Transactions of National conference on pellagra : held under the auspices of South Carolina State Board of Health at State Hospital for the Insane, Columbia, S.C., November 3 and 4, 1909.

TRANSACTIONS

OF

NATIONAL CONFERENCE

ON

Pellagra

Held Under Auspices of

South Carolina State Board of Health

at State Hospital for the Insane, Columbia, S. C.
November 3 and 4, 1909

COLUMBIA, S. C.,
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1910.

Called Meeting of Executive Committee of the State Board of Health to Conduct National Conference on Pellagra.

Columbia, S. C., Nov. 3, 1909.

The First National Conference on Pellagra was held under the auspices of the South Carolina State Board of Health at the State Hospital for the Insane, Columbia, South Carolina, November 3 and 4, 1909.

November Third—First Session.

The conference met at 2:30 p. m., and in the absence of the Chairman, Dr. Wilson, was called to order by Dr. C. F. Williams, Secretary of the State Board of Health, who introduced Governor Ansel to welcome those who had assembled to take part in the conference.

Governor Ansel was warmly received and his address heartily applauded.

The address of welcome was responded to, on behalf of the visiting delegates, by Dr. Wm. C. Woodward, Commissioner of Health, Washington, D. C., at the conclusion of which Dr. J. W. Babcock said: Governor Ansel has referred to me as having been the discoverer of pellagra in this country. Pellagra was discovered in this country many years before I made my observations upon it. It was discussed at a meeting of the asylum superintendents in Washington as far back as 1864. It was observed in Brooklyn, New York, by Dr. Sherwell, an eminent dermatologist. It was observed twenty-two years ago, so I have been informed, by Dr. Bass, of New Orleans, by Doctor Bemis of that city. It has probably prevailed in this institution for a generation. Furthermore, Dr. Harris, of Georgia, made a notable observation upon pellagra in 1902, and the hero of our modern civilization, the country doctor, observed it several years ago, notably Dr. McConnell, of Chester, S. C., also Dr. Wright, of Lincolnton, N. C., and Dr. Bellamy, of Wilmington. The disease was observed in the Alabama Asylum for Colored Insane before observations had been made in the South Carolina Hospital, so that if you and the Governor will pardon me, I do not wish to be placed in the false position of making any claims as to having discovered or made any early observations upon pellagra, because such is not the

case, and credit is due to all of the gentlemen whose names I have mentioned, and perhaps there are others. I am sure everyone of us is interested in bringing out the whole truth with regard to the pellagra problem, and we are all here in the interest of truth and justice to those gentlemen who are entitled to priority long before any observations that may have been made of this disease in this institution.

At the conclusion of Dr. Babcock's remarks the reading of papers was begun.

Dr. F. M. Sandwith, F. R. C. P., Gresham Professor of Physic, London, England, contributed a paper entitled "Introductory Remarks", which was read by Doctor Babcock in the absence of the author.

Assistant Surgeon General Kerr, U. S. P. H. and M.-H. Service, Washington, D. C., read a paper entitled "Pellagra: As a National Public Health Problem."

Mr. E. J. Watson, Commissioner Department of Agriculture, Commerce and Industries, Columbia, S. C., read a paper entitled "Economic Factors of the Pellagra Problem in South Carolina."

Passed Assistant Surgeon C. H. Lavinder, U. S. P. H. and M.-H. Service, Washington, D. C., read a paper "Notes on the Hematology of Pellagra."

These papers were discussed by Drs. R. N. Greene, Chattahoochee, Florida; C. C. Bass of New Orleans, Louisiana; Walter H. Buhlig, Chicago, Illinois, and Louis LeRoy, Memphis, Tennessee.

Dr. George A. Zeller, Superintendent State Hospital, Peoria, Illinois, read a paper entitled "Pellagra: Its Recognition in Illinois and the Means Taken to Control It."

Dr. J. F. Siler and Dr. H. J. Nichols, Captains Medical Corps, United States Army, read a joint paper entitled "Aspects of the Pellagra Situation in Illinois."

These papers were discussed by Drs. C. L. Minor, Asheville, N. C.; C. F. Williams, Columbia, S. C.; L. J. Pollock, Dunning, Ill.; C. H. Lavinder, Washington, D. C.; J. W. Mobley, Milledgeville, Ga.; J. W. Babcock, Columbia, S. C.; Julius C. Sosnowski, Charleston, S. C.; M. B. Young, Rock Hill, S. C.; H. E. Menage, New Orleans, La.; John W. Thomas, Pineville, La.; Geo. A. Zeller, Peoria, Ill., and I. W. Faison, Charlotte, N. C.

Dr. Isadore Dyer of New Orleans, La., contributed a paper entitled "Some Differential Points in the Skin Lesions of Pellagra.

Report of One Case With Removal of Symptoms," which was read by Dr. Menage in the absence of the author.

At this juncture the chairman announced the chairman of the following conference committees: The Committee on Publication, Dr. C. F. Williams; the Committee on Permanent Organization, Dr. Geo. A. Zeller; Committee on Statistics, Asst. Surgeon General Kerr; Committee on Resolutions, Dr. J. Howell Way; Committee on Ways and Means, Dr. J. M. Buchanan.

On motion, these nominations were confirmed by the conference, and the chairman of each committee empowered to select four additional members.

On motion the conference then adjourned until 8:30 p. m.

First Day—Evening Session.

The conference re-assembled at 8:30 p. m., and was called to order by Dr. W. J. Burdell, member of the South Carolina State Board of Health, Lugoff, South Carolina.

Dr. Geo. F. Gaumer, Yucatan, Mexico, contributed a paper entitled "Pellagra in Yucatan."

Dr. C. J. Manning, M. R. C. S., England, L. R. C. P. Ed., Medical Superintendent Lunatic Asylum, contributed a paper on "Psilosis Pigmentosa in Barbadoes", which was read by Dr. White in the absence of the author.

A contribution entitled "Pellagra in Jamaica" by Dr. D. J. Williams, Medical Superintendent, The Asylum, Kingston, Jamaica, W. I., was read.

Dr. A. Marie, Paris, France, contributed a paper entitled "Pellagrous Insanity Among the Arabs in Egypt", which was read by Dr. Mason in the absence of the author.

Dr. John Warnock, Medical Director, Government Hospital for the Insane, Abbassia, Cairo, Egypt, contributed a paper on "Pellagra in Egypt."

A paper entitled "A Note on Pellagra in Egypt", by R. G. White, M. D., Director, Serum Institute, Abbassia, Cairo, Egypt, was read by Dr. Reid Hunt.

On motion the conference then adjourned until Thursday, 10:00 a. m.

November Fourth—Second Day Morning Session.

The conference met at 10:00 a. m., and was called to order by Dr. Robert Wilson, President of the State Board of Health of South Carolina.

As there were so many papers on the program to be read, Dr. Wm. C. White moved that the reading of each paper be restricted to fifteen minutes and that the time allowed to any person for discussion be limited to five minutes.

The motion was duly seconded and carried.

Dr. E. B. Saunders, State Hospital, Columbia, S. C., read a paper entitled "The Surgical, Gynecological and Obstetric Aspects of Pellagra."

Dr. C. L. Minor of Asheville, N. C., moved that the cases of pellagra be now exhibited before the rest of the program is proceeded with.

Seconded and carried.

Several clinical cases were then presented and demonstrated by Drs. J. J. Watson, J. W. Babcock, H. E. McConnell and others.

Dr. J. W. Mobley of Milledgeville, Ga., read a paper entitled "Pellagra: Its Relation to Insanity and Certain Nervous Diseases."

This paper was discussed by Drs. W. H. Dial, Laurens, S. C.; J. W. Babcock, Columbia, S. C.; M. B. Young, Rock Hill, S. C., and B. R. Tucker, Richmond, Va.

Dr. J. H. Taylor of Columbia, S. C., read a paper entitled "The Question of Etiology of Pellagra."

This paper was discussed by Drs. William Allen, Charlotte, N. C.; Walter H. Buhlig, Chicago, Ill.; H. J. Nichols, United States Army; J. J. Watson, Columbia, S. C.; W. H. Dial, Laurens, S. C.; Hiram Byrd, Jacksonville, Fla.; J. D. Jones, Sweetwater, Ala.; Louis Leroy, Memphis, Tenn.

Mr. J. Swinton Whaley, Edisto Island, S. C., contributed a paper entitled "Personal Experience With Damaged Corn", after which Dr. Taylor was called upon to close the discussion.

Dr. C. C. Bass of New Orleans, La., read a paper entitled "Complement Fixation in Pellagra: Further Observations."

Dr. Howard Fox of New York City read a paper entitled "The Wassermann Reaction (Noguchi Modification) in Pellagra."

Drs. H. P. Cole and G. J. Winthrop of Mobile, Ala., contributed a joint paper entitled "Transfusion of Blood in Pellagra." This paper was read by Doctor Mason, Bacteriologist of the Alabama State Board of Health, in the absence of the authors.

The paper was discussed by Drs. J. H. Taylor, Columbia, S. C.; C. C. Bass, New Orleans, La.; M. B. Young, Rock Hill, S. C., and the discussion closed by Dr. Fox.

Dr. J. M. King of Nashville, Tenn., contributed a paper entitled "Report on Pellagra in Nashville, Tennessee", which was read by Dr. Louis Leroy in the absence of the author.

This paper was discussed by Dr. C. H. Lavinder of Washington, D. C., and Dr. W. E. Hibbert, Nashville, and the discussion closed by Dr. Leroy.

The chairman announced the following additional members of the Committee on Resolutions: Drs. Charles L. Minor, J. T. McAnally and C. C. Bass.

The additional members of Committee on Permanent Organization: Drs. Louis Leroy, E. M. Mason, W. O. Nisbet and B. L. Eiker.

Dr. H. E. McConnell of Chester, S. C., read a paper entitled "Facts and Theories of Pellagra."

The paper was discuseed by Dr. M. B. Young, Rock Hill, S. C., and in closing by the author of the paper, after which the conference on motion adjourned until 2:30 p. m.

Second Day—Afternoon Session.

The conference re-assembled at 3:00 p. m., with Dr. Wilson in the chair.

Dr. Wm. Allen of Charlotte, N. C., read a paper entitled "Ameba in Stools in Pellagra."

Discussed by Dr. H. J. Nichols, and in closing by the author of the paper.

Dr. J. J. Watson of Columbia, S. C., followed with a paper entitled "Symptomatology of Pellagra."

This paper was discussed by Dr. C. L. Minor, Asheville, N. C., and in closing by Dr. Watson.

Dr. T. W. L. Bailey, Clinton, S. C., read a paper entitled "Personal Observations on Pellagra."

