prevent the disease. The acarus farinae may possibly play a part in the entrance of this suspected organism into the kernel of otherwise sound corn. It has been suggested that this animal by its eating through of the epidermis materially assists in rendering the corn noxious.

ETIOLOGY OF PELLAGRA—THE ITALIAN MAIZE THEORY, OR THE THEORY OF LOMBROSO.*

By J. J. Watson, M. D., Columbia, S. C.

This theory is based upon the following facts, viz:

1—That maize is the staple cereal consumed by the inhabitants in pellagrous countries.

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*Printed in Journal of South Carolina Med. Association, November, 1908.

2—That pellagra is now recognized as a disease in every maize producing country.

3—That extracts made from damaged maize will produce, in man and animal, symptoms similar to pellagra.

4—That symptoms similar to pellagra have been produced in animals by feeding them on damaged maize or corn.

The North American continent is the natural habitat of zea maidis and, ergo, I believe the original home of pellagra. In 1600 Barnino described a condition that was evidently pellagra in the American Indians and he attributed it to the eating of Indian corn. Maffei, in the same year, noted that Indians eating damaged corn had a peculiar weakness, this was evidently pellagra. Authors after Barnino and Maffei do not speak of the malady in men, but references are found to a condition existing in horses in Mexico, which was characterized by the animal becoming paretic, tabetic, with loss of hair. This was attributed by the writers to the animal having eaten damaged corn.

The disease followed quickly the cultivation of corn in Spain, for in 1735, it was observed there by Casal. Frappoli, physician at the hospital of Milan, observed the disease there and gave it its present name (from pellis, skin, and aegra, disease). Pellagra, which means diseased skin, at this time was believed to be in some way associated with the eating of damaged corn; for, in 1776, an act was passed, by request of the sanitary committee of Venice, that no one in the public markets of Venice could offer for sale or exchange corn that had a bad odor or taste.

In 1848, Ballardini stated and demonstrated that pellagra was caused by eating damaged maize. At that time he had not become acquainted with the conditions that had rendered corn dangerous as an aliment, but later propounded the theory that dampness caused a fungus to develop on the maize and that the fungus caused the disease. He also recommended that those suffering with pellagra should not eat maize, and was gratified to observe cases of recent origin recover on a maize free diet. This circumstance convinced him in his theory as to causation. He was vigorously opposed in his opinion by a great many physicians, and he as vigorously maintained his position. The acrimonious (I presume) discussion that followed as a result of doctors disagreeing and the fact that the disease had increased in Lombardy from 14 per thousand in 1839, to 28 per thousand in 1856, naturally directed the attention of the public to the disease, and we find that a commission was appointed by the

Lombardian government to look into the report on the causation of the malady. This commission after the usual vacillations that are inseparable from ignorance reported that Ballardini's damaged maize theory in their opinion was correct, but that they could not concur in his opinion that a maize free diet had any effect on the disease (how queer). They inclined to the belief that the improvement that followed Ballardini's efforts was due to bettered conditions of alimentation.

Subseqently, Ballardini discovered in the copper-green mould that is on bad maize, a micro-organism—sporisorium maidis. He cultivated this fungus artificially and with it was able to produce in man a burning sensation in the mouth, pharynx, esophagus; gastritis, and diarrhoea. Chickens fed on this infected corn lost their feathers and showed other symptoms of pellagra. It has been shown and admitted by Ballardini that this is a very rare fungus on corn and not the cause of pellagra. Yet the finding of this fungus was of great moment, for it was the cause of further researches, and these studies have been of great practical value in preventing pellagra in Italy.

The present practically universally accepted theory of the causation of pellagra is the theory of Prof. Lombroso, of Turin. Evidently stimulated by Ballardini's work, he, in 1864, commenced experiments with bad maize and eventually arrived at conclusions. that after having been vigorously opposed, have been accepted by nearly every one who has given any time and study to the disease. The Italian physicians are fully satisfied that if damaged maize could be excluded from Italy, there would be no pellagra there. This practically universal opinion of men who live and have lived with the disease for generations, cannot be lightly passed over.

The prevention and treatment of pellagra in every country today is a result of Lombroso's labors. His theory is that certain fungi (penicilium, aspergillus, etc.) found on maize, when exposed to moisture produce in the corn a toxine. That this toxine, when taken into the system, produces pellagra. These organisms when injected into persons or animals, or when grown on any other culture medium than maize, are non-tonic. If grown on a culture medium of corn meal gruel it separates into three portions; upper portion or fungus, non-toxic; middle portion or liquid, toxic; lower portion or precipitate, toxic. He has made a 33 1-3 per cent. alcoholic extract from damaged maize that he calls "pellagro-zina," and with this extract has produced, in both animals and men, symptoms similiar to pellagra.

EXPERIMENT ON TWELVE HEALTHY MEN.

Twelve healthy men were given tr. of bad maize by mouth for many days, and all symptoms were noted. There were forty-three symptoms noted in ten cases; two cases were not affected. The symptoms, diarrhoea, increased appetite, soft feces, revulsion to food, weakness, lassitude, erythema, desquamation and skin lesions, were the most important. There was also a pronounced effect on the heart and kidneys. There was a diminution of weight from 4 to 20 pounds. Two cases increased in weight six and eight pounds. Many of these symptoms lasted two and a half months, in one case for nine months, after the experiment was discontinued. Six of the men were accustomed to alcohol. Two of these had no symptoms, and two resisted much more than the others. One of the subjects was a subject of psoriasis and the skin disease was cured by the experiment. Since then the tr. of bad maize has been used by Lombroso as a treatment of psoriasis, with success.

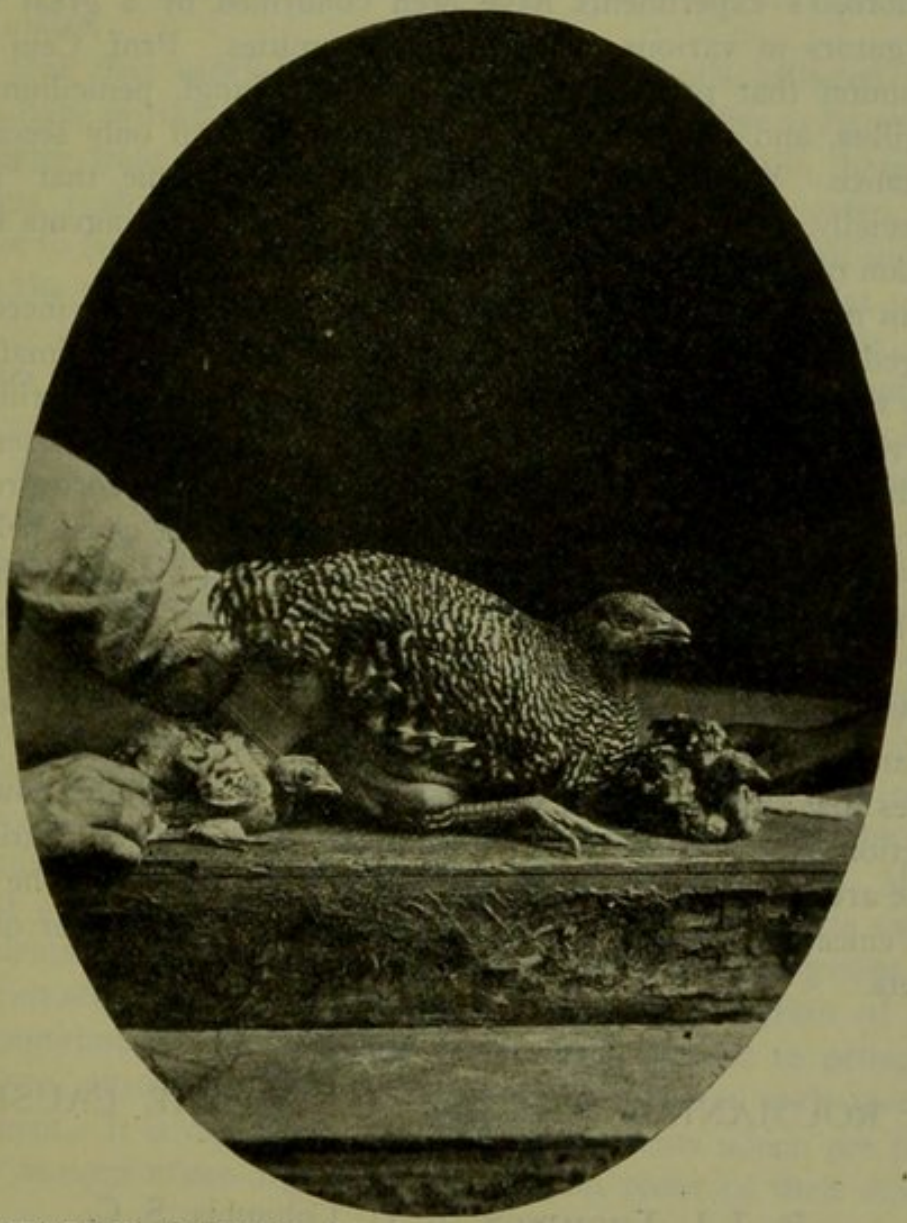
EXPERIMENT ON DOG.

Weight 15 pounds, was given 15 c. c. pellagrozina hyperdermatically. No symptoms for 45 minutes. After one hour he walked with his legs far apart, and his hind legs rigid. Photophobia, one hour and six minutes, slight tetanic contortions, reflexes exaggerated, very rigid, convulsion if least irritant is applied, drinks great deal of water, but cannot eat. After two and a quarter hours has general convulsions, cannot stand water. After three hours has real tetanic convulsion, after four hours is better, can be touched but cannot stand noise. Feces very soft and bad odor. After nineteen hours cannot walk, paresis of hind legs, has convulsions and profuse salivation. Died in twenty-four hours.

EXPERIMENT ON DOG.

Fed on bad meal, one-half pound each day from February 4th to May 4th. The dog gradually lost weight and strength. The last two days of his life he lost four and one-half pounds. May 4th, his gait was not natural. Rigidity of hind legs, reflexes exaggerated, appetite good, but cannot swallow food. Thirsty, tremor, slow respiration, intelligence normal. Diarrhoea. From May 4th he was fed on good breads and milks. May 5th, and 6th, has rough hair, convulsion when he tries to walk. Thirsty, not as intelligent. May 7th, dilated pupils, no action to light. Tetanic spasms on least irri-

tation. Respirations increased, while a healthy dog makes 12 respirations affected dog makes 112, whining with each respiration. May 8th, completely rigid, every three seconds has tetanic convulsions. Penis erect. General tremor. Cannot stand on his feet. Temp. 104 degrees. May 9th, cannot stand or drink. Rigidity of front legs. May 11th, death.



CHICKENS HATCHED AT SAME TIME, SAME BREED. THE SMALLER ONES HAVE PELLAGRA, FROM DAMAGED CORN.

EFFECTS ON CHICKENS.

Chickens when fed on infected corn lose feathers, become very poor, have erythema, with thickened and wrinkled skin. The feet and legs assume a rough, scaly condition, the feet and legs of a

chicken three months old resembling the legs of a very old chicken. They eventually become paretic, ataxic and die. Young chickens if infected do not develop, do not feather, have erythema, become paretic, ataxic and die. The photograph illustrates very plainly this effect, as all three chickens are the same age and breed—hatched in the same brood.

Lombroso's experiments have been confirmed by a great many investigators in various pellagrous communities. Prof. Ceni is of the opinion that pellagra is caused by the fungi, penicilium and aspergillus, and that the kind of alimentation is of only secondary importance. Yet he admits in a personal letter to me, that "maize is especially to be considered as one of the principal agents in the causation of pellegra."