Dr. B. L. Eiker of Leon, Ia., presented the following report of the Committee on Permanent Organization:

Mr. President: Your Committee on Permanent Organization begs leave to submit the following report: We recommend the formation of a National Association for the Study of Pellagra, and we further recommend that the next congress be held at Peoria, Illinois, in the month of June, 1910. Recognizing the distinguished services of Dr. J. W. Babcock of Columbia, S. C., in organizing and promoting this movement, we respectfully place him in nomination for president of this association. For first vice-president we recommend

Dr. Wm. A. White of the Government Hospital, and we further recommend that one vice-president be selected from each State in the Union interested in the study of this disease, said official to be appointed by the Secretary of the State Board of Health of each State. We further recommend that a permanent secretary and treasurer be elected by this association.

After reading the report Dr. Eiker moved its adoption, which was seconded by Dr. Louis Leroy and carried.

As Peoria, Illinois, was selected as the place for the next meeting, Dr. Louis Leroy nominated for Secretary-Treasurer Dr. Geo. A. Zeller, which motion was seconded by Dr. J. J. Watson and carried.

On motion of Dr. C. L. Minor the Committee on Permanent Organization was instructed to formulate a Constitution and By-Laws and present same at a subsequent session of the conference.

The Committee on Resolutions presented the following report:

Report of the Committee on Resolutions.

Dr. J. Howell Way of Waynesville, N. C., a member of the North Carolina State Board of Health, for the Committee on Resolutions submitted the following:

1. *Resolved,* That we appreciate most highly the splendid work performed by Dr. C. F. Williams, the efficient Secretary of the South Carolina State Board of Health, in organizing and successfully promoting this conference for the study of pellagra.

2. *Resolved,* That we tender our sincere thanks to Dr. J. W. Babcock, whom we justly recognize as the father of the movement for the study and control of pellagra in America, for his valuable labors and his many courtesies to this body during our session.

3. *Resolved,* That the purposes for which this conference was called can best be furthered by its formal organization into a permanent association of national scope, and that the Committee on Permanent Organization be instructed to report at this session.

4. *Resolved,* That this conference recognizes the widespread existence of pellagra in the United States and urges upon the National Government the necessity of bringing its powerful resources to bear upon the vital question of its cause, prevention and control.

5. *Resolved,* That while sound corn is in no way connected with pellagra, evidences of the relation between the use of spoiled corn and the prevalence of pellagra seem so apparent that we advise continued and systematic study of the subject, and, in the meantime,

we commend to corn growers the great importance of fully maturing corn upon the stalk before cutting the same.

6. *Resolved,* That the work of this conference be brought to the attention of the various State and Territorial Boards of Health and they severally be urged to specially investigate the disease, particularly as regards its prevalence, and that they also see that the proper inspection of corn products sold in the various States be had.

7. *Resolved,* That the Secretary be instructed to convey to the family of the late Cesare Lombroso our sympathy in their loss and our appreciation of his great work in the study of pellagra.

8. *Resolved,* That all papers and discussions of this conference be referred to the Committee on Publication with full power to publish the same as they deem fit.

Chairman Way moved the adoption of the report, seconded by Dr. I. M. Taylor of Morganton, N. C., who asked that it be adopted unanimously by a rising vote, which was done.

A paper entitled "Results of Stomach Analysis in Pellagra", by Dr. W. O. Nisbet of Charlotte, N. C., was read by title.

Dr. J. H. Randolph and Dr. R. N. Greene, State Hospital for Insane, Chattahoochee, Fla., read a joint paper entitled "Further Observations on Pellagra with Points on Prognosis."

Dr. Rea Parker, State Hospital, Williamsburg, Va., read a paper entitled "Clinical Observations of Four Cases of Pellagra."

Dr. J. M. Buchanan, State Hospital, Meridian, Miss., read a paper entitled "Report of Twelve Cases of Pellagra."

Dr. Sara A. Castle of Meridian, Miss., followed with remarks in which she detailed her observations regarding pellagra in Meridian.

Dr. J. Roddey Miller of Rock Hill, S. C., read a paper entitled "Report of a Case of Pellagra Universalis."

Dr. John Lunney of Darlington, S. C., reported "A Sporadic Case of Pellagra."

Dr. Theo. Maddox of Union, S. C., read a "Report of Six Cases of Pellagra", after which the conference adjourned until 8:30 p. m.

Second Day—Evening Session.

The conference re-assembled at 8:30 p. m., with Dr. Wilson in the chair.

Dr. H. H. Griffin, State Hospital, Columbia, S. C., read a paper entitled "Is Pellagra Contagious or Hereditary?"

This paper was discussed by Dr. John Forest of Charleston, S. C.

A paper entitled "Pellagra in Children", by Dr. M. B. Young, Rock Hill, S. C., was read by title.

Dr. Crown Torrence of Union, S. C., reported "A Case of Labor in a Pellagrin with Subsequent History of Mother and Child."

A paper by Dr. D. S. Pope, State Hospital, Columbia, S. C., entitled "Infants of Pellagrous Parents", was read by title.

Dr. A. B. Clarke of Plantersville, S. C., read a paper on "Diseases of the Eye in Pellagra."

Dr. Clarke was followed by Dr. E. M. Whaley of Columbia, S. C., who read a paper on "Eye Symptoms of Pellagra."

Dr. B. L. Eiker presented the Constitution which was prepared by the Committee on Permanent Organization. This Constitution in its amended form is as follows:

Preamble.

Recognizing the necessity that has arisen demanding the further study of pellagra in the United States, and in order to preserve and protect the health of our people from a scourge that threatens to become national, the National Association of Pellagra hereby adopts the following Constitution:

Article I.

This organization shall be known as the National Association for the Study of Pellagra.

Article II.

The membership of this association shall be composed of medical and scientific workers, who are interested in the prevention, study and control of pellagra. The charter members of this association shall be selected by the Board of Directors hereinafter provided for, from the list of attending physicians registered at the National Conference on Pellagra held at Columbia, S. C., November 3 and 4, 1909.

Article III.

The officers of this association shall be a President, a First Vice-President, a Secretary, Treasurer and Board of Directors.

Article IV.

The President, First Vice President, Secretary and Treasurer shall be elected annually and shall hold their offices for a period of one year, or until their successors are elected. The firs Vice-President, Secretary and Treasurer, together with four or more members, whose manner of selection shall be provided for in the By-Laws, shall constitute the Board of Directors.

Article V.

The meeting of this association shall be held annually at such time and place as may be agreed upon by a majority of the members present at the annual meeting preceding.

Article VI.

This association shall provide By-Laws, Rules and Regulations, not otherwise provided for, governing its deliberations, and the President shall have power to appoint committees to provide the By-Laws, Rules and Regulations above enumerated.

Article VII.

This Constitution may be amended at any annual meeting by a majority vote of all members present; provided that notice of such proposed amendment and vote thereon has first been publicly announced at a previous session of said meeting.

GEO. A. ZELLER, Peoria, Ill.,
LOUIS LEROY, Memphis, Tenn.,
E. M. MASON, Montgomery, Ala.,
E. L. EIKER, Leon, Iowa,
Committee.

Dr. Kerr, in speaking for the Committee on Statistics, said: I have spoken to the members of this committee and the membership has been made to include members of the State Boards of Health and particulary in those States where pellagra has attracted so much attention. It is the idea to continue and try to make a census of the number of cases of pellagra and, as was stated yesterday, this matter has been taken up by the Bureau, but we hope that through this committee and through the State Boards of Health we will be able to make a more complete report. As you are aware,

it is practically impossible to make an accurate census of cases of pellagra from year to year or to give an accurate estimate of the number of cases that have prevailed. It is more important, however, to secure enough statistics or as many as we can to determine the geographical distribution of the disease and for this purpose I see great necessity in trying to make a census and of collecting all statistics possible, and since every member and delegate of this conference is interested in this part of the subject, I would be very glad to have their co-operation, and if they have cases to report to us, we will be glad to include them in the compilation, but with one word of caution, namely, that we make them a minimum rather than a maximum number.

Dr. C. F. Williams reported, as Chairman of the Committee on Publication, that in compliance with the resolution offered by the Committee on Resolutions, the following committee had been appointed:

Dr. Hyde, Chicago Ill.; Dr. Woodward, Washington, D. C.; Dr. Byrd, Jacksonville, Fla.; Dr. Babcock, Columbia, S. C.

Our committee has met and outlined a plan for publishing the proceedings of the conference, and it is our hope to have the transactions ready for distribution by the middle of January, but as to whether or not this will be accomplished will depend largely upon the time it will take the stenographer to get out his notes, and the time that will be consumed in the revision of the discussions by the various participants, and the return of the corrected copy to us.

Dr. C. L. Minor called attention to an oversight in the election of officers, saying that no provision had been made for a Second Vice-President, and accordingly moved that Dr. C. F. Williams be elected as Second Vice-President, which motion was duly seconded and carried.

On motion the President was requested to appoint a Board of Directors to serve for one year.

Several lantern slides were then shown by Drs. J. J. Watson and C. C. Bass.

Doctor Wilson, on behalf of the State Board of Health, expressed thanks to the delegates for the hearty co-operation they had given, and which contributed so much in making the conference a success. He said he was much impressed with the earnestness and enthusiasm shown in the proceedings of the conference and he was sure that ultimately we would solve the mystery of pellagra which seems to impress us all so deeply He also wished to make acknowledgment

in behalf of the State Board of Health of the work which Dr. Babcock has done and is doing in furthering this cause. Dr. Babcock's enthusiasm had been an inspiration and the conference was largely due to his activity. (Applause.)

There being no further business, scientific or otherwise, to come before the meeting the conference then adjourned.

The Board was then called to order and the following resolution adopted and the Secretary instructed to forward a copy to Mr. Rockefeller:

Resolved, That the Executive Committee of the State Board of Health of South Carolina convey to Mr. Rockefeller their sincere appreciation of his generous public spirit as expressed by his munificent gift for the eradication of uncinariasis, which has exerted such a deleterious influence upon the physical and mental development of so large a portion of the population of the South.

There being no further business the Board adjourned at 11:30 p. m.

(Signed) C. F. WILLIAMS,
Secretary.

Attest:

INTRODUCTORY REMARKS

F. M. SANDWITH, M. D., F. R. C. P.

Gresham Professor of Physics, Vice-President of the Society of Tropical Medicine and Hygiene, Etc.

LONDON, ENG.

Although my professional engagements in London prevent my accepting the courteous invitation of the South Carolina State Board of Health to attend the National Conference on Pellagra, I gladly avail myself of their further request that I should write a short paper on the subject.