From my own observations and studies I am fully convinced that damaged maize is the cause of the disease here. Our climate and season both are conducive to the thorough maturing and curing and preserving of Indian corn, and therefore in exceptional cases only does this cause the disease. I am of the opinion that corn properly gathered and housed in the Southern States will not cause pellagra. All of the cases I have seen in this State give a history of having eaten bought, or rather shipped, meal at some time during the year, and a great many of them ate it continually. I therefore believe that Western corn goes through a heat before it reaches its destination, and when offered for sale is infected with the fungi, as the samples that I have secured from the markets here plainly show. By inspection you will see that the samples of corn from Turin and Venice are not as much affected as samples No. 1 and 2. The Turin and Venice corn would not be allowed to be sold in either of their markets.

THE ROUMANIAN THEORY AS TO THE CAUSE OF PELLAGRA.*

By J. L. Thompson, M. D., Columbia, S. C.

It appears that pellagra presents different symptoms in different countries although in a general way the disease is identical. It is recognized that one of the foremost students of Roumanian pellagra is Victor Barbes, professor of Pathological Anatomy, of Bucharest. His chief work has not been translated yet into English and I there-

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fore undertake to present briefly some of his opinions as to causation.

The history and distribution of the disease show that pellagra is associated with nourishment from maize. As this appears indisputable there remain two possibilities, first that in sound maize substances can be present which under certain conditions can affect an organism injuriously, or second, that the disease is produced by spoiled maize.

It is true that careful investigations in all lands afflicted with pellagra have proven that pellagra appears where maize is harvested in an unripe state, or is spoiled through dampness. Still, there are pellagrous regions where, apparently, sound maize is eaten. It seems not to be doubted that these last two assumptions are of importance in the etiology of pellagra only with the limitation that often apparently sound maize is affected with a fungus and bacteria, and it remains undecided whether in entirely sound maize poisonous substances are found. This much is certain, that the more noticeable the spoiled conditions of maize, the more poisonous substances it contains.

In regard to the apparently sound maize the investigations of pellagra are interesting. Barbes reports particularly the fact that even in sound maize there are numerous organisms of minute size, often more than in that which has the appearance of being spoiled. But when the maize remains several days exposed to a dry heat of 70 degrees, it contains very few bacteria. These, produced by pure culture were not injurious to animals. Only those produced by the special culture of the bacteria of putrefaction were poisonous. More important are the results which were obtained with extracts taken from polenta and corn bread, according to which 10 grams of watery extract introduced intravenously to every kilogram of the animal experimented on, produced appearances similar to pellagra, for instance, gastro-intestinal disturbances, sometimes with mental derangement. It is doubtless a question of ferments which are produced by various micro-organisms in a certain phase of their development.

Although these investigations cannot be regarded as final, still they agree with the statistical data of Lombroso and our own, according to which scarcely 25 per cent. of pellagrous sufferers can be shown to be in the habit of eating spoiled maize. Also, Lombroso emphasized the fact that cured pellagrous patients, when they go back to a maize diet, even though it be apparently sound, always

become pellagrous again; also it appears that pellagrous persons in certain seasons of the year are especially susceptible to even very small quantities of pellagra-bearing substances.

In general these experiments show that one cannot draw a sharp line of distinction between sound and unsound maize, and that maize kept in the customary way always contains numerous micro-organisms, and their products; for certain damp parts of a grain of maize, especially the embryo, offer favorable conditions for the development of bacteria; so that Lombroso admits that most of the apparently sound maize can, in a certain sense, be regarded as spoiled. From all this one can assume that maize in general, not of itself, but in consequence of its being easily spoiled, may be regarded as pellagra-bearing.

We come now to the discussion of apparently spoiled maize, which according to the "zei-toxic-theory" causes pellagra. As far back as 1776 the board of health of Venice passed regulations for the prevention of pellagra, which were based on the view that pellagra is caused by eating spoiled maize. It was absolutely forbidden to harvest the maize in flooded districts, or to use the spoiled maize as food for man or beast. Such maize was not allowed to be mixed with the good food or to be sold. The public dealers were under obligations to give notice to the health officers as often as they had notice of such maize. This same duty devolved on the millers who were not allowed to grind such maize; also the bringing of such maize to be ground had to be reported by the officers to the board of health.

All things taken together, it stands proven that where the maize is not kept sufficiently dry and when there is a scarcity of food pellagra shows itself the most. It has often been asserted that maize brought in from other regions, especially when brought in by coastwise vessels, being usually exposed to a high degree of dampness, predisposes to pellagra even though the maize was sound when shipped.

We must distinguish between unripe and spoiled maize. The unripe grain of maize is whitish or dirty white and almost entirely shriveled, only having a thin layer of transparent hard substance on the outside, so that the embryo and perispor appear dirty brown, or blackish. The kernel often lies in a hollow which contains a brown blackish powder, or a crumbly decomposed substance (this is what our farmers call black-hearted corn) and often the surface of the grain is covered with a mold or black dots, while on the inside

the hollows are often covered with mites, scarcely observable to the naked eye. These gnaw the grains a little and then the fungus penetrates the inside.

The ripe grain of maize that is spoiled is often cracked open or wrinkled, being only covered with roundish black spots without lustre. The root of the grain is shriveled and blackish or occasionally with greenish spots. The embryo of the grain is often shriveled and surrounding it are hollows which contain a greenish powder, beetles and mites. According to Lombroso, spoiled corn meal is whitish yellow or greenish brown with mouldy smell and a bitter or sharp taste. A grain of spoiled maize when warmed in the hand has not an agreeable smell, like healthy cooked maize. We have microscopically examined sections of a grain of spoiled maize and found that the black points on the surface consist of colonies of thick bacilli which multiply and extend themselves inside of the root substance in the form of a string.

The spoiling of maize comes from abnormally great dampness, and from the fact that it is often harvested when unripe and still further that when harvested it is not carefully handled and dried, but is kept in damp warehouses, or not protected from dampness. If maize really caused pellagra, per se, then the disease would be much more general than it really is. In Roumania, for example, are about 5,000,000 peasants, whose chief food is maize, and still we have, according to statistics of the year 1898, only about 20,000 pellagrous persons.

Maize is eaten in the north and south of Italy, yet the disease is unknown in Sicily, while in Lombardy the disease is very common. In Burgundy much maize is eaten and yet the Burgundian peasants do not pay tribute to pellagra as do the peasants from Landes (in S. W. France). The Mexicans eat perhaps as much maize as the Roumanians and the Italians, yet the disease is almost unknown to the Mexicans. Maize is pretty much the same in all regions of the world, still the conditions under which it is grown, harvested, preserved and prepared for food are different.

It is asserted that in Roumania only the white or yellow maize causes pellagra, while red maize is harmless. In these are varieties of maize with greater power of resistance in respect to spoiling, whose introduction and general use would perhaps be able to limit the spread of pellagra, but there are no sufficient investigations about this. In Roumania the peasant, in consequence of his relations to the land owner, or renter, and in consequence of his failure

to observe the laws, which are intended to lighten the heavy lot of the tillers of the soil, is not able to sow or harvest the maize at the proper time; also the Roumanian peasant does not carefully clean his maize after the harvest, preserves it in unventilated granaries, where, in consequence of the poor construction of the cribs, it is only poorly or may be not at all protected against injury during the fall and winter; or on the other hand, the maize is piled up in the loft or in the ante-room where it is deprived of the sun's rays and exposed to dampness. To these conditions must be added climatic influence, as a rainy, cold or early fall hinders even the early sown maize from ripening thoroughly. Hence it is easily understood that a grain rich in fat and carbohydrates, planted and harvested under such circumstances produces toxins by fermentation, which were extracted by Lombroso as weil as Babes, and in case of animals produced symptoms which, while not showing the entire clinical complex of pellagra, showed an analogy of it.

In Italy no doubt the same conditions explain the fact that maize produces pellagra. In Southern Italy the conditions are entirely different, for the mild climate of summer, as well as of winter, permit the industrious, the lazy, the rich and the poor countryman alike to raise his maize crop early and then preserve it well dried. This is the case in Burgundy where the maize is dried in ovens. In Mexico maize is subjected to a special treatment with lime or ashes, which are rich in potash, thus the germs are prevented from developing and giving occasion for the grain to decompose and form toxins before it is eaten. In these regions maize does not produce pellagra, nor in Ireland where the maize is well aired.

THE PROTOZOAN THEORY OF PELLAGRA.*

By J. H. Taylor, M. D., Columbia, S. C.

When so universally recognized an authority on a special subject, as Lombroso is on pellagra, renders a verdict seemingly final, it is perhaps presumptuous for anyone to entertain a different view. It is only in the spirit of endeavor to cover the whole ground of the etiology of pellagra that I undertake to read these notes, based upon Sambon's recently expressed protozoan theory. The desperate

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efforts to associate maize with pellagra really borders on the ludicrous when one reviews the numerous fungi and bacteria that have been isolated from this cereal and its products, and with which men of eminence, not pseudo-scientists, claim to have reproduced the symptoms of the disease in animals. One after another these claims have been disproven, and there now remains to be considered only the toxine theory of Lombroso. This conviction of the maize guilt is so firmly fixed among the Italians, at least, that where a case presents showing the classic symptoms, but with which they can associate maize in no way, then it becomes a case of "pseudo-pellagra," and not the true disease. However, if men of such splendid ability as Sir Patrick Manson and Sambon, of the London School of Tropical Medicine, question the maize theory and look for another cause, they do this with reason, and their opinions cannot be put aside lightly.

Sambon acknowledges the close association of aspergillus and penicilium with pellagra, but calls attention to the wide variation between the symptoms of pellagra and those of all other fungoid diseases—thrush, ring worm, actinomycosis, etc.—and, moreover, he emphasizes the presence of this fungus in millet, vetch, barley, wheat, rye, and grapes, the ingestion of which is harmless. He believes that with a deeper knowledge of the protozoa we shall find the cause of pellagra here, basing this belief on the similar symptoms and pathology with syphilis and sleeping sickness, both established protozoan diseases, and pointing out in his magnificent article (Remarks on the Geographical Distribution and Etiology of Pellagra) numerous discrepancies in the maize theory that need explanation, and until it is forthcoming, warrant one in holding to his skepticism.

POINTS AGAINST THE MAIZE THEORY.

1. The cultivation and extensive use of maize antedates by about 200 years the appearance of pellagra in Italy.

2. There are extensive regions today where pellagra is rampant, but where corn is neither grown nor eaten, while on the other hand, there are large areas where this cereal is the principal article of diet, with all its fungi and bacteria, but pellagra is unknown.

3. The disease shows a "tendency to spread slowly but widely and independently of maize cultivation and maize theories."

4. It attacks chiefly persons of the rural districts and seldom those in towns, though both use maize as a staple article of diet; and, again, of individuals in the same household, living under identical

food conditions, often one only is attacked, the others remaining absolutely free of symptoms.

5. If due, as claimed, to a toxine, then children should, as in ergotism, be most often attacked. On the contrary, the adult is by great odds the principal sufferer in pellagra.

6. If a toxine, why the distinct periodical appearance of the symptoms?

7. If a toxine, why is the poison not transmitted through the mother's milk?

SIMILARITY OF PELLAGRA AND PROTOZOAN DISEASES.

With these facts in mind, now let us consider in detail the striking similarities between this disease and the two protozoan diseases, syphilis and sleeping sickness.

Regarding the sleeping sickness, the first recorded observation of the specific organism in man was observed by Nepveu, a Frenchman, about 1891, though he did not grasp the significance of his observation, nor did he associate it with any definite disease. Ten years later an English colonial surgeon in the River Gambia Colony, in examining the blood of a supposedly intractable case of malaria noted free in the plasma a very minute motile organism, the nature of which he, too, failed to recognize, though he thought it might stand in a causative relation to the symptoms. Six months later, still puzzled and at a loss how to associate the organism with the disease, he called to his assistance another English physician, Dutton, who was making a study of malaria in the colonies. Dutton at once grasped the significance of the observation and recorded his beliefs in its relation to sleeping sickness. Since this time there has been abundant verification of their association from many quarters, and the theory is generally accepted. This parasite was the tryponosome hominis, and is a small transparent non-pigmented, actively-motile organism about the diameter of a red blood cell, spindle shaped, and moving with a wriggling, spiral-like motion. All we know of its life history is the fact that it multiplies in the blood by longitudinal fission, that it is conveyed from animal to man by an intermediate host, the tsetse fly being the one known carrier. There are a number of other trypanosomes, but this is the only one known to be pathogenic to man.