I feel that my first words must be those of congratulation and encouragement to the many physicians in the Southern States who are now working at the various problems connected with pellagra. Congratulation in the first place because they have discovered the existence of the disease, because they have impressd this discovery upon others so that the presence of pellagra is now thoroughly recognized by competent observers, and because they have provided us with a literature on the subject in the English language. This can not but be of interest to me, because, until I began to write on pellagra, there were apparently no literary contributions in our tongue, if we except two or three accounts written by travelers to describe cases they had seen in Italy. The English text books were either silent on the subject or frankly ignorant.

If I venture also to encourage American co-workers, it is not with any improper desire to draw attention to my own work, but because I happen to be in the unique position (with regard to this one disease) of being able to appreciate their difficulties.

So long ago as 1893, while preparing a paper on ankylostomiasis, I became aware that some of my peasant patients at Kasr-el-Ainy hospital in Egypt were suffering from dermatitis, bald tongue, diarrhoea, pains in the back, alteration of the knee jerk, insomnia and melancholia, all symptoms which could not legitimately be attributed to the anaemia caused by the hook worms. Quite ignorant at that time of pellagra, which was not known to exist outside Europe and Mexico, I determined to study the disease in Italy.

Landing at Venice, I was disappointed to find that the physicians of the general hospitals and lunatic asylums there were not interested in the disease, though at Milan and in its neighborhood I found many who were well acquainted with it, especially in the lunatic asylums and at the *pellagrosarii* or farm-sanatoria for the care of chronic patients.

I had no difficulty in identifiying the pellagra of lower Egypt with that of Italy, though the complications of the disease are obviously different in an Italian carnivorous alcoholic, and in an Egyptian, who habitually takes little meat and no alcohol and is likewise infested with entozoa (*ankylostomum duodenale* and *Schistosomum haematobuim*).

Upon my return to Egypt I succeeded in interesting a few of my English colleagues in pellagra, but I had to wait many years to induce the Egyptian government to make any inquiry into the prevalence of the disease, though my facts and figures had never been disputed.

Maize (zea mays) was introduced into Egypt as a cereal from Syria about 1840, yet pellagra was not discovered until 1893, though it had, perhaps, been present for years in the country districts.

In spite of denial from American authorities on medicine, I have always suspected that pellagra might exist, unrecognized, in the South, and at one time I requested my friends to put me into communication with the poorest folk of the maize-eating districts. I was referred to a settlement in eastern Virginia for pauper negroes, but, on investigation, I found that the inmates lived in stone houses on pork rations and I came to the conclusion that the word poverty represented no condition in America which could compare with the misery of the impoverished peasants of Italy, Roumania or Egypt.

During the South African war I found myself surrounded by poor colored folk living on maize, and I naturally expected to meet with some pellagra among them, but medical men practicing in the country assured me that no such disease had ever been seen. Yet, in the year 1900, I saw two cases of pellagra among the lunatics of Robben Island, Cape Town, and had previously recognized a third case at Bethlehem Hospital (London), which had been imported from South Africa.

This reawakened my suspicion with regard to the United States, and I was not surprised in 1902 to hear of the pellagrous farmer, reported by Dr. H. F. Harris of Georgia.

In April, 1905, I had the good fortune to be at Boston, Mass.,

during an epidemic of cerebro-spinal meningitis, and the very first patient whom I saw at the City Hospital, by the courtesy of Dr. C. F. Withington, was an Italian immigrant, who also displayed a well marked pellagrous eruption. This leads one to wonder whether the United States Public Health and Marine Hospital Service officers, who examine the emigrants at Naples and other Italian ports, should not include pellagra among the prohibitory diseases.

Now that the diagnosis of pellagra has been firmly established in so many States, it would be well to find out for certain how many people are attacked by the disease in the South. In order to arrive at any correct figures, it might be well to institute compulsory notification of the disease, at least as a temporary measure. In Italy there has been a law to that effect since 1888. The lunatic asylums will continue to yield a certain number of advanced cases, but pellagra should be searched for among the out-patients of general hospitals, and in the private practice of country doctors. I would also recommend that agricultural laborers should be examined in the States where pellagra is known to be prevalent, such as Georgia and North and South Carolina. This might be done in February or March, when the eruption is likely to be present.

In 1901-'02 I obtained permission to examine 500 Egyptian peasants who were actually at work in the fields; they all stoutly denied that they were ill, and their employers, who worked with them, stated that they could all do a fair day's work. Yet in every field I found early cases of pellagra, varying from 15 per cent. in well-to-do districts to 62 per cent. in the inhabitants of the poorest hamlets.

Though maize was the last of the great grain crops of the world to be brought within the domain of civilized agriculture, its production has now attained such magnitude that in some years it constitutes the greatest cereal crop of the world. As at least three-quarters of the world's maize crop is grown in the United States, the cultivation and curing of this cereal are of supreme importance to every American citizen. Already the question has excited the interest of investigators like Dr. Carl L. Alsberg.

If a complete census could be obtained of the pellagrous it might be found that the two sexes suffer equally, unless the women have a less varied diet than the men, and I shall be surprised to learn that the children (after the age of 10) are fairly exempt, as more than one American writer has stated during the last two years.

One of the objections to the diseased maize theory of causation

is that cases are sometimes reported of pellagra occurring among those who have never eaten maize. In examining more than 1,000 cases of pellagra, I have of course often met with individuals who stated that they were not maize eaters, but on cross-examination every one of them pleaded guilty to having occasionally eaten bread which was partly made from maize flour. I therefore venture to suggest that any *undoubted* case of pellagra should be thoroughly questioned before we inculpate a second cereal or attempt to overthrow the belief that diseased maize is a potent factor in the etiology.

Two cases of pellagra have recently been recorded in patients who had never been out of the British Isles, but the published accounts do not tally with the disease as I know it, though the symptoms were somewhat similar to pellagra. One of the patients had never eaten maize but had devoured raw oatmeal and rice.

A recent writer said that he thought maize might bear the same relation to pellagra as the swamp does to malaria. I would prefer to say that maize may be to pellagra as the mosquito is to malaria, remembering always that an uninfected Anopheles is unable to communicate malaria to any human being.

The diagnosis of pellagra is usually not difficult to anyone acquainted with the disease, but two groups of patients have often puzzled me; in the demented, unable to give any account of themselves, it is sometimes not easy to determine whether pellagra was or was not the foundation of their mental failure, though sometimes the reappearance of a rash at the advent of spring will help to decide the question. Another doubtful class of lunatics is affected with real or "pseudo-general paralysis," and in exceptional instances doubt may still prevail after the autopsy.

Anyone hesitating between the diagnosis of pellagra and leprosy must be profoundly ignorant of both diseases.

I notice that some, whose attention has lately been drawn to the study of pellagra, are puzzled by the use of the term "pseudo-pellagra," used by French writers. I have never employed this word myself; I have never heard it made use of, and I know of no circumstances under which it need be used. Roussel, a French physician, who wrote on pellagra between 1842 and 1866, called certain cases of "sporadic pellagra" in France, which only faintly resembled the endemic disease, "pseudo-pellagra."

Most of the cases of so-called "sporadic pellagra" clearly rested on errors of diagnosis during the years which followed Roussel's discovery that pellagra occurred in the centre of France.

Dejeanne, in 1871, subjected these French cases to a thorough scrutiny and wrote "these are maladies differing widely among themselves, and all of them very different from endemic pellagra not only in the etiology but also in the nature and concatenation of the symptoms." Surely, it is unnecessary to revive this antiquated discussion.

The treatment of early cases, without mental symptoms, can be successfully accomplished by putting the patient on a liberal diet, excluding maize and by ridding him of the hookworms which are so often co-existent, but the pellagrous symptoms return if he is allowed to resume a diet of musty maize. Various preparations of arsenic are useful in advanced cases, but when the brain is attacked there is small hope for the patient, unless by sero-therapy. Pellagra is essentially a disease which cries for preventive measures.

Italy, by preventive measures, has, since 1888, reduced the mortality of pellagra from 3483 to 1635, though during the years 1883 to 1907 the maize area under cultivation has increased from 5.79 to 6.33 per cent. of the whole country. This points the moral that it is the quality, not the quantity, of maize which is at fault. There are certain general axioms which prove true in Italy and Egypt, and it will doubtless be found that they hold good in the United States also:

1. In districts where no maize is cultivated or habitually eaten, pellagra does not exist.

2. There are many districts where maize has been cultivated for many years and yet pellagra has not appeared.

3. Well-to-do people in pellagra districts, living on varied diet and consuming maize as an occasional, and not as the staple cereal, usually escape pellagra.

4. It is not good maize or good maize flour which produce pellagra, the disease requires for its production the habitual use of damaged maize in some form.

There is a vast Italian literature dealing with the question of what the damage may be and there is now a considerable consensus of opinion in favor of incriminating *penicillum glaucum* in ordinary pellagra.

5. We are constantly being reminded by sceptics that the maize area of the world is infinitely greater than the pellagra area. This is not the point. The question is, does not pellagra distribution correspond very nearly with the areas upon which human beings live who eat damaged maize or products made from damaged maize?

May I be allowed to conclude these somewhat disjointed remarks

by emphasizing Dr. James H. Randolph's dictum: "There undoubtedly have been many cases overlooked in the past, but the greatest danger to be avoided in the future is not so much the failure to recognize the conditions, but rather a too great eagerness to diagnose as pellagra many related disorders.

We are now waiting, in the confident hope that some of the pellagra problems, so long unsolved, may be successfully mastered in the United States.

NOTES ON THE HEMATOLOGY OF PELLAGRA

J. W. KERR

Assistant Surgeon-General U. S. Public Health and Marine Hospital Service.

When the invitation of the State Board of Health of South Carolina to attend this Conference was received by the surgeon general, he was obliged to forego the pleasure of acceptance because of previous important engagements. Nevertheless, he is fully aware of the great importance of pellagra from the standpoint of the public health, and as he could not be present himself, designated me to represent him in the Conference. On account of the recent activities of the service in relation to pellagra, and because investigations of the disease already begun in the hygienic laboratory are to be greatly extended, it was his determination that the service should also be represented by a number of other officers, among them being Passed Assistant Surgeon Lavinder, who, as you are aware, has devoted much time and study to the disease—some of these studies having been made here in Columbia.

That pellagra is a serious disease there can be no doubt, and it is evident from the studies already made that its occurrence in the United States is a matter of grave concern. Even if there were no evidence that the disease is on the increase, the experience of countries where it is endemic should be sufficient to stimulate active interest and warn against the dangers of its continued occurrence.

In certain parts of Europe, where pellagra has long prevailed, it remains a menace to the physical and mental integrity of large numbers of the population, and who can estimate the economic losses it has occasioned through sickness and death and interruption to commercial activities? That they are enormous is evidenced by the prolonged and careful attention given to it by many of the ablest minds of those countries, and that it is a national problem of the first rank is shown by the governmental efforts in those countries looking to its control.

The problem of pellagra in Europe and the New World is in some respects analagous to beri beri in the Orient. Both diseases are held to be associated with the consumption of important articles of diet; both are capable of becoming veritable scourges among the people of the countries where they become endemic, and in neither disease has the etiology been definitely determined. This analogy

might be extended to include communicability, and in some respects symptomatology, and treatment of the disease. In neither disease is there evidence that communicability plays a part in their continued prevalence, and in neither has it been deemed advisable or necessary by the National Government to institute quarantine procedures.

The Public Health Service in the exercise of its quarantine functions is ever alive to the importance of exotic diseases, and has accordingly taken cognizance of reported cases in this country and abroad. Through its international quarantine relations the Service is in a position to have its officers familiarize themselves with the public health importance of such diseases, and make reports thereon, as has been done in the cases of beri beri and pellagra, reports to the effect that Italian pellagrins have been and are being rejected as immigrants by the officers now on duty in Italy.

On account of such reports and in view of the long experience of Europe with pellagra and of the Orient with beri beri, it must be considered that those diseases are not directly communicable, and that where they prevail there also must their causes be sought. In other words, pellagra has been discovered in our country, it has in all probability prevailed for a considerable period, its cause or causes are to be found among us, and the occurrence of the disease involves problems that will require the most earnest investigation with the view to their solution.

When the first cases of pellagra were reported from Alabama in 1907 an officer of the Service, who had had extensive service in Naples, Italy—one of the endemic centres of the disease—invited attention to the probable increasing importance of the subject in relation to the public health, and recommended that investigations be undertaken. Thereupon, the surgeon general requested an officer of the Service, stationed in Mobile, to report upon the disease with particular reference to its prevalence in Alabama, which he did July 20, 1907.

Subsequently reports of the occurrence of pellagra in other Southern States began to appear, and on April 27, 1908, Passed Assistant Surgeon C. H. Lavinder, who was then on duty at the Marine Hospital, Wilmington, N. C., reported that several cases of the disease had been observed in that city, and that there had been admitted to the Marine Hospital a case which subsequently developed characteristic symptoms. In view of this report, the surgeon general invited the attention of Dr. Lavinder to the increasing prevalence of

the disease and to the possibility that it might soon assume importance both from public health and economic standpoints. Dr. Lavinder was, therefore, instructed to prepare a precis on the subject, and as a result a manuscript was submitted July 8, 1908, which was immediately published and widely distributed. It was expected when this article was issued that it would be the beginning of a very thorough investigation of the disease, and the progress of events in relation to pellagra have demonstrated the wisdom of this determination.

In his annual report for 1908, the surgeon general stated that pellagra should receive unremitting study, and he decided to detail an officer of the service who should devote his entire time to investigations of the disease. It was with this object in view that Dr. Lavinder undertook preliminary studies in the hygienic laboratory and later came to this city to avail himself of the excellent clinical advantages offered.

On March 26, 1909, an outline of the proposed investigations were submitted to the advistory board of the hygienic laboratory, which board is composed of four officers of the Government and five other members eminent in their respective scientific fields and connected with corporate laboratories of like character. It was the unanimous opinion of the board that the primary investigations should be made in South Carolina and other places in this country in order that Dr. Lavinder might be better prepared to make comparable studies of the disease abroad.

On account of the clinical facilities available a working laboratory was established at the State Hospital for the Insane in Columbia, and the necessary material in that institution was most courteously placed at the disposal of Dr. Lavinder by Dr. J. W. Babcock, superintendent of the asylum, who evinced every desire to lend aid during the investigations. Indeed, the State of South Carolina has displayed great interest in the pellagra problem from the beginning, and through Dr. C. F. Williams, its State health officer, has done much to stimulate interest in the grave situation which appears to be developing not only in the South, but in other sections of the country.

Early in July, 1909, reports were received from Nashville, Tenn., and Chicago, Ill., stating that pellagra was thought to exist there, and on request Dr. Lavinder was ordered to Nashville, where he found 15 cases and to Dunning, Ill., where were diagnosed three cases. Up to this time the disease had been reported only from the

Southern States. It was a matter of much interest, therefore, to find the disease existent also in the North Central States.

In August, 1909, the disease was reported from the General Hos- Health and Marine Hospital Service, which was held in Washington, June 2, 1909. Dr. Williams of South Carolina, who was invited to introduce the subject, presented a statistical paper by Lavinder, Williams and Babcock, which was published by the Bureau and contains records of approximately 1,200 cases scattered over 13 States. During the discussion Dr. H. F. Harris, secretary of the State Board of Health of Georgia, referred to a case of pellagra reported by himself in 1902, and stated that within the past two or three years the disease had increased in a remarkable manner.

Since the Conference reports have been received of the occurrence of pellagra in a number of other States, and conservative estimates of the total number of cases have increased from 1,200 to over 5,000. It is not to be inferred from this, however, that the increasing number of cases is due wholly to an increasing prevalence of the disease. It is far more likely that it is largely a matter of discovering existing cases as knowledge of the disease spreads and skill in diagnosis develops among a profession hitherto largely unfamiliar with the subject.

It is of great importance to determine the geographical distribution of the disease, and above all, its prevalence in this country. The bureau has therefore prepared and distributed blanks to health officers throughout the country on which to make regular reports of cases of pellagra. Copies of these blanks are presented for your information and it is earnestly requested that the influence of this Conference be exerted to secure co-operation in the collection of the statistical data so much desired.

While the collection of statistics is of primary importance, it is only one phase of the pellagra problem. Arrangements were therefore made for co-operation on the part of the Public Health Service and Bureau of Plant Industry in the study of the topics in which each was especially interested. In addition the surgeon general determined to concentrate the energies of a number of scientific workers on certain phases of the problem with the view to their elucidation. He has accordingly appointed a pellagra commission, whose membership consists of Passed Assistant Surgeon John F. Anderson, director of the hygienic laboratory; Dr. Reid Hunt, chief of the division of pharmacology of the hygienic laboratory; Surgeon M. J. Rosenau, who is now professor of preventive

medicine and hygiene in Harvard University; Passed Assistant Surgeons C. H. Lavinder and John D. Long of the hygienic laboratory; Dr. William A. White, superintendent of the Government Hospital for the Insane, Washington, D. C., and Dr. Nicolas Achuccaro of the Government Hospital for the Insane, Washington, D. C.

The investigations of pellagra already begun will be greatly extended so as to include epidemiological, pathological, clinical and pharmacological studies with the hope of throwing additional light on the problem as presented in America today.

Pellagra has only recently been recognized in the United States, and the medical profession as a whole is not at all familiar with its manifestations. In order that the 400 or more medical officers of the service might speedily become so, the surgeon general on September 10, 1909, issued a circular letter inviting their attention to the subject and calling for special reports from time to time of cases of the disease coming under observation. In addition, 23,000 publications relating to pellagra have been published for distribution by the bureau as a means of further disseminating information among health authorities and the medical profession generally.

From the foregoing outline of the steps already taken, it is seen that the surgeon general is deeply interested in pellagra and that he regards it as a public health problem of national importance. This does not mean that there is cause for panic, and he is convinced that the present popular interest in the disease will resolve itself into reasonable prophylaxis and improved sanitation.

The belief that there is some relation between pellagra and the use of corn as food would seem too universal and too profound to permit of rejection except in the case of demonstrative proof to the contrary. The exact nature of this relation awaits final solution. In the meantime it appears from all evidence that sound corn is a highly nutritious and valuable food, and to counsel its total rejection would be inadvisable, except for purposes of investigation.

Finally, it must be stated that, while the task before this Conference is one of unusual difficulty, it is worthy of the most profound deliberation, and I am delegated by the surgeon general to assure you of his deep interest in your work. He realizes that it is through Conferences such as this that those in attendance will receive inspiration, and the people as a whole be given wise counsel with respect to prophylaxis and improved habits of living.

ECONOMIC FACTORS OF THE PELLAGRA PROBLEM IN SOUTH CAROLINA

E. J. WATSON

Commissioner Department of Agriculture, Commerce and Industries

COLUMBIA, S. C.

Mr. Chairman and Gentlemen of the Conference:

When I stand here before such a body of distinguished scientists and professional men from all parts of this nation and from other nations, I feel there must be a good reason why I was asked to do so. I am one of the official guardians of the welfare of the people and the agricultural and industrial future of the Southern States—the representative of the government of this commonwealth in the care of these varied economic interests. You are here to counsel as to a grave danger that threatens not alone these economic interests in this State but in the whole nation, and it is timely that we daily toilers for the uplift of the people and the fullest development of natural resources, we practical protectors of sources of possible disaster, should meet with you and counsel with you, and then in our turn take the results of your investigations and urge our lawmakers to place the power of protection of the unwarned people of the commonwealth in our hands. This is the reason I am before you to talk to you of "The Economic Factors of the Pellagra Problem." At least I so take it. It is a strong reason. "Strong reasons make strong actions," and I hope that this conference will make reasons so strong that this State and all the other States shall not delay in taking strong actions.

"The sweat of industry would dry, and die,
But for the end it works to."

In this problem of the hour the end is the same that my official oath binds me to work to—the fullest economic development of my State and the ultimate happiness and prosperity of her people.

"Ignorance is the curse of God," it has been said, and it is the mission of you distinguished scientists to remove that curse. I am here to pledge you that when you place the instruments of removal at our disposal and point the way you will not find wanting willing workers in my sphere of endeavor.

I am not here to attempt to discuss the etiology of pellagra, to attempt to say whether pellagra has its origin in impure Indian corn—the original wild grass of the Mayas of Central America, brought to us centuries after our country was discovered, via Mexico by the Toltecs and the Aztecs, given to us by the Indians and sent by returning explorers to Spain, Italy and other countries. I know that within the heart of the corn kernel, carefully encased in a cylinder of oil, are life-living cells. The vital principle men of my class do not understand, but it is there within the womb of the kernel, ready when healthy to burst forth into a beautiful, sturdy plant and bring forth an ear of corn "within whose yellow heart there is health and strength for all the nations." We do *not* KNOW that in this kernel lurks also grim Death. There are reasons to believe that this is the case. We do know that "'Tis safer to avoid what's grown than question how it was born." We know that pellagra exists in this State and is causing deaths among our people.