Now, regarding syphilis and its protozoan nature, there has been demonstrated free in the blood of syphilitics and in the scrapings from chancres, papules, condylomas; in the aspirated fluid of enlarged lymph glands, etc., an organism closely allied to the try-

panosomes, namely the spirocheta pallida, or treponema pallidum. Moreover, Metchnikoff has demonstrated it in the lesions of artificially infected apes. As our endeavor is to show the very suggestive similarity in many respects between pellagra, sleeping sickness and syphilis, the latter manifested in the symptoms of general paresis and the pathology of general paresis and tabes dorsalis, it is of vital importance that Schaudinn has found that the fluids of infected persons react specifically, of which reaction Wassermann has made a special study and has found a positive response in general paresis and tabes, which establishes the true syphilitic nature of these conditions.

We shall now consider the parallel to be drawn between these three diseases, two of them established protozoan diseases, and shall find them most striking and compelling of our attention.

Type: They are all essentially slow progressive toxaemias, the duration of pellagra being from a few weeks to twenty or more years; that of sleeping sickness from three months to three years; and syphilis from a few months in the malignant type to an indefinite period.

Remissions: In each we may have definite and distinct remissions, lasting from a few weeks to months or years in syphilis; from one spring to the next in pellagra; and in sleeping sickness before the final stage we have short but distinct exacerbations and remissions.

SYMPTOMATIC SIMILARITY.

In all we have the prodromes of general malaise, headache, languor and mild digestive disturbances, which clinical symptoms are exhibited by all diseases of a microbic nature, and are readily explained by the thory of intoxications by ptomaines engendered by the organisms which, as they become more generalized, will produce more pronounced symptoms. A rise in temperature is constant in sleeping sickness, usual in syphilis, and occasional in pellagra.

Skin: A salient feature of each is a striking skin eruption, characteristic and practically constant, due in all probability to a localized deposit of virus, with resulting changes in the texture and appearance of the involved areas. In pellagra and syphilis this lesion is symmetrical, while in sleeping sickness there is no definite arrangement. In pellagra it appears on exposed surfaces; in sleeping sickness and syphilis it may be general over the body and limbs. In pellagra it appears first as an erythema, later taking on the pigmented, scaly character, as usually seen, and is often pruritic. In

sleeping sickness a peculiar itchy eruption is an early manifestation, and appears as an ill-defined erythema in patches distinctly ringed, in some cases seven to eight inches in diameter. Again, it may appear as rubelloid spots of congestion, shading off gradually into normal skin, and finally it may present as measly patches with the skin appearing slightly thickened and swollen. The skin manifestations of syphilis are, of course, legion, and the appearances of both sleeping sickness and pellagra may be simulated exactly.

General Symptoms: In the three there is common to each that gradual tendency to weakness, emphasized especially in the lower limbs in pellagra and general paresis, accompanied by progressive anemia and emaciation.

Nervous Symptoms: We come now to possibly the most remarkable of all the similarities; namely, that of the nervous symptoms in pellagra and general paresis. In that type of pellagra where the spinal and mental symptoms stand forth prominently we have the picture of general paresis so closely simulated in every respect that it is extremely difficult to differentiate the two with the nervous symptoms alone considered. The one point of difference that has been called attention to is the absence of motor speech derangements in pellagra, which is a constant symptom of general paresis. Tuczek, indeed, claims that the conditions undoubtedly do coalesce, beginning with pellagroid symptoms and finally becoming true general paresis. As a rule the nervous symptoms in pellagra are not progressive, while in general paresis they are essentially so. In sleeping sickness we have the same headache and mental hebetude, the patient appearing apathetic, with cerebration retarded; muscular spasm, epileptic seizures and tremors often appear, paretic symptoms in general gradually supervening. The deeper reflexes are primarily exaggerated, followed by a total loss; later, contractions of the flexors of the arms and legs appear and rigidity of certain groups of muscles is usual. The mental state, according to Jackson, is not usually that of general paresis, mania and the delirum of exaltation rarely being observed, while on the contrary despondency and a consciousness of wretchedness is the rule. In other respects it resembles general paresis.

Pathology: Here we have further verification of the relationship between syphilis and pellagra in the spinal cord changes. Lombroso states that in the most typical cases they suggest that in incipient tabes, with this difference: while in pellagra few changes are found below the dorsal region, in tabes the lumbar region is chiefly affected,

though the cervical may be most involved. Both show degenerative changes in definite portions of the spinal cord, tabes attacking the posterior columns especially and pellagra the lateral or both posterior and lateral. Both diseases show a combination sclerosis. Marie considers that this sclerosis of pellagra resembles more closely that of general paresis than tabes—both, you will observe, being syphilitic lesions. Edema of the central nervous system and a chronic lepto-meningitis is common to general paresis and pellagra, while in sleeping sickness we have a universal meningo-encephalitis in the form of a small round-cell infiltration.

Treatment: Professor Neisser, of Breslau, at the last German conference for internal medicine, reported his experiments with syphilis on apes in Java, where he demonstrated an extraordinary influence of atoxyl on infected animals, where it acts as a true specific and prevents the development of the spirochetes, so that when given early the disease is stopped completely and the animal can later be re-infected. In this institution (the South Carolina State Hospital for the Insane) we are at the present moment having remarkable results from atoxyl in pellagra when taken early in the disease, and the same results are reported in the case of sleeping sickness.

In conclusion let me quote from Sir Patrick Manson's lectures on tropical diseases: "We cannot be too cautious about adopting decided views on so fundamental a matter as the etiology of a disease. Everything depends on this—diagnosis, prevention and treatment." Recently two Americans, McNeil and Noby, have discovered a way of cultivating protozoa outside of the body, and among other things have demonstrated their presence in blood culture where they were not to be found by ordinary microscopical examination. It is not quite possible that with further knowledge we may yet find pellagra to be a protozoan disease?

A THEORY AS TO THE CAUSE OF THE RECENT APPEARANCE IN THIS COUNTRY OF PELLAGRA.*

By John McCampbell, M. D., Supt. State Hospital, Morganton, N. C.

I suppose that no one any longer seriously doubts the existence of pellagra in the South. Sufficient and indisputable evidence has been

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brought forward to establish this fact beyond a peradventure. It will be noticed further that practically all reports of cases have been made within the past eighteen months, indicating that it has made its appearance in recent years, since it is not at all likely that the medical profession would long overlook or fail to diagnose a disease so distinctive in character and so fatal in effect.

The question naturally arises: why this invasion? So far as I am able to determine practically all authorities on the subject attribute the disease to the use of damaged corn as an article of diet—that is, corn which has undergone some change, probably, putrefactive or fermentative, since sound corn never produces it.

Now it is well known that corn and its products have always figured largely in the diet of the people of the South, perhaps more so in the past than at present, yet there was a comparative exemption from pellagra until recently. Granting that the disease is with us and probably of recent appearance, and that it is due to the ingestion of damaged corn, we, in our prophylactic efforts would naturally look to the quality of our corn and determine, if possible, why it contains this toxic substance.

It has occurred to me, and hence my theory, that our present manner of harvesting may have something to do with it. Under the present day methods of intensive farming, it is a very common practice to cut the corn, stalk and all, at the ground, often in a state of immaturity, in order that the ground may be planted with some other crop, usually wheat or rye, and this corn is packed wet and possibly green, in a shock, where conditions are favorable for fermenting and heating and possibly the development of the products which give rise, when eaten, to the disease. This method of harvesting is in contrast to that prevailing some years ago, when it was the custom to cut the top just above the ear, leaving the ear and remainder of the stalk to stand in the field until they were both fully matured and perfectly dry, before gathering. At this time, nothing was cut as before described, except a late crop, to escape the frost, and this was usually fed to horses and cattle, being considered unfit for bread, and it is suggestive that while pellagra was unknown, "blind staggers" in domestic animals was comparatively common. Further confirmation of this theory is found in the fact, that in Italy and especially in the Lombardy and Piedmont districts, the hot bed of pellagra, the manner of harvesting corn is somewhat similar to that now prevailing in this country, inasmuch as owing to climatic condition it is necessary to gather corn in a partially green

state, or at least, in a condition necessitating further drying; and I understand the Italian Government has issued special instructions for this procedure, recognizing that it was in a faulty curing process that the poison developed.

The idea which I have attempted to set forth may be far-fetched and based upon inaccurate and insufficient observation, yet I believe it to be, at least, worthy of further consideration.

PERSONAL EXPERIENCE WITH SOME CASES OF NERVOUS AND MENTAL DISEASES SHOWING THE PELLAGRA SYNDROME.*

By Isaac M. Taylor, M. D., Broadoaks Sanatorium, Morganton, N. C.

In presenting to you today the notes of seven cases of pellagra which have come under my observation since 1905, I will have to confess that I had not made the diagnosis while they were under treatment, but since the attention of the profession has been drawn to the occurrence of pellagra in America, I have recalled the cases, and from memory and imperfect notes this report is made. Let me also call your attention to the class of persons represented by this series of cases, all of them well to do people. In none of them was the disease the result of privation or enforced use of damaged food stuffs. They represent the occurrence in about three hundred patients, all white, of the nervous, insane and inebriate classes, and covering a period of about seven years. I can recall no cases in my private practice, which was limited to about four years in country practice among the very people who might be supposed to be the most exposed to the conditions which cause the disease.

I cannot assume to give information regarding the cause, symptoms or treatment of this disease, which would be anything but a compilation from books at your command, and can say that with a mind open to conviction, I await the American study of the disease and accept the conclusions of the physicians of Southern Europe, that pellagra is a disease entity, and that its ocurrence among those who use maize as a food points to damaged corn as carrying the disease producing elements.

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There is no longer any doubt in my mind that we have pellagra in the Southern States, this conclusion has been reached by the study of our cases by accurate diagnosticians, and if there was left room for doubt, it has been removed by the testimony of Drs. Babcock and Watson, who have seen in the hospitals for pellagra in Southern Europe cases identical in appearance and symptoms with those under their care in Columbia. I cannot doubt that the cases which I present in this paper were cases of pellagra. One had previously been under the care of Dr. Babcock; another was under Dr. McCampbell, and independently we have reached the same conclusion. Looking back over the twenty-three years during which I have had the care of insane persons, I know I have seen many similar cases.

One case under treatment at the South Carolina Hospital for the Insane, and pointed out to me in September, was perfectly familiar as a type often seen in the wards for demented cases, and the dermatitis associated with diarrhoæ and other symptoms have always heretofore been accounted for by us as a trophoneurosis incident to the degeneration of the brain and central nervous system controlling nutrition. Omitting the dermatitis as an essential to the syndrome, or recognizing that the skin involvement, even in fatal cases, may be so insignificant as to escape observation, I am willing to accept "pellagra sine pellagra" as a prominent cause in the degenerating dementias, and as an equal factor with undemonstrated tuberculosis infection, in the causes of death due to exhaustion of dementia and chronic melancholia in our hospital records.