"Judgment and reason have been grand jurymen since before Noah was a sailor," and this grand jury has upon the testimony of many scientific witnesses rendered a true bill against King Corn. You are here assembled today to try the case as petit jurors and to render the verdict. If it be "guilty" then there is all the more reason for me to be here. When a serious charge—in this case the charge of murder—is written on the indictment, the subject of the charge must be held under grave suspicion, and such proper precautions as are possible to prevent further crime should be taken. Surely "'Tis safer to avoid what's grown than question how it was born."

Our chief in our sphere of endeavor, that distinguished official, Secretary Wilson, who has done so much for the people of this commonwealth, tells me. "I recognize fully the necessity of giving every attention to the pellagra disease," and "am naturally interested in the question of the relation of Indian corn to the disease."

Corn stands indicted! When such danger threatens I for one believe in hoisting the red flag and taking such precautions as our limited knowledge will permit. The corn crop of this country is the principal crop of the country. Last year it was worth nearly as much as the great crops of cotton, hay and wheat combined. It amounts to over two and a half billions of bushels, out of the three and a quarter billions in the whole world, and represents wealth—"wealth," as Mr. Wilson says, "taken out of the soil in four months"—of over a billion and a half dollars, enough "to cancel the interest

bearing debt of the United States and to pay for the Panama Canal and fifty battleships." There has been an increase in value of $600,000,000 since 1902, a sum "equal to the gold in the treasury of a rich nation." In this little State the corn crop last year had jumped to nearly 30,000,000 bushels, worth over 26 1-2 millions of dollars, a phenomenal increase in two years of practically ten millions of dollars. In 1908 the nine cotton-growing States—the Carolinas, Alabama, Georgia, Mississippi, Louisiana, Texas, Oklahoma and Arkansas, produced 561,103,000 bushels of corn—forty per cent. more than Pennsylvania, Michigan, Wisconsin, Minnesota, Kansas, North Dakota, South Dakota, Colorado and New Mexico, which produced 158,475,000 bushels. In the South corn-growing is just beginning to reach for its flood tide, and we are hourly bringing every influence possible to bear to reach the goal.

Corn is grown on 80 per cent. of the farms of the nation and on one-eighteenth of the agricultural acreage, the centre of production being in Illinois. For the past thirty years the North Central group of States has furnished about three-fourths of the total crop of the nation. Some one has figured it out that if the ears of the country's corn crop were placed end to end they would encircle the earth at the equator over 1,100 times; put in cars of 1,000 bushels each the crop would make a train extending around the world, and if the trains were composed of 50 cars each it would require 50,000 locomotives to haul them to market.

No wonder we guardians of the economics of the country are awake and up and doing, and already hard at work investigating sources of supply. No wonder we are hunting out evidence-ex parte, if you will, which even gives a reasonable presumption to connect deaths of man and beast with the utilization of impure corn and corn products. No wonder, while we regard Mr. Rockefeller's princely gift for the eradication of the hookworm at its true value, we say one million for the battle against the disease of pellagra would be far more valuable. In the one case the nature of the disease and its remedy is known; in the other the remedy is unknown. No wonder we are searching for evidence to show whether death-dealing qualities charged against King Corn are developed in the home-raised goods or in the product brought in from other States.

Notwithstanding this State is now raising practically thirty million bushels of corn the people of South Carolina are spending outside of the State for corn the sum of $6,000,000, and this does not include the expenditures—which are practically all outside the State

—for grits, meal, corn-flakes, cornstarch, yeast, adulteration in flour, and—well, I might as well include it too—corn whiskey! How great a sum these products amount to I dare not estimate. And South Carolina has no inspection law!

South Carolina buys these products principally through the markets at Nashville, St. Louis, Cincinnati and Richmond, according to the state of the market, and they are sold through commission men. Practically no Texas corn reaches this State, and incidentally it may be said, Texas corn seldom if ever spoils even on export, as the climate is such as to thoroughly cure it. This is also largely true of the home-raised product. Through the markets named the corn and corn products reaching the South Atlantic Seaboard comes from the corn belt States and the States further West. The principal shipping States, the figures showing in even numbers the portion of the annual crops shipped out of the County where grown, are Illinois, 131,363,000 bushels; Iowa, 77,600,000; Nebraska, 74,000,000; Kansas, 34,364,000; Ohio, 32,802,000; South Dakota, 19,000,000; Indiana, 41,350,000; Missouri, 20,363,000; Tennessee, 11,875,000, and so on. All know what the weather conditions did to the crop in the third named of these States this summer, but I am not arraigning any one State or set of States. I would merely show whence comes our supplies bought out of the State.

I said we had already begun to searchingly investigate cases in various portions of the State with a view to tracing, if possible, the sources of corn and corn products supply fed to man and beast, and getting such preliminary data as possible, for we have thousands of our population who never fail to have their corn bread and hominy—and this is not confined alone to the laboring classes, to town or farm, but to all classes. We also have thousands of head of stock hourly endangered if the indictment against King Corn be sustained. Indeed, the entire economic outlook is placed in jeopardy.

Let us see what we have found. A physician in the Piedmont section tells me, "I have treated in all ten cases of pellagra. Six of these are dead. I have seen a number of other cases in consultation with other physicians. While I believe most of these cases were caused by eating meal from damaged corn, still I could not prove that any of them came from any certain supply, for the reason that there is no inspection and any and all kinds of meal is sold without restraint. We need a corn and meal inspection law worse than anything I know of."

Another physician in the Piedmont, who has observed 75 cases,

states that some of them he could not trace to impure corn supply. He tells me of a white patient "Traceable directly to spotted corn from a crop which was grown on lowland and overflowed by a swollen creek before harvesting. One of his patients, a white woman, told him she had eaten of corn contrary to directions and was immediately made worse."

Another close medical student of the disease says: "Those cases under my observation have been consumers of grits and chiefly bread made from corn meal. These corn products, as a rule, have been shipped here from the West. I can not say that my cases can be traced to the effects of corn, but can say that the uses of meal or grits by those having pellagra produced an exacerbation of some of the symptoms. There has been no consistent effort made to prove the meal or grits mouldy or spoilt."

Another close student says: "I have observed twenty-four cases of pellagra in this vicinity since the spring of 1903. They have given a history of eating store-bought or Western meal. But I have not traced any of these cases to any special supply. Twenty of these cases were whites, some of them in good circumstances, and four were negroes. Thirteen were white females, one negro female, seven white males, and three negro males. Of the nine cases I have observed in death, all show mental derangement before death. Nearly all of the meals used here in town is Western or shipped meal. On inquiry among the commission men here I find they buy most of their meal from Nashville or Richmond. A lot of the corn ground in Nashville, they say, comes from further West."

A leading physician in upper Carolina writes me this: "Our people are not yet restricted to any single article of food. However, I will state that all cases coming under my observation have been eaters of corn meal and grits. Two of them have been great lovers of corn bread, and practically the only corn bread eaters in their families. And the only ones so far having developed any symptoms whatever of pellagra. All of my cases have occurred in separate families. I notice in public print that there are two cases who never ate corn. This in my judgment is unfortunate, and misleading, since it has been only a few years when our wheaten flour was largely adulterated with heart corn. Lombroso and a few other scientists have proven positively that the disease is produced by the injection of damaged corn. At present we have no authentic information that it has been caused by anything else, nor is it hereditary. What the future will reveal I know not but am anxiously waiting."

Another physician presents another phase of the inquiry, when he says: "I have observed for years that stock fed on corn over which the creek had run would have 'blind staggers,' and believe this is only a symptom of pellagra. I have also noticed dogs burying bread in the earth, which would soon mould and I believe this is a cause of the so-called hydrophobia and is nothing more than pellagra. From these observations I am convinced that spoiled corn plays a potent source of this trouble."

And just here I may say that I have had reports of the killing of a considerable number of hogs fed on corn purchased by their owner, known to be spoiled. He fed it against advice given by his neighbors and the death of the animals ensued. Another case reported from the coast: The owner of a number of horses bought some spoiled Western corn. A ration was fed a few and they died. Orders were given to feed it no longer. A new stableman who had not heard the order came along some weeks later, fed the corn and $3,600 worth of horses were sacrificed.

The cases of the children in a middle county who ate warm meal from their father's grist mill when it was grinding bad "Western corn," ending in their death from pellagra, while others of the family eating only products of pure home-raised corn will be explained perhaps by others at this conference. It seems conclusive.

We have found cases where carloads of bad Western corn had been placed on the floors of warehouses, leased for the purpose, by commission men before being offered to customers.

Instances have been reported of corn being ground into so-called meal and shipped in after it had been distilled, but specific cases we have been unable to find.

I am told that much impure corn is distilled into corn whiskey—in fact, that this is a common use for corn that cannot be used otherwise. Apropos of this, one physician writes me, "One of my cases (pellagra) was a hard drinker; never ate corn, of course drank corn whiskey. Whether corn in this case was the cause is questionable."

But why enumerate? Surely "unquiet meals make ill digestion," and ex parte statements implicate King Corn in the ruining of the digestion of the people of the commonwealth sufficiently to make us take notice.

What has really awakened us to activity, however, is the case of a carload of corn, and we are in possession of facts and names. This car of corn was shipped in from the West last June. A whole-

sale merchant went up to look at it and found it in a damaged condition. He offered a price, but the agent who was trying to sell it refused to accept the price, stating "that he could get a better price by selling it to a mill, and it was returned to Nashville, Tenn." Whether this corn came back to this State in the shape of meal, was sent to some other State, sold locally in the form of meal, or was sold to distillers, can only be a matter of conjecture. It is certain, however, that this damaged product got into the stomachs of human beings in one form or another. The same merchant says that the corn shipped in this year has been in much better condition than heretofore, and that two years ago it was exceedingly bad.

A commission merchant of ten years' continuous operation in one of the large cities, referring to adulterated food, impure grain, tells me: "I do not believe there has ever been a time since I have been in the grain business when so very much swindling has been going on as at present," and tells me also of short weights in meal.

But, perhaps, I have bored you with this hastily drawn picture of conditions leading us to take cognizance of the indictment and display vital interest in your conference.

The Federal government, under the new Pure Food Law, doubtless finds itself unable at this time to afford adequate protection. In the meantime, if it is true that bad corn and bad corn products produce pellagra—or even if it isn't true—and we must go on the doctrine of an ounce of prevention, then the hour has arrived for the States to act speedily and decisively and at least provide measures of protection against the making of such States as South Carolina the dumping ground for the damaged and dangerous products of the country. We have some laws on our statute books, but they do not carry with them means or machinery for their rigid enforcement. Enough signs of danger—sheet lightning they may be—have appeared to make decisive action imperative if the agricultural interests and even the lives of the people are to be of any regard, and I am no alarmist.

There are in this country today only thirty States with any kinds of laws regulating the sale and inspection of concentrated feed stuffs. Fourteen of these are Eastern States, including Ohio, Indiana, Illinois, Wisconsin and Michigan. Ten are Southern States, and only six are Western States, even classing Oklahoma in the latter. The other five are North and South Dakota, Washington, Iowa and Kansas. Missouri and Nebraska are among the States that have no laws unless recently passed. Meal is exempted from

the provisions of the laws in nearly every State in which we are concerned.

This agitation of the whole question as to whether or not corn is the producing agency of the disease of pellagra—indeed the mere discovery of the disease—should admonish our people to do what we have been pleading with them for other economic reasons in season and out of season to do for their own protection, namely, raise their own home supplies. In this climate corn matures and is liable to be pure, and our people owe it to themselves and their posterity to do their part. Today there is a mere handful of grist mills in operation within the State and their business is purely local. The first step, then, from an economic standpoint, is for the people of the affected States to raise and manufacture, for human food purposes, their own corn, and the second is for them to put in full force a complete inspection system, with means and men to execute the laws when once enacted.

I fear I have gone a little more exhaustively in this matter than your indulgence would warrant. My only excuse is that I realize the vital importance of this conference, not alone to the men, women and children of the present generation, but to posterity in this nation, in other nations, and in this proud, prosperous and prospering little commonwealth. I trust that your deliberations will lead to results that will themselves, in time, lead to the happiness of the homes of our common country, and to the prosperity and above all to the preservation of the health of the occupants of those homes.

NOTES ON THE HEMATOLOGY OF PELLAGRA.

C. H. LAVINDER

Past Assistant Surgeon, U. S. Public Health and Marine Hospital Service

WASHINGTON, D. C.

A rather cursory review of the literature of a subject so well worked as pellagra impresses one with the idea that the study of the blood in this disease has been somewhat neglected. Carletti,[1] in a contribution on the hematology of pellagra in 1903, said that up to that time hematological researches had all been of a partial nature and devoted to some particular phases of the question; and that there did not exist in all literature a complete study of the subject. So far as I can learn from a somewhat hurried review of the literature since then, this statement is practically true today. Contributions are constantly being made on various points of the subject, but complete studies appear to be wanting.

This seems to me an interesting and not unprofitable field for American investigators. My work on this subject has been limited and the results as yet have not been satisfactorily collated, but a brief review of some points may prove interesting, if not stimulating, to those interested in pellagra.

Blood counts, hemaglobin estimation and differential leucocyte counts.—Carletti,[1] summing up the literature of cell counts and hemaglobin estimation, reviews briefly the work of Lombroso, Capezzuoli, Sepilli, Agostini and D'Ancona and Randi, all of whom obtained somewhat similar results, i. e., a fairly constant, though not excessive reduction both in the number of red cells and in the hemaglobin. Only one of these workers reported on white cell counts (Sepilli) and he found the number of leucocytes normal.

With regard to qualitative changes in the cells Carletti states that many observers have made studies on this point but did not seem to find sufficient changes to interest them. He quotes, however, Agostini's results, which were pallor of red cells, slight tendency to rouleaux formation, abundant small cells (microcytes?), poikilocytosis, large leucocytes with large granules (?), and abundant pigment (?).

Carletti, in summing up his own work, states that he found a constant mild reduction in the number of red cells, much variability in the number of white cells, but never a leucocytosis; constant

reduction in hemoglobin (65 to 75 per cent.) with a low color index (0.75 to 0.80). As for qualitative changes in the cells he found constant, but not grave, modifications with a rather large number of small red cells (microcytes?); the varieties of leucocytes he found retained their normal proportions, with a possible increase in the large mononuclears; eosinophiles he saw very rarely.

Among more recent workers, Galesesco and Slatinéano,[2] from an examination of 31 cases, report a constant diminution in the number of red cells (3 to 4 millions) without qualitative changes; hemaglobin (von Fleischl) from 70 to 90 per cent.; slight increase in the number of leucocytes (9 to 10 thousand), differential counts, polynuclears from 55 to 78 per cent., lymphocytes 17 to 33 per cent., large mononuclears 10 to 22 per cent., eosinophiles 2 to 4 per cent. They conclude that there is a constant, large mononuclear increase from which no deduction can be drawn as to etiology.

Fratini[3] made a study of 34 cases, both acute and chronic forms. He used the Thoma-Zeiss counting apparatus and the von Fleischl hemaglobinometer, studied fresh specimens and specimens stained with Ehrlich's triacid stain after fixation in bichloride of mercury; and made differential counts. His results were: A rather constant reduction in the number of red cells (3 to 4½ million) and in the hemaglobin (55 to 92 per cent.); white cell counts ranging from 7,412 to 11,418; differential leucocyte counts somewhat variable, but a rather constant slight increase in eosinophiles (presumably, of course, eosinophilic leucocytes), which he attemps to explain as due to the alterations of the intestinal epithelium so common in pellagra. His figures showed polynuclears, 55 to 76 per cent.; large lymphocytes, 2 to 5 per cent.; small lymphocytes, 18 to 39 per cent.; eosinophiles, 2 to 9 per cent. As for qualitative changes in red cells he reports rather constantly macrocytes, microcytes, and poikilocytes. He also states that he found nothing resembling a protozoal parasite.

Manson[4] states that Sambon and Terni, in Italy, and Grigorescu and Galasescu, in Roumania, have noticed a relative increase of the large mononuclear leucocytes.

Fiorini and Gavini,[5] in a study of nonalienated pellagrins, report an absence of leucocytosis, but a typical mononuclear increase and a decided eosinophilia.

Masini,[6] in a study of the eosinophile cell in the blood of pellagrins, concludes that, contrary to what is found in many acute infectious diseases, there is produced in the pellagrous intoxication a

conspicuous and decided eosinophilia, which occurs in cycles corresponding with the increase or diminution of the pellagrogenous toxines; that is, the more toxemia the greater the eosinophilia. He suggests that this constant eosinophilia may prove at times a valuable aid in early or differential diagnosis.

Peserico,[6] making differential counts in several cases, obtained the following results: Polynuclears, 53.7 to 67.4 per cent.; lymphocytes, 26.1 to 37.4 per cent.; large mononuclears and transitionals, 1.2 to 7.7 per cent. He speaks of the nongranular, large mononuclear as the *bete noir* of hematology and expresses much doubt regarding this cell.

These results in many ways seem decidedly discordant, but one may at least conclude that there is a very frequent, usually mild, anemia of the secondary type, and that if there are qualitative changes in the red cells, they are only such as one would expect. The differential leucocyte counts seem almost too discordant to reconcile in any way, but there would seem a majority opinion of a definite, relative, large mononuclear increase.

While working on pellagra at the State Hospital for the Insane here in Columbia, S. C., during the past summer, I made blood counts on 24 cases. The results are given in the table below:

Number.	Male or Female.	White or Negro.	Age.	Per Cent. Hemaglobin (Dare)	Number White Cells.	Number Red Cells.	Mental Involvement.	Arsenic.	Remarks.
1	male	white	60	75	4,000	3,424,000	no	no	Chronic, mild, neurasthenic.
2	male	white	60	75	8,600	4,684,000	no	yes	Chronic, mild, neurasthenic.
3	male	white	60	64	6,222	4,272,000	no	yes	Chronic, mild, neurasthenic.
4	female	colored	52	65	4,508	4,842,000	yes	no	Chronic, mild.
5	female	colored	52	83	10,115	4,824,000	yes	yes	Chronic, mild.
6	female	colored	25	45	6,755	4,368,000	yes	yes	Emaciated, moist erythema, spasticity.
7	female	colored	30	45	4,800	4,888,000	yes	yes	Emaciating, dry erythema; paralytic.*
8	female	colored	25	45	8,266	2,826,640	yes	yes	Emaciating, moist erythema; improved.
9	female	colored	35	38	9,633	2,920,000	yes	yes	Fairly well nourished, dry erythema.
10	female	colored	25	64	13,000	4,148,800	yes	yes	Emaciating; moist erythema; increased reflexes.
11	male	white	39	88	7,066	5,520,000	no	no	Dry erythema; neurasthenic; alcoholic.
12	female	colored	25	38	6,044	3,400,000	yes	yes	Slight emaciation; dry erythema; syphilis; round worms.
13	male	colored	33	95	8,200	5,500,000	yes	yes	Well nourished; much excited; dry erythema.
14	male	white	55	57	11,980	3,800,000	yes	yes	Arterio-sclerosis; dry erythema.
15	female	colored	16	88	13,066	4,636,000	yes	no	Generalized, moist erythema; emaciating paretic.
16	female	white	31	59	10,844	5,604,800	yes	no	Well nourished; dry erythema; paretic.
17	female	colored	22	74	9,754	3,864,000	yes	no	Well nourished; dry erythema; hookworms.
18	female	colored	44	70	17,310	4,708,000	yes	no	Dry erythema; arterio-sclerosis; much excited.
19	female	colored	37	76	5,544	5,008,000	yes	no	Pellagroid; diagnosis doubtful.
20	female	colored	33	96	8,880	4,620,000	yes	no	Cured case; no symptoms for over a year.
21	female	colored	50	93	7,344	4,440,000	yes	no	Cured case; no symptoms for over a year.
22	female	white	25	80	17,400	4,976,000	yes	no	Suicidal and excited; dry erythema; losing weight.
23	female	colored	38	86	12,010	5,504,000	yes	no	Dry erythema; slight loss of weight; mildly excited.
24	female	white	22	60	5,800	4,580,000	no	no	Chronic; mild.

*The word paralytic in this table is used in the sense adopted by writers on pellagra, that is paretic rather than actually paralytic.

The technique of making these counts was as follows: The Thoma-Zeiss counting chamber, with Turck's ruling, was used; in counting red cells the four-corner unit squares (25 small squares each) were counted in two preparations and if the results were discordant, a third was counted; in counting white cells the whole ruled space was counted in two preparations, and again if results were discordant, a third preparation was counted. The hemaglobin estimation was made with a new Dare instrument, which gave very uniform readings in normal individuals.

The cases were mostly of an advanced type with many secondary nervous changes and nearly all were alienated. Most of them were negro women. The greatest number of the counts were made at the same time of day (morning) and under the same circumstances as far as possible. Many of the patients had been taking arsenic in some form with full dosage, either at the time of the examination or not a very long while previously.

A brief analysis of this table shows a frequent reduction in red cells and in hemaglobin, and the color loss would seem proportionately greater than the cell loss (chlorotic type of anemia). There is a great variation in the number of leucocytes with an occasional decided leucocytosis, not clinically explicable by complications or otherwise. It is interesting to note that numbers 1, 2 and 3 in the table are the same individual, before and after arsenical treatment; and likewise 4 and 5 are made in the same individual; 19, 20 and 21 are also of interest as doubtful or cured cases.