I must reserve the opinion that there is sometimes associated with acute disease with profound nervous and digestive symptoms an exfoliative dermatitis, which is not due to a specific infection. I have recently had such a case under my observation in a very nervous man under treatment for morphia habit. The diarrhœa was so promptly checked and improvement in the dermatitis was so rapid, that I am convinced that the trouble was due to the nervous condition and faulty nutrition. With this introduction I ask your consideration of the following cases:

Case I: Female, age 45, married, two grown children, of highly nervous family, brunette and given to tempers, had used opiates and alcoholics since establishment of menses for dysmenorrhoea, and in that way had acquired the habits. Had just been through a course of treatment for rapid withdrawal of drugs and alcohol, and developing mental symptoms, was sent to my care. On admission was quiet and agreeable, in a few days began to show mental symptoms

of highly hysterical character, developing a mild mania followed by depression. Was admitted in May and remained until October; when admitted had an old eruption on hands, arms and face and on back of neck. During all the time of her stay she ate very poorly, and while the eruption was prominent was practically without food; no note of a diarrhœa, but mouth and tongue were red and irritated. There was a gradual improvement under tonics, though was still in poor physical condition when discharged, immediately relapsed into drug habit at home and went to another institution for treatment, is still living. No account of a recurrence of the eruption and the general symptoms.

Case II: Male, age 31, single, business man, under care from July to October. Following business reverses had become very religious; very depressed; would go for days without food; on admission was very depressed and very much emaciated; was not confined to bed, but gradually lost flesh and strength; complained that the taking of food hurt him; pigmented areas on face and backs of hands and arms developed during August and continued afterward; tongue red and toward the end an uncontrollable diarrhœa; died.

Case III: Female, age 26, lady of highest culture; had been for six years under treatment by best surgeons and physicians for various uterine troubles, undergoing several operations, and in the year before coming to me for the second time under the care of a prominent Philadelphia specialist, for developing mental trouble, following a prolonged neurasthenic condition. Had improved while under treatment at the North, but immediately relapsed at home, and was growing worse when admitted in July, having first been seen in May, was under care until October. Was in fair flesh when admitted; almost refused food; under compulsion would eat, but soon vomited food; very weak and unable to walk or stand; gradually lost ground; developed diffuse irritated areas on arms, neck and face, and pigmented and irritated spots on exposed parts of body; tongue very red, mouth irritated; diarrhœa came on, there was gradual loss of strength, and finally death.

Case IV: Male, age 24, stable man, admitted in July and under care for two weeks; in delirium tremens when admitted; was troubled with dermatitis of hands, face and arms; no note of diarrhœa or stomatitis; made quick recovery and was discharged. About a year afterward saw him in consultation; found him demented, with con-

stant diarrhœa, a constant fever and delusions; he was admitted to the State Hospital where he died in a few weeks.

Case V: Male, age 20, married, farmer, had been drinking steadily for a year of the poorest quality of corn whiskey; for a month before admission in October had been insane; was maniacal; a large part of body, hands and arms was covered by an eczematous eruption; had a severe stomatitis; a constant diarrhœa; almost total refusal of food; steadily lost ground and died on the eighth day.

Case VI: Female, single, age 34, office worker, alcoholic, had been for a month drinking heavily; very nervous appetite, eating almost nothing; under care from November to March; on admission face was splotched with a discrete eruption, and backs of hands and arms showed an old dry pigmented eruptive area. With withdrawal of stimulants, tonic treatment and good food, eruption disappeared and skin became smooth; died some months after leaving my care in another hospital with delirium tremens.

Case VII: Female, married, age 53, was depressed when admitted in June; had been for some time in South Carolina Hospital for Insane; had a diffuse dermatitis on hands, arms and body; appetite almost nothing; very much emaciated, and a constant diarrhœa and troublesome stomatitis. There was a gradual failure and the end came within a fortnight. I thought in this case there was a tuberculosis infection of the bowel or peritoneum, and the eruption incident to the starvation process, but in the light of present knowledge I think with Dr. Babcock that this was a case of pellagra.

In these cases the use of alcohol was a factor in four, two men and two women, all except one have died. In no case was the use of damaged corn traced, but I assume the liability of all our people from the universal use of corn products as food. I think those who live in town and indiscriminately buy meal from their grocers are more in danger than those in the country where care is ordinarily taken in the selection of grain for bread.

In conclusion, I wish to express the opinion that pellagra has been with us a long time, we have been misled by the authors of the text books who have dismissed the subject with a few lines, but I am sure I have been seeing these cases for twenty years, and I can believe that under diagnosis of chronic diarrhœa, intestinal indigestion, eczema and sunburn many cases have been seen and dismissed. I am inclined to think, though my experience with negro patients is very limited, that many so-called scrofulous negroes, are really pellagrous. We are beginning a great undertaking to eradicate this dis-

ease from our country. There are remedies medicinal and hygienic. The principal remedy seems to me in prevention, and the chief factor in prevention is the education of the people to rigid inspection of corn offered for sale for man or beast.

CLINICAL OBSERVATIONS OF PELLAGRA*

By H. E. McConnell, M. D., Chester, S. C.

Some four years ago or more in my work as a general practitioner, my attention was called to a peculiar condition or disease among some of my patients in a mill village near town. The first symptom I was usually called to prescribe for was a sore mouth and indigestion, and a severe diarrhœa. This would usually be in the early spring, and as the weather would get warmer a peculiar red and rough erythematous eruption would appear on the backs of the hands. I looked upon the disease at this time as a severe form of stomatitis; and, at times when the tongue and gums would bleed, I thought of scurvy, especially as at that time of year fresh vegetables were scarce, and the mill folks were eating large quantities of canned goods. But the clinical picture did not quite fit for a diagnosis of scurvy. Well do I remember getting down all my text books and trying to fit a diagnosis to the disease; but very little could I find in the text books. Finally, in my reading I came across an extract in the *Medical News,* quoted from a French medical journal, giving a description of pellagra. I realized at once that this was the disease with which my patients were afflicted. I had two patients die of the disease in 1904, both females over forty years of age. If the claim to priority in recognition of the disease in this State is of any significance, I am certain I diagnosed it as early as 1904, but, unfortunately, did not report my cases, though I called the attention of my doctor friends to the disease. In the last three years I have had eight cases under observation, five females and three males. Of these eight, four are dead and four are still under observation. Of these four; one is apparently cured; one, now in this institution (State Hospital), is very ill; the other two are men at work, one on a farm, and one as a clerk in the county dispensary, who says that he is well, and that he cured himself by drinking corn whiskey, and plenty of it. In the ten cases observed, seven were females, and all

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of these are dead except one. All of the men are living; so it is evident that the men withstand the disease better than women; and that women are more prone to the diease than men.

As to the cardinal symptoms of the disease—salivation, skin eruption, and mental disturbances—I mention them in the order that they seem to occur to me. Instead of tropho-neurotic disease, I would most certainly call it a gastro-intestinal disease, as I am confident that the stomach and the small intestines are the parts of the body first involved; and that the skin eruption and the trophic and neurotic symptoms appear later, and are produced by the absorption of the toxins from the gastro-intestinal mucosa. If we accept the theory that the disease is contracted by eating spoiled or fermented corn products, we must also accept the theory that it is a toxine or fatty oil as suggested by Lombroso; and that it is unaffected by heat, as all corn products are cooked well before eating.

One of my patients, whom you will see today, took sick in the spring of 1907. I was called to her, and found her suffering with a sore mouth, fiery red and pointed tongue, denuded of epithelium, buccal mucosa also very red, marked salivation, and burning sensation in the stomach, and a diarrhœa. I did not at this time recognize hers as a case of pellagra. I treated her at this time for stomatitis and diarrhœa. During all this time she showed no special nervous symptoms, except she was weak and discouraged as to her condition. During this time she was nursing a baby. In the fall and winter she improved, and was able to do her housework; and not until the spring of 1908, when I was called again to see her for her sore mouth, did I realize she had pellagra; then I found a red erythematous rash on the back of her hands, going up no higher at first than the cuff or edge of her sleeve.

This erythematous eruption on the hands is the most constant and diagnostic sign of pellagra. They all have it; or, at least all of my cases have had it. The eruption is similar in all cases, and only varies in intensity. It needs to be seen only once to be recognized; and if you ever shake hands with one of these patients, you never forget the sensation; the skin of the hand is dead, harsh, and rough. The eruption appears first on the back of the hands, then on the forehead, sides of the neck, and in the later stages on the elbows and dorsal surfaces of the feet. The neurotic symptoms are among the last to appear, and continually grow worse as a rule until you see them as you see in this patient today. Usually the first thing they complain of is insomnia, pain in back and back of neck, general mus-

cular weakness, staggering gait, and marked depression. These patients most always have a frown on their foreheads, are irritable and melancholic. The patellar reflex is exaggerated, no marked irregularity of pupils, eyesight gets bad—almost all of these patients have double vision; and towards the end they sink into a melancholic condition, and die from exhaustion after having clonic convulsions a day or so before death; although one of my patients was acutely maniacal a week before her death.

Though I have had six cases to die, as yet I have made no autopsies, neither have I made any microscopical examinations of blood or feces; though I have made chemical anyalsis of the urine, and found nothing abnormal. I am so confident that the poison first spends its energies upon the stomach and small intestines that I believe if we could make our diagnosis earlier, and give treatment directly to these organs, we could expect a larger percentage of cures. Therefore, I shall expect some of my colleagues, who are making a specialty of stomach analyses, to help us to make an earlier and more definite diagnosis, and suggest a specific cure if possible.

I have one patient to present as cured, the oldest of all of the ten cases, 67 years. He has had the disease for three years, and, during the summer of 1907, was so weak that he was often confined to his bed for two or three weeks at a time. The treatment of his case was with hydrastis, 15 drops before meals, nitro-muriatic acid dil., 10 drops after meals, with plenty of milk, eggs, and vegetable broth, and by use of the leucodescent light treatment applied over the region of the stomach until the skin was reddened. This man has been perfectly well all summer, and has spent most of his time working his garden in the sunshine. As yet there has been no return of the sore mouth, eruption on hands, or diarrhœa.

If the diease is due to eating bread, etc., made from musty meal, then we must see to it that our "johnny cakes and muffins" are made from good sound meal like the kind our fathers were raised on. Meal from good sound corn, that has stood in the field until frost, after the fodder has been pulled and was thoroughly dry when harvested; and also selected—the best corn in the crib being put in a bin for meal, and the rest fed to the stock. I believe the Western plan for harvesting corn that is fast being adopted in the South, of cutting and shocking the corn just when the fodder is ripe, and the grain still soft, may have something to do with the development of the fungus that produces the disease. For the center of one of these shocks of corn, with its heat and moisture, would be a splendid place

for the development of any fungus growth. If we have to ship corn from the West, let us ship it in the ear, then we can more readily tell when it is sound.

Before closing my paper, I would like to ask for the sake of the pellagrous patient, that all such terms as "awful, dreadful, and leprous," as applied to the disease in the newspapers be left off, as many of these patients read the papers, and it has a very depressing effect upon them. I wish also especially to thank Dr. Babcock for his untiring energy and zeal in bringing this conference together, and his kindness to me during the past summer in spending a day with me before his trip abroad, and verifying the diagnosis of the cases which I had to show him.

REPORT OF THREE CASES OF PELLAGRA IN ONE FAMILY.

By Dr. D. B. Frontis, Ridge Spring, S. C.

I desire to present to you this little boy, Julian C., age 9 years, for your inspection and diagnosis. You will notice dry, red, and scaly eruption on back of hands, wrists, face and neck. Notice that he has stumbling gate and walks with feet far apart. Patella reflex is exaggerated. He has had the disease six years, the first manifestation was at two years of age. There were three children in this family, all affected with the same disease. An older brother died in 1906 at 11 years of age, duration of disease eight years. The sister died 1907, 14 years of age, duration of disease ten years. The father is a miller and all of the children were born and reared at the same place. When disease first appeared, it was supposed to be rhus poisoning and was treated with domestic remedies. The skin on hands and feet was first affected and I would describe it as an exfoliative dermatitis at first etythematous, then raw and weeping; still later scaly and crusty, and under latter sometimes foul smelling pus. Cutaneous manifestations of disease; worse in spring and fall, disappearing entirely during winter, save a certain amount of pigmentation and scaring as the disease progressed. Later the elbows, knees, face and neck were affected.

In the last year of life of the fatal cases of boy and girl, there was persistent diarrhœa. Body orfices all affected. Mucous membrane of mouth denuded of epithelium and very red and in last weeks ulcerated. Margin of lips at junction of mucous membrane, more

or less fissured. Some involment of nares and external auditory meatus. Epistaxis troublesome at one time in case of boy. Rectum very raw, red, areola of two or three inches, some fissures and disposition to bleed on defecation. This condition is present in the little patient before you. Prepuce in boys affected. Labia in girl were swollen, raw on inner surface, dry and fissured on cutaneous surface. Urethra much swollen in last weeks, incontinence of urine. The girl presented marked symptoms of mental aberation, but never violent. Speech disconnected and often wandering. There was sleeplessness. Decubitus-opisthotonos at one time with slight convulsions in the girl, but more commonly these patients would lie curled up with knees very close to face.

The spring exacerbations were usually preceded by two or three weeks malaise and loss of appetite. During height of disease, the appetite was variable.

While the writer realized that the clinical picture in these cases was different from anything he had ever seen, correct diagnosis was not made until this summer when especial attention was drawn to the subject of pellagra by Dr. J. W. Babcock. The fact that these three cases occurred in one family and are children of a miller is very interesting in connection with the generally accepted etiology of the disease. Neither the father nor mother nor a brother-in-law who has lived with them for a numbers of years, have shown any symptoms of the disease.

The father says that he has very rarely ground or used meal other than from home-grown corn.

Another curious fact is that the boy who died, very rarely ate any corn products as he did not like them. Still as he had eaten some it would show that it was not the quantity of corn product used that was dangerous, but that infection might occur from a single or a very limited consumption of toxin-bearing maize. It is further very interesting to note that infection of these cases occurred singly and at intervals, the children showing first symptoms of the disease at four, three and two years, in the order of their development.

These dates were given by the parents; none of the cases were seen by me until four years ago. It may be worth something in considering the etiology of these cases to note that this family lives close by a water mill with a pond above, and there is always dampness.

The writer had another fatal case of this disease this summer in a female, age 60, who applied in the spring for treatment for diar-

rhœa and indigestion and also had the dermatitis on back of hands and wrists. This condition persisted with exacerbations until her death five months later. Cannot say that there were no previous manifestation of the disease, but it seemed to run a rapid course and symptoms were very similar to the girl reported above.

There was the diarrhœa with frequently blood in stools. Mucous membrane of mouth very red and sore, and later dry and fissured lips, very red, raw areola around rectum. Labia very much swollen and fissured on cutaneous margins. Incontinence of urine in last three weeks of life and marked mental aberation and sleeplessness.

The curled up decubitus was marked in this case—never any opisthotonos. Reflexes were not tested. All my cases had some fever at height of exacerbations but never constant. All were of the white race.

FOUR CASES OF PELLAGRA.*

By G. A. Neuffer, M. D., Abbeville, S. C.

Within the past two years, I have treated four cases, presenting a complex group of symptoms and conditions entirely different from anything I had ever seen during an active practice extending over a period of twenty-four years. The first two of these cases I treated and death supervened, with myself and consulting physicians entirely at sea as to diagnosis. In December, 1907, I read Dr. J. W. Babcock's report to the State Board of Health on "Pellagra," and I at once realized that this was the disease that I had been contending with. My colleagues who had seen the cases readily agreed with me in this opinion. Cases Nos. 3 and 4, I diagnosed as pellagra. Case No. 4 was seen by Dr. C. F. Williams, of Columbia, S. C., secretary of the State Board of Health, in consultation with Dr. C. C. Gambrell and myself. After a careful and detailed examination, Dr. Williams confirmed our diagnosis.

Case No. I: Mrs. McC., age 22, married, no children. I treated her from Aug. 6, 1906, to Oct. 12, 1906. When first seen I diagnosed stomatitis, accompanied by a peculiar eruption on the back of the hands, which I did not undertake to name; treatment for stomatitis did no good. Her symptoms briefly stated were as follows: Fever ranging from 100 to 102 F., a redness and swelling of

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the skin over the backs of both hands and forearms, with intense itching; sore mouth, red slick tongue, gums swollen, free flow of saliva; no appetite; vagina and cervix uteri presenting same condition as mouth, discharge and itching intense; pain over epigastrium; rapid emaciation; obstinate diarrhoea; and for two weeks before her death, which occurred Oct. 12, 1906, she suffered with acute mania, requiring hypodermics of morphine and atropine to control her. This patient was seen by several of my brother physicians, but none of us made a diagnosis, nor had we the remotest idea of the correct one. I do not deem it necessary to give you in detail the treatment of this case, suffice it to say that she had every kind of treatment, both internal and external, that three physicians could think of, but all without the slightest benefit, and death ensued as stated above.

Case II: Mary Smith, negress; age 27, married, no children. I treated this case from April 13, 1907, to June 12, 1907. She presented identically the same conditions as Case No. 1, and had exactly the same train of symptoms. Dr. Gambrell, who saw her with me, as well as myself, was particularly struck with the marked similarity of the two cases. She received about the same treatment as Case No. 1, and grew steadily and rapidly worse. The family becoming dissatisfied, on June 12, 1907, I was dismissed, and Dr. J. C. Hill took charge of her. She lived some two or three weeks after this, and Dr. Hill did not make a diagnosis. Dr. Hill also told me that for some time before her death she had acute mania.

Case III: Mrs. L., age 50, married, six children. About the first of April, 1908, she sent to me for some medicine for sore mouth. After sending back several times, always with the same message that the medicine had done her no good, I visited her on April 10, 1908. Found her weak, emaciated, and with tongue slick and red, buccal surfaces red and inflamed; intense nausea, pain over epigastrium, intractable diarrhœa, temperature 100 to 101 deg. Fahrenheit; rectum, vulva, and vagina red, inflamed and great itching. In this case there was no typical eruption on back of hands, but some discoloration. Treatment did no good and the patient died April 22, 1908.

Case IV: Mrs. C., aged 40, married, four children. For several years patient has suffered woth articular rheumatism. During the winter of 1907 she had an eruption on back of hands which she called "chapped hands." I saw her May 3, 1908. She complained of dysentery and sore mouth. I treated her several days without any improvement, when my attention was called to an eruption on the

back of her hands, the eruption was the peculiar one that I had seen in my Cases Nos. 1 and 2, and I at once saw that I had pellagra to deal with. This patient presented the same train of symptoms as Cases 1 and 2. Peculiar eruption on hands, intractable diarrhoea, sore and inflamed tongue, mouth, vagina and rectum; temperature 100 to 102; pain over epigastrium; nausea. Dr. C. F. Williams, of Columbia, came up and saw the case with Dr. Gambrell and myself. He agreed in a diagnosis of pellagra, and advised symptomatic treatment, and gave as his opinion that the patient would not live 60 days. Her symptoms did not yield, and her condition was growing worse steadily. At one of my visits, one of his sisters said to me, "Doctor, you gave Ma a mouth wash when she had sore mouth, and it did her all the good." After thinking awhile, I recalled the fact that I had used hydrogen dioxide as a mouth wash with her mother. Nothing I had done had benefited my patient, and I was willing to try any suggestion, so I sent over some hydrogen dioxide and the patient began using it as a mouth wash. The sore mouth got better at once, and this suggested the internal use of it. I then gave 15 drops hydrogen dioxide in water, every three hours. She began to improve at once, and by June 1st all symptoms of pellagra had disappeared. This patient is still living, and under observation. I keep her on a tonic of iron, quinine and strychnine, cut out all products of Indian corn in her food, have her to eat generously of meat, and am giving soamin twice a week to prevent a return of the trouble. She is now doing her own house work.

A CASE OF PELLAGRA.

By Dr. R. A. Lancaster, of Columbia.

W. S., negro boy, aged 10 years, was brought to my office by his mother, who gave me the following history: For the past four years the boy has been getting sick in the early spring, suffering from sore mouth, persistent diarrhoea, soreness of spine and general muscular soreness and weakness, and during these attacks an eruption appears on hands, feet and face. She says that he begins to improve about the latter part of summer and gradually regains his health until the following spring.

His condition when I first saw him in April was distressing. His mouth and throat were so sore that he could not chew his food, and he was having from 15 to 20 stools during the twenty-four hours.

He was too weak to walk any distance, reflexes exaggerated, complained of much soreness of spine and limbs, whilst the back of his hands, forearms and lower extremities are covered with dry, dark exzematous eruption.

The sore mouth and diarrhoea improved temporarily under appropriate remedies, but he would have frequent relapses and exacerbations. After the middle of August the boy began to improve, and at the present time all trace of the disease, except a well marked anemia, have disappeared.

I made the diagnosis of pellagra upon the first visit, which diagnosis was afterwards confirmed by Drs. Williams and Watson.

THE SUPPOSED RELATIONSHIP OF DAMAGED GRAIN TO EPIZOOTIC CEREBRO-SPINAL MENINGITIS OF HORSES (BLIND STAGGERS).

By M. Ray Powers, D. V. S., Clemson College, S. C.

I will endeavor to give a brief history and description of this fatal disease of horses which is of common occurrence in this State during wet seasons.

Dr. Large, of New York, first reported this disease in America in 1850. In 1852 and 1865 it appeared in Denmark and Germany and a very serious outbreak was reported in Egypt in 1876, where about 6,000 horses, mules and asses perished in the vicinity of Cairo alone.

Since its first appearance in this country numerous outbreaks have been reported at frequent intervals. An outbreak occurred in Virginia and North Carolina in 1886 and assumed such proportion that the citizens of these States appealed to the United States Department of Agriculture for aid. In response to this appeal Dr. W. H. Harbaugh, of the Bureau of Animal Industry, was detailed to make an investigation, and his report was published in the third annual report of that bureau. Another serious outbreak occurred in Kansas in 1901 and was investigated by Dr. Mayo, of the Kansas agricultural experiment station. The result of his investigation was published in bulletin No. 24 of that station.

Frequent outbreaks of this disease have since been reported in New Jersey, Maryland, Virginia, North Carolina, South Carolina, Georgia, Louisiana and other Southern States. In fact, this disease now occurs almost every year in different portions of the United States and its appearance is now expected after rainy seasons sim-

ilar to those of 1901 and 1906, when molds and fungi thrive on fodder and grain.

The records of this office show that in 1901 this disease appeared in at least nine counties of this State and caused a loss which was estimated as exceeding $10,000. Following the wet summer of 1906, the disease again appeared and, judging from the cases already reported, we may expect another serious outbreak this winter.

Severe losses occurred in other Southern States during these same years and Dr. R. W. Hickman, of the Bureau of Animal Industry, spent considerable time investigating an outbreak in North Carolina. His report is now available as circular 122 of that bureau.

OCCURRENCE.

This disease does not seem to be confined to any particular locality, as it has appeared in the upper portions of the State as well as in the low country. We must admit, however, that outbreaks are more frequent in the low, flat country than in the hilly portions of the State. Horses are more frequenly attacked than mules; in fact, I have never had opportunity to see a mule so affected, although the loss of a few mules has been reported to this office.

Cases occasionally appear sporadically, although more frequently the disease extends over a large area, hence the name, "epizootic cerebro-spinal meningitis." It is rare, however, for this disease to attack all the horses on one plantation.

CAUSE.

While many theories have been advanced as to the causative agent, we are compelled to admit that the specific cause is unknown. The cause has been attributed to almost everything from unsanitary condition of the stables to sudden changes in the weather, but while we must admit that these may be predisposing causes, we know that they are not of primary importance as causative factors.

No pathogenic microbe or virus has ever been isolated as the causative agent. Furthermore, we have often noticed that the disease is not communicable from animal to animal, as it frequently happens that one horse in a team will be affected, while the mate, which has been kept under identically the same conditions (eating from the same manger, occupying the same stall, etc.), remains perfectly healthy. This and other observations lead us to believe that the

symptoms are produced by the introduction of some narcotic poison rather than by the growth of micro-organisms within the system.