Differential leucocyte counts were made in several of these cases. The results generally showed a relative, large mononuclear increase with an absence of eosinophilia, except in cases with such complications as round worms or hookworms. I do not tabulate these counts, as I do not find the work satisfactory and distrust the results. As will be seen from the literature reviewed above, discordant results have been obtained by other workers in making differential, leucocyte counts.

The technique adopted by me was briefly: Smears made on slides, by using the edge of another slide, stained by one of the numerous modifications of the Romanowsky stain, usually Wright's; and in making the counts the fringes of the smear were avoided as far as possible. The total number of cells counted was usually 500 or more. A mechanical stage was used.

The classification was the usual one of Ehrlich: (1) Polymorphonuclear neutrophiles, (2) small mononuclears or any nongranu-

lar cells smaller than a polynuclear, (3) large mononuclears or any nongranular cell larger than a polynuclear, with a round or oval nucleus, (4) transitionals, any cell within the same limits as a large mononuclear but with an indented nucleus, (5) eosinophiles, (6) mast cells (Emerson).[8]

A brief review of the literature on this point and my own experience leads me to question the results achieved and I gravely doubt whether, from the work done, we can make any definite statements as to just what proportions the various forms of leucocytes in pellagra may bear to each other. The only one general conclusion which occurs in the work of several is a relative, large mononuclear increase. This I found with my technique above described. It seems to me that unless each worker states in full the details of the technique employed, the interpretation and correlation of results will give rise to much confusion. The especial points I would emphasize in the technique are the manner of making smears, the question of counting fringes, and the classification of the various cells. If smears are made on slides in the manner done by me and fringes are counted the results will be very different from counts made from the centers of smears. Perhaps cover glass smears, made in the usual way, might avoid this difficulty. No matter what classification of cells is used the personal equation of the counter must also have its effect, for there are many doubtful cells.

With regard to qualitative changes in the red cells there are undoubtedly, as one would naturally expect, such changes as are found in any secondary anemia, these changes being in great respect, of course, dependent upon the grade of the anemia. In a study of fresh smears, stained smears and smears made by "vital staining" I found only such changes as these in my work.

I made no accurate observations on coagulation time, but in drawing blood for various purposes from a number of pellagrins I have never observed any failure of fairly prompt and thorough clotting.

Bacteriology of the blood.—The bacteriology of the blood in pellagra forms a rather interesting chapter in the history of this disease. Bacteria, thought to be specific in their nature, have several times been described in the blood of pellagrins. Majocchi, Cuboni, Paltauf, Heider[9] and others did much work on the so-called *Bacterium maydis;* Carrarioli[4] reported the *Bacillus pellagrae.* P. Marie[10] states that he observed in two cases rather constantly a micrococcus, which, however, he could not grow.

Quite recently Tizzoni[11] has reported a specific bacterium which

he calls *Streptobacillus pellagrae* and which he finds constantly in the blood of pellagrins, as well as in the stools, the organs after death, and in spoiled corn.

Tizzoni[11 & 12] first obtained this organism by simply incubating at 37 degrees C. blood and spinal fluid drawn from a recent cadaver, dead of acute pellagra. In his later work on the blood of chronic pellagrins his technique, with the blood, was to draw 5 to 10 c. c., allow serum to separate for 24 hours at 37 degrees, and then add this serum to an equal quantity of ordinary bouillon, incubate this mixture at 37 degrees for 3 or 4 days, and make transplants on defibrinated, rabbit's blood agar, as well as inject into guinea pigs. He has also observed this micro-organism in many cases by making simply stained smears.

Wood, of Wilmington, N. C., reports that he has several times isolated this micro-organism from the blood of pellagrins.

I have been unable to obtain for study a culture of this organism and in my work I have tried a number of times to isolate it, but have been invariably unsuccessful. In fact, my experience in bacteriological research on the blood of pellagrins has been uniformly negative.

Briefly, to sum up the work, I have studied stained liver and spleen smears made at autopsy, have made thick blood smears, laked the hemaglobin and stained the slide, have stained fresh and dried smears in various ways, have incubated blood and citrated blood (citrate of soda, 1 per cent.) and studied smears therefrom, and have put the sediment of citrated blood which had stood for some time, into the peritoneum of rats; I have followed Tizzoni's technique of adding bouillon to serum, planted blood and citrated blood on various culture media; I have injected blood into chickens, rabbits, guinea pigs and rats—all with constantly negative results.

One fact I think worthy of note, and that is that one must exercise great care in fresh smears of blood, or citrated blood, which has stood for a while. I have repeatedly been deceived by numerous small particules of protoplasm, like Muller's blood dust, which have a very active Brownian motion and much resemble motile bacteria, or living parasites.

In conclusion I would say:

(1) That there seems to be present in pellagrins a fairly constant secondary anemia, usually not of a severe type, with corresponding qualitative changes in the red blood cells.

(2) That leucocytosis is rarely seen in pellagra and that this is probably not a phenomenon of uncomplicated pellagra.

(3) That the results obtained by various workers on differential leucocyte counts are very discordant and that conclusions should be drawn therefrom with much hesitation; though a relative, large mononuclear increase seems probable.

(4) That nothing resembling a protozoal parasite has been reported as observed in the blood of pellagrins.

(5) That I have found, in a limited experience, the blood of pellagrins in South Carolina uniformly sterile in cultural work and not infective for ordinary laboratory animals; and that I have not been able to isolate Tizzoni's micro-organism.

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DISCUSSION ON THE PAPERS OF DRS. KERR AND LAVINDER

Dr. R. N. Greene, Chattahoochee, Florida: I wish to make a few remarks on the paper of Dr. Lavinder. Up to the moment of leaving the hospital in Florida for the purpose of attending this conference, we had been making differential blood counts in cases of pellagra, and our findings have been largely in conformity with those reported by Dr. Lavinder. The inference to be drawn, it seems to me, is this: that instead of making the old stereotype statement, as we have been making it, in the absence of distinct signs, that in measles or influenza large mononuclear leucocytes usually indicate malaria, it seems we must add to that list pellagra. In our investi-

gations and examinations we used the azure method in combination with eosin, the smears being taken by the same men and the counts made by the same men in each case.

Dr. C. C. Bass, New Orleans, Louisiana: My work in connection with pellagra has been chiefly along the line of hematology, and experimental, and I want to emphasize what Dr. Lavinder said, namely, that blood cultures have with me been universally negative, and not only have these cultures been negative, but animal injections have been likewise negative in results. In my injections I have used macerated cord, macerated liver, macerated spleen, and brain also, but always getting negative results. Those familiar with the pathology of the disease know that there is usually a short portion of the cord very much softened. I have taken that part of the cord, injected it into rabbits, guinea pigs and chickens, all of which have remained perfectly normal. One set ran as long as three months. Therefore, it seems highly probable from Dr. Lavinder's experience, and from the work I have been able to do, we cannot inoculate rabbits, guienea pigs and chickens with any bacterium that may be present in the blood of patients, or in the lesion which such patients have. I want to emphasize the unreliability of differential blood counts, and the absolute necessity of not drawing any conclusions whatever from differential blood counts made by differential men, especially with reference to the large and small mononuclear leucocytes. Of all things in the world, men will disagree on in differential blood counts it is the proportion of large and small mononuclear leucocytes.

Dr. Walter H. Buhlig, Chicago, Illinois: I am very sorry to say that Dr. James A. Egan, Secretary of the State Board of Health of Illinois, is not here in person to represent the Illinois delegation. The serious illness of his wife has prevented. In his absence he has asked me to represent him.

Dr. Egan began some of this work in Illinois soon after the disease, pellagra, was discovered at the Peoria State Hospital for the Insane by Dr. Zellar, Superintendent of the Hospital, who is on the program today to read a paper. We have gone over a number of subjects there, but our work is more or less elementary as yet. While we have had a number of striking results, they are only suggestive. I simply want to say a few words about the hematological side of the question. We have made 26 or 27 complete blood counts—

26 red counts, I believe—which on an average ran about 4,200,000, although we found cases with two million and a half, and five or six cases of about five million, and the remainder in between these. As a rule, we found the color index a little low, that is, it was of the chlorotic type. Now and then we found it 1 or .9. In one case it was 1.3. That was a case with a very low red count, two and a half million.

With reference to the variation of reds, we found some striking changes making a picture of a severe anemia. Megalocytes were usually present, but not so often as the microcytes. In one case I found megaloblasts. This was the case of a very sick pellagrin in which the examination was made the day before death. I also found in that same patient one or two red corpuscles with nuclear particles. We also looked for polychromatophilia, and while we found it now and then, it was not a constant characteristic. Basophilic granules were not found in the red blood corpuscles. We did not learn anything from the blood platelets.

The white blood corpuscle counts were different from those reported here. One case only had a leucopenia of four thousand; about half of the cases were normal, estimating about 10,000 as the upper limit of the normal. The remainder of the counts were above 10,000, say from 12,000 to 14,000, excepting a few high counts. In one case we found 48,000 whites; in another case there were 36,000. In the one with 48,000 I made an autopsy subsequently, but I did not find anything to account for the increase, except a little spot of broncho-pneumonia about the size of the end of my thumb, which could not have produced this leucocytosis. Our experience has been that patients who have a high count die. We had eight high counts, and of these five patients died. Of these five, all of them had over 14,000 whites. Of the three cases with high counts that lived, one gave a count of 12,600, one 14,000, and another 19,000, the latter a severe case.

In regard to the differential count, we did not find any increase in the large mononuclears as reported here. The large mononuclear is one of the hardest things to tell from a small mononuclear; sometimes you can say this is a small mononuclear and this is a large one, but there are sizes between when one is not so sure. Although we did not find any increase in the large mononuclears, we did find four or five cases with an increase in the small mononuclears, running between 45 and 50 per cent., with a count of over 10,000, making a true leucocytosis. But when the count was over 10,000 or 12,000

we usually found an increase in the polymorpholnuclears, but never any lessening of the eosinophiles as one would get in a true leucocytosis from infection. Once we saw an eosinophile percentage of twelve.

The anemias were secondary in type, sometimes leaning toward the chlorotic side, and at other times leaning toward the pernicious anemia picture. One has to contend with this difficulty in interpreting blood counts in pellagrous patients, namely, that he has not seen the patient before, and, therefore, does not know anything about the blood of those patients before they contracted pellagra.