If these symptoms are produced by micro-organisms our laboratory methods must be at fault or the organism must be ultra-microscopic. Furthermore, the inoculation into susceptible animals of defibrinated and citrated blood, as well as brain emulsion from affected animals, fails to produce these symptoms.

Poisonous plants have also been suggested as the possible cause. This can not be accepted, however, as a large number of animals which escape are kept under identically the same conditions as the animals which are affected.

As practically every outbreak occurs among animals receiving damaged corn or fodder, the majority of investigators believe the causative agent to be moulds or fungi which grow on the damaged food. From investigations and from reports received, it would be difficult to connect this disease with any particular food, although in the majority of cases the affected animals received damaged corn. This last theory is further strengthened by the observation that when owners of affected animals change the food and water completely no further cases develop, while in instances where owners refuse to do this new cases appear.

Dr. Mayo's investigations would lead us to believe that the symptoms are produced by a fungus (aspergillus glaucus), the spores of which enter the circulation and cause inflammatory conditions and abscess of the brain. He claims to have recovered this fungus from the lesions.

While damaged food is probably the most rational cause of the disease, we must not overlook the fact that several investigators have carried on feeding experiments with negative results. Dr. Law explains this and the fact that all animals receiving damaged food are not affected by stating "that the cryptogams probably vary under different conditions of life and elaborate various products at different stages of their growth, thus accounting for the presence of the disease at one time and its absence at another under what may seem identical conditions.

SYMPTOMS AND COURSE.

The symptoms usually exhibited are dullness and extreme muscular weakness (shown by hanging of the head and general depression). There is often a paralysis of the throat, making swallowing difficult or impossible and saliva drips from the mouth. Paralysis is occasionally present and the animals stagger and fall or may stand

with the legs braced or with body resting against the side of the stable. Twitching of the muscles is often observed, and, in some instances, a rigid contraction of certain groups of muscles is noticed. Animals often become delirious and stand with the head pressed against the wall or walk persistently in a circle. The temperature ranges from normal to 105 or 106 F., although in some instances it remains normal or even subnormal. Respiration is accelerated and the pulse variable, being soft and weak in some cases and hard and wiry in others. The visible mucosæ are usually very much congested.

Severe cases usually die within six to forty-eight hours, the animals falling after a short time and remaining in a comatose condition until death. In mild cases the animals may recover, but are seldom of much value, as they develop into what are commonly termed "dummies." The mortality in South Carolina is at least 95 per cent.

POST MORTEM APPEARANCE.

Congestion of the blood vessels of the meninges and medulla appear to be constant and areas of necrotic or softened nervous tissue are often found. In one instance, in particular, a softened amorphus area larger than a silver dollar was found in the left cerebral hemisphere. In connection with this I might add that the horse was apparently in normal condition when placed in the stable at night, but was delirious when found in the morning. This horse died within six hours and (as this lesion must have been well developed before the symptoms appeared) the hopelessness of medicinal treatment is at once apparent.

Five brains have been forwarded from this office to Washington, D. C., for examination and the following is an extract from a letter received from the acting chief of the Bureau of Animal Industry:

"Upon receipt of these tissues rabbits were inoculated intracerebrally with an oblongata emulsion, but they have thus far remained in an apparently healthy condition. Upon cutting through the hemispheres an irregular completely softened amorphous area was found in the right cerebral lobe of each of the four brains examined, the result of the breaking down of the cephalic tissue. The predilection for the motor areas of the cortex on the right ride was noteworthy. In brain No. 2 a complicating hemorrhage was observed around the necrotic mass, giving rise to hemorrhagic inflammation of the brain.

"On microscopic examination these areas of degeneration were found to be destructive rather than exudative and show the alterations in every respect similar to those found in epizootic leuco encephalitis, usually termed cerebro-spinal meningitis. The blood vessels in the substance of the brain present an inflammatory condition of the walls, some being ruptured, others distended. The surrounding tissue shows a perivasculitis and contains many infiltrated leucocytes. The softened centers appear as hyaline or gelatinous degenerated areas of a homogeneous nature, which is frequently seen to be contracted, leaving vacuoles containing a faint fibrinous material. The neuroglia fibrila show a gradual disintegration, which is also apparent in the neuroglia cells, the protoplasm of which fails to take any distinctive stain. These are finally broken down into the debris of the constituent parts.

"The location of the above described softened areas probably controls the various symptoms produced and would account for the different actions of the animals you have observed."

TREATMENT.

Medical treatment is unsatisfactory and practically useless, as the central nervous system has usually undergone such degenerative changes before the symptoms develop. When the animal is noticed before the ability to swallow is lost, a purgative is administered. When the animal is unable to swallow, hypodermic injections of eserin or arecoline hydrobromate or intravenous injection of barium chloride is given to accomplish the same purpose. Iodide of potassium is also given when possible.

As the disease is so fatal, every effort is made to check its spread. The animals are given complete change of food and water and moved to another stable if possible. The abandoned stable is then throughly disinfected and allowed to remain vacant for two or three months.

Where these measures are adopted, additional cases seldom develop.

THE PATHOLOGY OF PELLAGRA.*

By H. H. Griffin, M. D., State Hospital, Columbia, S. C.

It is generally conceded by the authorities on pellagra that our knowledge of the pathological findings is deficient and indefinite.

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CASE OF PELLAGRA, STATE HOSPITAL FOR THE INSANE, COLUMBIA, S. C. COURTESY OF DR. J. W. BABCOCK.

The anatomic changes are neither constant nor characteristic. In a chronic disease such as pellagra, presenting such a variety of symptoms, and with the frequent presence of intercurrent affections and senile evolutionary changes we could scarcely expect to find a definite single morbid condition.

Pellagra being a general disorder of the system, general nutritional derangements with more or less wasting of the adipose and muscular tissues are encountered. We find chronic degenerative changes, particularly atrophy and fatty degeneration in the liver, kidneys, spleen, intestines and lungs, the tissues being generally anaemic. Usually there are found abnormal pigmentary deposits such as we encounter in senility, especially in the ganglionic cells, the muscles of the heart, the hepatic cells and in the spleen.

Having in mind the clinical syndrome of intestinal, nervous and skin symptoms, we will consider the more constant changes in the above named systems. These are:

1st. The changes in the intestinal tract, according to Tuczek.

Atrophy of the muscular coat with occasional hyperaemia, and ulceration of the lower tract especially in rectum. Other observers assert that there is no ulceration.

2nd. Changes in the nervous system.

The most constant post mortem lesion is in the lateral columns of the spinal cord, and this condition seems peculiar to this disorder. It is in the dorsal region that we find these changes most pronounced, the condition being one of degeneration. We also note a degenerative condition in the posterior columns in the cervical and dorsal regions. Practically no changes are seen in the lumbar cord. In addition to the above Lombroso has found degenerative changes in the posterior roots of the spinal cord. He says that in the most typical cases the spinal cord lesions remind one of incipient tabes, except that few changes are found below the dorsal region, whereas in tabes the lumbar region is chiefly affected. Changes in the brain are generally negative. Occasionally there is fatty degeneration or calcification of the intima of small blood vessels, and pigmentations in the adventitial coats. Where the mental symptoms have been pronounced and of long standing an atrophy is found, as would be expected. In recent cases the degenerative cord lesions may be entirely absent or very slight.

3rd. The changes in the skin.

The eruption is of an erythematous type. Red spots first appear, which quickly become dark and desquamate; the surface beneath

the scaly covering is red, thickened, rough and fissured. In some cases the epidermis is shriveled and scaly, the whole affected part having a blackish appearance.

The blood findings are such as we would observe in cases of marked anaemia. Slight lymphocytosis is observed.

In conclusion, it is manifest that our knowledge of the pathology of pellagra is meagre, and it seems to me that it is important at this stage of our studies that we have authoritative records of the pathological findings in a number of cases well distributed geographically which harmonize with the recognized pathology of European and Egyptian cases. I will quote the history and pathology of a single case from Sandwith:

An Egytian woman, aged 33, was admitted under my care December, 1898, complaining of diarrhoea, general debility and pain in the lower extremities. She was extremely emaciated and weighed only 67 pounds. The symptoms of pellagra were: slight roughness and atrophy of the skin on the dorsal aspect of the hands and feet, and also on the back; a very denuded tongue, diarrhoea, and motions passed in bed; dementia with persistent muttering, absent knee-jerks, some sleeplessness and tenderness on both sides of the spine near the 3rd dorsal, and 1st, 2nd and 3rd lumbar vertebrae. The diarrhoea was somewhat checked in hospital, though her mental condition required bromide and chloral. The temperature was practically normal throughout, but her weight fell persistently to 61 pounds, and she died Jan. 23, 1899, twenty-six days after admission. No ankylostoma eggs were found in the faeces, though she was extremely anaemic. The urine contained a distinct trace of albumen. At the post mortem examination there was slight atheroma of the aorta; the heart weighed 185 gms; the lungs were congested and oedematous, but otherwise normal; the liver weighed only 790 gms., the spleen 140 gms.; the kidneys each weighed 70 gms. and were markedly cirrhotic; the brain weighed 1050 gms., and was very oedematous, while the membranes of the vertex were thickened and opaque.

The spinal cord, when examined in London, showed marked changes.

(1) Marchi's Method: The paleness of the posterior columns was very noticeable, but under the microscope no recently degenerative fibres could be seen. The cells of the anterior horn were pigmented.

(2) Weigert-Pal Method: The lack of fibres in the posterior columns was very marked, both sacral and lumbar regions being affected equally. In the middorsal region a pair of normal roots entered the cord, and wedged itself in between the atrophied fibres of the median and external columns; this root could be traced up to the upper cervical region, where again the incoming roots contained

more normal fibres. A small wedge-shaped tract was also visible, just outside the anterior horns in the cervical region.

(3) Aniline-blue-black method: The increase of the connective tissue in the posterior columns was very marked and distributed itself in exact correspondence with the condition of the roots above described, namely: a pair of roots which had undergone no degeneration in the dorsal region showed no increase of the connective tissue in the area it occupied in the cord. There was no increased vascularity of the cord, the cells of the anterior horn and the nucleus and nucleolous were distinct. The increase of the connective tissue was limited to the posterior columns except in the wedge-shaped tract above described in the cervical region, which appeared darker owing to the smallness of the film in this area.

(4) Van Geison's Method: There was no evidence of any recent inflammatory action in the gray matter. There was some thickening of the walls of the blood vessels, especially in the posterior columns, though it was not limited to this region.

The posterior roots of the cervical, dorsal and lumbar regions were also examined in this case by the Marchi, Weigert-Pal, and Stroebe methods. Marchi's method showed very little recent degeneration, though it was obvious from the lack of staining that a very considerable amount of degeneration had taken place, and this was made evident by staining by the Weigert-Pal method. The greatest amount of destruction seemed to have taken place in the dorsal and lumbar regions, and to a lesser extent in the cervical region; the same condition was also shown by the Stroebe stain; only a few axis cylinders could be seen in each root. The patient died of pellagra and chronic kidney disease, but there was no possibility of knowing how many years she had suffered from pellagra.

The cord degeneration would appear to be of root origin and affects the extra-medullary, as well as the intra-medullary portion of the posterior roots. The degeneration in the cervical region of this cord was most marked in the columns of Goll, the columns of Burdach being affected to a lesser degree.

Since then I have had many other sections cut for me which have been examined by experts, but unfortunately nothing of pathological interest was revealed. The absence of cord degeneration in these cases was due to the fact that the patient had either had pellagra for too short a time, say one year or less, or that though they had suffered from pellagra for three or more years, the clinical signs of the disease were not very far advanced. In other words, spinal cord degeneration as discovered by the miscroscope is a comparatively late lesion in the disease.

REPORT ON THE EXAMINATION OF THE EYES IN EIGHTEEN CASES OF PELLAGRA.*

By E. M. Whaley, M. D., Columbia, S. C.

In making this report it will be remembered that the patients we are dealing with are insane, which would naturally make us expect to find the nervous element much in prominence. They are usually well advanced in the disease, except a few in whom it has recently developed. As this is a preliminary report we would better leave our conclusions to some later date when we can add the results of the examinations of a larger number. All pellagra patients are unresponsive and no field examinations could be made. With few exceptions the examination had to be made while the patient was in bed.

The dilated pupil so much spoken of in reports on this disease is conspicuous by its absence, in the eighteen cases herein reported on, it having occurred in only one case. Two cases resisted the action of homatropin for two hours, where the others reacted in the usual twenty minutes. Hypersensitiveness to light with contracted pupils was the rule. It is possible that the dilating center for the pupils being lower down and subject to more sensitive stimuli is counteracted to such an extent in cerebrally irritative subjects where the higher centers are affected, that we have a contraction of the pupil in insane cases, which we do not have in those cases where the medulla and ciliospinal centers, with which the skin (sensory) reflexes are concerned, are involved—as in milder cases where the higher centers are not involved we have a dilated pupil.

In acute cases with overwhelming poison at the season when this poison seems to be most virulent we should expect to find a dilatation of the pupil due to a paresis of all the pupillary centers.

Shallow anterior chambers were found in sixty-four per cent. of the cases. Intra-ocular tension plus in only one case, and that only in one eye. In six cases the corneal sensibility was subnormal, two hypersensitive, and one could not be tested on account of perforating corneal ulcer, and another having paresis.

Strabismus could not be detected when there was not other evident cause, and nystagmus, which is dependent on the muscular paresis, was absent.

Where the gastro-intestinal symptoms are very prominent and the inflammation extends to the mouth and post-nasal space, we find an

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obstruction of the lachrymal duct, due to contiguity of surface. This was noted in five cases, all of which had the mucous membranes very much affected by the disease.

The Argyle-Robertson pupil was present in one case. Sensory reflex could not be elicited except in one case, and that one only a suspected case.

The retinæ, optic nerves, and internal eye structures fail to give any symptoms that we could put down as distinctive of pellagra. Some arteriosclerosis, optic nerve and retinal inflammation were present, but could not be said to be due to pellagra.

I hope to be able to keep us this investigation as we have access to more and more cases. Apart from these insane cases I have had the good fortune to have examined three cases in the last year who have since developed this disease, and all of these had photophobia of slight degree without the inflammatory changes that usually accompany this symptom. There was no record as to the pupils at the time, but I remember one to have been dilated and the other to have been contracted. The third one could not be seen on account of the whole cornea being covered with a superficial opacity, not due to previous inflammation, and which disappeared entirely under the use of dionin in three weeks without reaction. A point for consideration in all these results is the season in which the examination is made, as it is reported by our Italian friends that, whatever the poison may be, it is more virulent at some times of the year than at others.

We are not trying in this paper to bring forward any new symptoms, but to encourage investigation and thought as to the early manifestations of this dreadful disease that may show itself in the eye early enough for us to institute intelligent treatment before our patient is beyond assistance.

THE DIAGNOSIS AND TREATMENT OF PELLAGRA.*

By J. W. Babcock, M. D., Columbia, S. C.

The writer visited several institutions in Italy in July, 1908, and satisfied himself that the cases there diagnosed as pellagra are one and the same disease as he had seen in South Carolina and made the subject of a special preliminary report to the S. C. State Board of

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Health in December, 1907. This identification of the two diseases was made the subject of an Associated Press dispatch from Milan, July 15th, at the instance of Mr. W. Bayard Cutting, Jr., deputy consul general of the United States, who had become especially interested in the subject. It is hardly necessary to give full particulars of the visits leading up to this identification at the present time, but the brief notes taken on several of the more interesting cases may be worthy of record.

Case I: Female, 54 years old; has had signs of pellagra two years; has improved lately, but still has dermatitis on shoulders and chest. The skin on the backs of hands and forearms is dry, thickened and pigmented. She still has diarrhœa, but no stomatitis.

Case II: Female, admitted one year ago; said to be in stage of remission, but the skin on hands, forearms and forehead is dry and discolored. She has slight diarrhœa and is demented. She has also a trilobed goitre.

Case III: Female; skin on hands and face now dry and thickened; has had diarrhœa.

Case IV: Female; age about 30 years; confined to bed; a case of "pellagra sine pellagra." She does not show any eruption, but the mental symptoms are those of marked delirium of exhaustion. She has some diarrhœa. The diagnosis in these cases must be made from the especial type of mental symptoms, the diarrhœa, and the increased knee-jerks, as well as from the fact that she comes from a family of peasants whose food is largely maize and who live in a region known to be especially pellagrous. The prognosis as to life in such cases is very unfavorable.

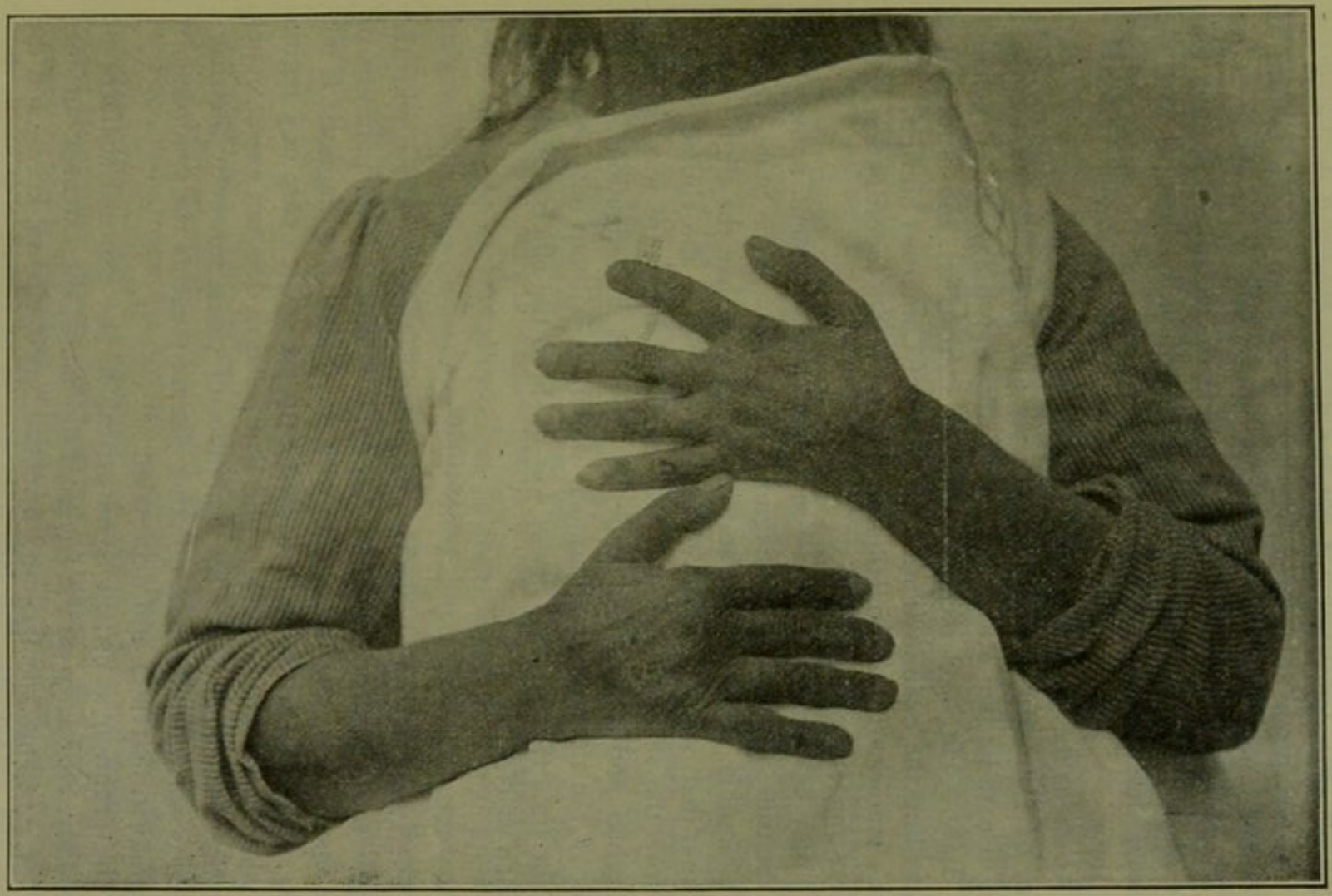
Case V: Male, 60 years old; skin on backs of hands and feet dry and brown; has intestinal form of pellagra, which is not now well marked. Mentally, a case of melancholy with stupor.

Case VI: Male of 50 years; a typical case; erythematous eczema on backs of hands and feet; feet also swollen and scaly; tongue red, rough, cracked and slightly slimy; has diarrhœa, and is melancholy.

Case VII: Man, 30 years old; hands red; feet very black, encrusted and swollen. Skin over upper part of chest red and scaly; face erythematous; has stomatitis; all reflexes increased, especially knee-jerks.

Case VIII: Male, hands and feet show usual results of pellagrous inflammation, the feet in particular being swollen, black and scaly; all reflexes exaggerated; ankle clonus formerly present also (a rare symptom). Has some nystagmus now, but formerly it was marked. He has also had Romberg's symptom.

Case IX: Male; very hypochondriacal; has erythema of hands and feet; nails of hands and feet thickened and black; tongue red and rough; neck pigmented from previous inflammation. This is a case of the "crossed form" of pellagra, that is, the left hand and right foot are considerably involved with pellagrous inflammation, while the right hand and left foot are only slightly affected.



CASE OF PELLAGRA, MOMBELLO ASYLUM, ITALY. COURTESY OF ASYLUM OFFICIALS.

I saw a number of other cases at Mombello, either in the stage of remission or convalescing. All were adults over 30 years old. Mentally, all were depressed and melancholy.

The symptoms presented by these cases seen in the asylum at Mombello, near Milan, as well as by those I saw in the pellagrosario at Mogliano, near Venice, were sufficient to confirm the opinion that the disease I have observed in South Carolina is identical with the disease described as pellagra by the physicians and medical writers of Italy.

At about the same time, that is early in July, 1908, Dr. J. J. Watson, of Columbia, S. C., visited Italy and independently identified the disease he had studied at home with the disease called pellagra in Italy. He also received especial instruction and information regarding the examination of maize from Lombroso himself. Watson's studies of, and contribution to, the whole investigation are of great importance and value.

I may be pardoned for one more personal statement, namely, that at the time of the original diagnosis of pellagra in the State Hospital in Columbia, no one of the staff knew, or even had the slightest intimation that pellagra had ever been recognized in the United States.* Before the report was published, available literature was carefully searched and references made to previous investigations, as was proper, but the tentative diagnosis was made without outside help up to the time that an oral report was made to the officers of the State Board of Health.

DIAGNOSIS.

The symptoms and pathology of pellagra seem to class it with the degenerative nervous diseases like syphilis and general paralysis of the insane. To one unfamiliar with the disease the diagnosis is not easy, especially when he is dealing with an isolated case in a region in which he is authoritatively told that pellagra does not exist. After familiarizing one's self with a number of cases, however, the diagnosis is not difficult, since no other disease presents the syndrome of dermatitis, diarrhœa and depression.

The eruption is usually symmetrical on the exposed surfaces of the body, especially on the backs of the hands, the extensor surfaces of the forearms, the face and forehead, neck, elbows, knees, and the

*Jan., 1909.—Since the publication of the report on pellagra in the 28th Annual Report of the South Carolina Board of Health the disease has been recognized in the asylums of North Carolina, Georgia, Florida and Mississippi, and the Baptist Orphans' Home in Tennessee. It had already been recognized in the State hospitals of Alabama and South Carolina.

backs of the feet if the patient goes barefooted. The skin lesions are asymmetrical when the exposure is not symmetrical. The tongue is denuded or bald.

Insomnia is a prominent symptom. The knee-joints are at first increased, then diminished, and finally abolished. They may react differently on the two sides. There is usually pain on pressure in the dorsal region. Digestive disturbances are an early, and an obstinate diarrhœa a late symptom.

In the later stages nervous and mental symptoms occur: headache, vertigo, ringing in the ears, paraesthesias and neuralgias. It is said that in the final stage of the established disease, while melancholia is the common type, yet frequently melancholia, nausea and "folie circulaire" are so intermingled as to constitute the clinical picture of pellagrous insanity.

Remembering that cases often become insane after the disappearance of the dermatitis, it is important that asylum physicians should be on the alert for cases of "pellagra sine pellagra." Students in the London School of Tropical Medicine are warned that they should be able to make the diagnosis of pellagra regardless of the presence or absence of the eczematous eruption. The resemblance between this condition and acute delirium should be kept constantly in mind.

These comments from Warnock are pertinent: "The dark, flaky rash of pellagra leaves the diseased skin paler than that of the surrounding parts, with a darker areola along the line where the diseased meets the healthy skin. This paleness does not always persist, but gradually the skin assumes a brownish, shrivelled appearance and its texture becomes thinner, especially around the neck." The sites of the old rashes are those exposed surfaces of the body already described.

TREATMENT.

Prophylaxis: Theoretically, at least, much is to be expected in the line of prevention. If damaged corn causes pellagra, then under the Food and Drugs Act proper inspection should be maintained over shipped corn at least. Other efforts will be directed towards the proper cultivation, drying and housing of maize. But even with the example of the Italian laws before us, and the benefits said to accrue from them, it is hardly to be expected that our lawmakers will pass any stringent laws until it is established how widely pellagra exists in this country, and whether or not damaged maize is the sole cause of the disease. In a year's time it is hardly to be expected that our

ideas on the subject of pellagra should be fully matured. The surprise is that the belief in the existence of pellagra in our country should have been even tentatively accepted by so many phyisicans in so short a time. The whole problem is in its incipiency, and many of its factors are sub judice.

Diet: As a rule, the patient should not be allowed any food derived from Indian corn. It is best to deny the products of maize known to be sound, in view of the possibility of introducing a new supply of the unknown poison. A generous dietary should be given, including fresh meats and vegetables.

Therapeutics: Quinine, cod liver oil and other tonics are beneficial, but to Lombroso belongs the credit of introducing arsenic as a remedy for pellagra, and the experience has proven this the most useful single remedy. It is usually given in the form of Fowler's solution, up to ten drops thrice daily, with, of course, the usual precautions. Some writers have expressed the opinion that ultimately a sero-therapy will be applicable to cases of pellagra.

When the commonly associated anchylostomum worms are found they should be removed by thymol, or the combination of eucalyptol, chloroform and castor oil.

In the spring of 1907, Babes, of Bucharest, began to employ atoxyl in the treatment of pellagra. He gave it subcutaneously in doses of gr. 1 1-2 to 3, in aqueous solution, every 4 to 7 days. In all 65 cases were treated, of which 35, or 53.8 per cent. were cured after a few weeks; 24, or 37 per cent. showed marked improvement; and 6, or 9.2 per cent. were not improved. Control cases treated at the same time without atoxyl showed practically no improvement.

A recent (1908) report from the Cairo, Egypt, asylum says in conclusion about atoxyl: "Without taking too favorable a view as to the duration of the success, we can already maintain that a means has been discovered in the treatment of this terrible malady, which can do more than all others tried heretofore, and which seems destined, in combination with rational diet, to check the misery caused by this disease."

As to the use of atoxyl in the State Hospital at Columbia, our experience extends over six weeks only, and while still too limited for a definite or final opinion, we have arrived at these tentative conclusions:

1. The most striking effect is the control of the severity of the diarrhœa. The stools are reduced from 15 or 20 a day to 3 or 4 in most instances; and in recent cases the diarrhœa appears to be cured.

2. In recent cases in the first stage the rash disappears after 3 or 4 injections of grains 1 to 2 given 4 to 7 days apart.

3. Unmixed cases of the second stage slowly improve, both as to the rash and diarrhœa.

4. Mixed cases and cases in the third stage do not improve either as to intestinal or skin symptoms.

5. We have noted no marked mental improvement indicating recovery in any of our cases. Their minds seem damaged, and while it is too early for final conclusions, their condition suggests dementia.

6. It is but fair to add that similar benefits have been observed heretofore following the use of Fowler's solution.

PROGNOSIS.

In one word the prognosis of pellagra has been said to be unfavorable. If, however, it is recognized in the prodromal or first stage, and the patient be removed from the alleged cause and properly treated, recovery may follow. In the second stage the outlook is less favorable; while in the third stage it is absolutely hopeless. Such has been the unanimous verdict regarding this dread disease until within the last year when a new development of the old treatment by arsenic has been introduced. These are the organic preparations of arsenic, atoxyl, soamin, etc., which appear to hold out some encouragement, except in advanced or exhausted cases.

Finally, I trust I may be permitted to submit some of the conclusions I have arrived at, here and elsewhere, regarding the condition we have met today to discuss. These are:

CONCLUSIONS.

1. From accumulating evidence there seems to be no doubt of the existence of a pellagroid disease in North Carolina, South Carolina, Georgia, Alabama, and Tennessee.

2. While sporadic cases have been observed also in other States—Louisiana, Texas, Arkansas, Virginia and elsewhere—reported statistics are not yet sufficient for a final statement regarding its presence to any extent.

3. Although the malady presents many points of resemblance to the Italian and Egyptian forms of pellagra, yet striking differences are to be recognized.

4. Among these differences may be noted the overwhelming number of females attacked; the great and early mortality of the disease;

the unusual invasion by the eczematous rash of unexposed parts of the body, notably the inner side of the thighs, the coccygeal region, the vulva and anus, as well as upon the usually described exposed sites of the disease.

5. Yet these differences need not impair the diagnosis of pellagra, since similar differences are noted in cases in adjoining provinces in Italy and elsewhere.

6. By exclusion we are driven to the diagnosis of "possibly true pellagra" (Lavinder) in several Southern communities.

7. The cause of the malady has not yet been determined, but the association of all our cases with corn cannot be denied.

8. Some observers are raising the question of its communicability which, therefore, also calls for investigation.

9. Treatment by atoxyl (and probably by its congener, soamin) holds out the best hope from drugs, but the older Fowler's solution cannot yet be cast aside.

The attention of the board having been forcibly directed, by the result of the conference, to the existence of pellagra in our State, efforts were made through this office to determine its prevalence and distribution by sending to all of the physicians in the State the following letter of inquiry:

Columbia, S. C., Oct. 31st., 1908.

Dear Doctor: The recent observation of a number of cases of so-called pellagra in several Southern hospitals for the insane, and the publication of reports and papers on the subject, serve to render at this time the question of existence and prevalence of such a disease as pellagra of interest.

My apology for troubling you for a personal opinion as well as for statistical information, is the wide-spread interest involved in the problem.

An early answer to the appended questions will be greatly appreciated.

Yours truly,
C. F. WILLIAMS,
Secretary and State Health Officer.

No. 1. Have you had cases like pellagra, or with an eczematous eruption on the exposed surfaces of the body accompanied with diarrhœa and mental depression?

No. 2. How many cases have you seen?

No. 3. How long since you recognized the disease?

No. 4. If at all, how long do you think the disease has existed in your section?

No. 5. What proportion of cases were male, ...; female, ...; white, ...; black, ...?

No. 6. What was the occupation of those affected?

No. 7. Do the products of Indian corn (hominy and meal) form a part of the dietary of the patients, and are these products derived from native or shipped corn?

No. 8. Were your patients from poor, ...; moderate, ...; or well-to-do classes, ...; and were they from the city, ...; town, ...; or country, ...?

This inquiry has brought out the following facts: That of the 942 circulars sent out, 269 replies have been received—89 in the affirmative and 180 in the negative. Of the 89 answers in the affirmative we have reported 187 cases; 109 white, 78 colored; 57 males, 130 females.

A tabulation of the reports received from the various counties show the following distribution: Abbeville, 13 cases; Aiken, 3; Anderson, 8; Calhoun, 1; Charleston, 21; Chester, 20; Darlington, 3; Dorchester, 4; Fairfield, 1; Florence, 2; Georgetown, 5; Greenville, 2; Greenwood, 9; Hampton, 3; Horry, 2; Kershaw, 9; Lancaster, 10; Laurens, 6; Lee, 6; Lexington, 1; Marion, 4; Newberry, 4; Oconee, 2; Orangeburg, 1; Pickens, 4; Richland, 7; Saluda, 3; Spartanburg, 20; Sumter, 3; Union, 2; Williamsburg, 2; York, 6.

The remaining counties—Bamberg, Barnwell, Beaufort, Berkeley, Cherokee, Chesterfield, Clarendon, Colleton, Edgefield and Marlboro—report no cases, though only 18 reports were received from these counties, while our list shows that 148 physicians are engaged in practice in them.

No cases admitted to the State Hospital for the Insane are included in the above tabulation, though the records of that institution show that all of the counties except three from which no cases were reported should be included in the affirmative column. It is to be regretted that our inquiry was not more generally answered, and this

fact prevents us giving with any degree of accuracy the number of cases of pellagra in our State.

In answer to the question, how long do you think the disease has existed in your section? a period of 23 years is covered, though by far the majority of physicians do not go back farther than 5 years. It might be inferred that the reason for this is due to the fact that the younger members of the profession are the ones from whom the reports were received. This, however, is not the case, for many reports were received from men who have been in practice for twenty or more years, and they do not recall cases farther back than five years. It is the general opinion that the disease is on the increase, and if we accept the maize theory as the cause of pellagra, this increase may be explained, as pointed out by Dr. McCampbell, to our present method of harvesting corn.

The occupation given in the reports received is as follows: Housewife, 40; laundress, 6; carpenter, 3; laborer, 30; cook, 5; minister, 1; farmer, 20; housemaid, 3; mill operative, 34; prostitute, 1; school teacher, 1; student, 2; convict, 3; merchant, 1; clerk, 2; saleslady, 1; children, 18; not stated, 17.

Question 7, as to whether Indian corn, native or shipped, formed a part of the patients' dietary, was not as generally answered as could be desired. The answers, however, are sufficient to implicate both home-raised and imported corn. The histories given by Dr. Frontis, of Ridge Spring, of three children in a miller's family are among the most interesting from this standpoint yet reported. While this question was not answered by all physicians, the general assumption may be made that a very large majority of these patients, if not all, have eaten the products of Indian corn. So that the association of cases of pellagra with Indian corn cannot be lightly set aside.

Replies to question 8, referring to the social conditions of the patients, indicate that the majority are from the poorer classes, though not all were confined to this class, for a good number were reported in moderate circumstances, and quite a few belonged to the well-to-do class. The answers also show that 103 dwelt in towns or villages and 84 in the country. The latter condition, we believe, is due to the fact that the town cases were more generally reported than those in the country.

Summing up, it will be seen that only about one-fourth of the physicians replied to the inquiries sent out, and from this number we have reported 187 cases, the distribution of which indicates that the disease prevails in nearly every county in the State. Making allow-

ance for possible duplication of cases in the reports received, and considering the number of physicians who did not reply, we believe a conservative estimate would place the number of cases of pellagra in South Carolina at about 500.

With the preliminary report made last year by the medical officers of the State Hospital for the Insane as a basis, our board has pushed forward its investigations as rapidly as scientific accuracy would permit, and we believe that we have now brought forward sufficient evidence to stimulate every physician in the State to take renewed interest in the subject of pellagra, so that in the future we may lay before the world data of sufficient accuracy to form a foundation for investigations that will lead to solving the problems of the origin, prevention and cure of the disease.