Dr. Louis Leroy, Memphis, Tennessee: I have but a few cases to report in this line. My findings have been largely in accord with what has been thus far reported. I was surprised to hear the second speaker say that he had been using the eosin and azure stain, for this is the one I have used in my work. I have found in counting all cells a fringe with an average of from 10 to 12 per cent. of large mononuclears, and about 17 to 18 per cent. of small mononuclears, or lymphocytes. I have noticed regular degeneration in a few cases, that is, polychromatic staining, and I have noticed a little irregularity in some of the small cells. I have seen also not infrequently as in other secondary anemias small round cells which apparently were thicker than usual, or spherical in appearance, or outline, that is, not being provided with the usual concavity which we find with the normal red cells. Further than that our cases give an average of 7,000 leucocytes, with the exception of one case which I saw with Dr. Litterer, in which 15,000 leucocytes were found, and in which a staphylococcus infection was also present. The nucleated reds have been entirely absent in the few cases I have had a chance to observe. Altogether the picture has struck me as being a secondary anemia of the chlorotic type. The hemoglobin index has averaged a little over nine-tenths per cent. only. In the fatal cases there has been a great deal of inanition. The counts have been taken from two days to two weeks before these patients have died. I have not had a chance to make blood counts except in those cases that have died a short time afterward.

Dr. C. L. Minor, Asheville, N. C.: There have been several cases of pellagra in Asheville during the past season, the first being diagnosed by my friend Dr. Dunn, who had two or three cases during his term of service at our City Hospital, and I had three in my

service following his. The cases observed by us, with one exception, left the hospital at the end of the season in very good condition, though they will doubtless relapse next spring. They were all of the ignorant poor class and, like all Southerners, all had eaten freely of corn meal, often of the cheapest sort. They applied for admission chiefly on account of the dermatitis and diarrhœa, the dermatitis having in every case been treated for various skin conditions by their home doctors.

The mild cases all did seemingly well, and at discharge the hands showed only a little yellowish discoloration, not enough to attract attention, and in great contrast to the angry red condition on admission.

The stomatitis was present in all, but was mild save in the fatal case, an acute one, where it was severe and distressing.

Diarrhœa was present in all, but in itself would not have justified a diagnosis, the skin lesions being the chief diagnostic feature in each case. In only one case was there any indication present or past of mental involvement, this developing just after discharge from my service of a case that had been under Dr. Dunn previously.

In one of my cases the woman had had the disease for four years, recurring each spring, but she paid very little attention to the hands or the diarrhœa till this year. In this case there was not merely a pronounced dermatitis of the backs of the hands and lower forearms and to a less degree of the flexor surface of the wrists and palms, but there was an eruption on the forehead, chin and back of the neck which was peculiar in that its surface was dark brown, dry, rough and papillary, looking somewhat like shagreen. This cleared up much more slowly than the hands.

With reference to the treatment, we, of course, tried arsenic and many other things, all with little effect. What did do good, however, was the rest, cleanliness, care and good food.

As to its possible former existence in our community, I am sure that had any of us in the past seen such a dermatitis which we misdiagnosed we could not have failed to remember it, and none of our doctors recall any such skin lesions further than five years back, hence I believe the present appearance of the disease is in the nature of a new and more or less epidemic outbreak.

As one of the speakers has said, when spoiled corn meal as the etiological factor is backed up by such scientific workers as the Italians we cannot possibly afford to entitrely reject it without careful study of the whole question. Now that the disease is attracting

so much attention from the profession, I am sure that doctors throughout the South will begin to diagnose the increasing number of cases, and the patients, reading of it, will with the first appearance of symptoms consult the doctor and with early diagnosis they will yield better results to treatment.

DR. WALKER: If I remember rightly, the first case of pellagra I saw was about 22 years ago, and since then I have seen several cases in my practice, and I would like to ask Dr. Williams to give the record of one case which I think he saw 12 or 15 years ago.

DR. C. F. WILLIAMS, Columbia, South Carolina: The case Dr. Walker has asked me to report was not seen by me as long ago as he mentioned. I have been practicing medicine ten years, and this case was observed during the first year of my professional experience. I think it was in 1899 when I saw it. The case was a typical one of pellagra, but at that time we termed it exzema. There was intense stomatitis, profuse diarrhœa, rapid emaciation, and finally delirium and death. We will be able to show you such cases here this evening.

PELLAGRA—ITS RECOGNITION IN ILLINOIS AND THE MEASURES TAKEN TO CONTROL IT

GEO. A. ZELLER

Superintendent Peoria State Hospital
PEORIA, ILL.

The announcement of the recognition of pellagra in Illinois during the past summer made perhaps as profound an impression upon the medical profession of the State as any single occurrence within its history.

Hitherto, if in masked form, the disease was present at all in any of the deceptive cutaneous eruptions, such as eczema, sunburn, psoriasis, pemphigus, erysipelas or pityriasis, or had manifested itself in the form of aphthæ, stomatitis, acute or chronic diarrhœa and dysentery, it was overlooked and included as one of these. If a doubt arose in the mind of the practitioner and he consulted his textbook he was told the disease was limited to Italy and does not occur in the United States, and if greater inquisitiveness were to lead him to consult the latest and greatest work on modern medicine, he would be rewarded with just ten lines upon the subject.

We in Illinois were doubly excused if we excluded our State from debatable territory, even if inquiries came from the Southern States as to the presence of the disease. You of the South have always had diseases which we had no occasion to fear, and even in the worst yellow fever epidemics, when the State was filled with refugees from Memphis, Vicksburg and other points, we looked upon their presence with impunity, conscious that the disęase could not find an abiding place in our latitude.

If we ever quarantined against the South it was only in the extreme southern part of the State along the Ohio River. Almost as unconcernedly do we even now view the agitation caused by the discovery of the hookworm, for we are told that only a sporadic case can occur in our latitude, and under no circumstances would the larvæ mature to spread further infection.

Why, then, be agitated when an inquiry came from South Carolina last winter as to whether we had pellagra?

Of course not, for we were immune on climatic grounds and upon the adidtional assurance that it does not occur in the United States.

Even its recognition by Dr. Willhite in the Dunning Institute at Chicago in June of this year, and its verification by Dr. Lavinder,

occasioned no particular comment, neither did the presence of the disease in the Elgin, Illinois, State Hospital, as reported by Superintendent Podstata, attract special attention. In the former institution there have been to date twenty-six cases, with fifteen deaths, and in the latter a lesser number of cases, also several deaths.

I must confess that I was floating along in fancied security, ascribing to trophic changes, sunburns and even worse misfortunes, the many cutaneous lesions that developed from time to time among those who died of my twenty-one hundred and fifty patients.

Intestinal symptoms were accounted for with equal plausibility inasmuch as asylum diarrhœa has reached the dignity of being included in some textbooks on intestinal diseases.

I never accepted it as a permissible or excusable condition, and fought its return each summer with a vigorous sanitary campaign, an anti-fly crusade and finally by the addition of seven lesser kitchens for the preparation of special diet.

Ours was the asylum for the incurable insane, and was made up of the residual population of the other asylums and the insane inmates of the almshouses. Many were decrepit, with terminal dementia quite common, and an annual death rate of fourteen per cent. was not apparently out of proportion, but whenever a death from preventable disease occurred I went carefully into the causes that led up to it, and every such case was thoroughly discussed at the staff meeting, where nine active members gather daily for that purpose.

One day Dr. F. J. Griffin, one of my assistant physicians, announced to me that he thought he had a case of pellagra, and I went with him to the cottage at once and saw a most typical manifestation, and in the case presented I immediately recognized a condition that had long been with us. It was not the individual case or the dozen more that I found sitting about the porches with more or less pronounced symptoms of the disease, but the memories which they evoked gave them a significance far out of proportion to the actual number of patients at hand. They brought up the existence of similar lesions of varying degree where explanations to the friends of the patients had become necessary.

Instantly my mind went back to the constantly increasing number of sunburns of the previous years, and I realized that we were dealing with a condition that was not new. I went to the correspondence files and quoted the following words in the case of the death of a very excellent lady whose body was shipped to Canada:

"One afternoon she sunburned her hands slightly while out in the woods, where she was taken with a number of other patients for amusement and pleasure."

In another instance in 1908, where the friends became suspicious of excoriated hands and accused us of burning the patient, the following language was used:

"Being an invalid he was allowed to remain in the open air as much as possible, and during this time his hands became sunburned, as is not infrequently the case with these patients. The sunburns were kept in proper dressing and were practically healed at the time of his death, and were in no way a factor causing it."

In March of the present year, immediately upon my return from a hurried tour of Europe, I called the thirty-head attendants into my office and lectured to them at length upon the necessity of observing greater care in exposing patients to the sunlight, citing the numerous instances where the lesions had become sufficiently pronounced to cause distress if not to contribute in a measure to a fatal termination.

The Peoria State Hospital is a cottage-plan institution of thirty buildings and twenty-one hundred and fifty patients. Each cottage has a front and rear ground floor porch co-equal with the dimensions of the building, and capable of accommodating the entire ward. The open door prevails throughout, and upon these porches the patients spend almost the whole of each pleasant day, and for this reason, unless carefully shifted from the sunny to the shady side, are exposed to an unusual degree of sunlight. Since no less authority than Lombroso himself says that no diagnostic eye can differentiate between pellagra and sunburn, we thought that we had a logical explanation of these lesions, and sought to prevent them upon this hypothesis.

Our biennial report for 1906 and 1908 each makes mention of a fatality due to scalds incurred while bathing patients. In each instance the scald extended to a line about four inches above the ankles, bullæ formed and desquamation followed. The treatment applied was that usual in a burn of the second degree. The greatest concern was felt in these cases, and no effort was spared to prolong their lives. They were certain to become the subject of the most rigid investigation and severe criticism of institutional methods was sure to ensue. The one patient survived one day, the other eleven days, and in both instances the coroner was called. The nurses in charge stoutly denied having scalded the patients, but appearances

were so overwhelmingly against them that their summary dismissal was considered a minimum penalty, and was promptly ordered.

I will exhibit photographs today that would exculpate those nurses and strike a blow at circumstantial evidence that no criminologist would deem possible. In fact, I have already written to them and tendered them their former positions, and assured them that if the feeling that they were instrumental in the loss of a human life was weighing upon them, they could dismiss the thought in the newer light that has come to us by reason of the recognition of this new-old disease. I am aware that I invite ridicule when I go back to the dead and attempt to ascribe a condition to causes other than those accepted at the time, but my own testimony before the coroner's jury was that in neither case were the scalds sufficient to produce death in a normal individual; but I had no other explanation to offer at the time, nor would one of you have advanced a different theory, although in the future such occurrences will at least cause us to exclude pellagra before we condemn a possibly innocent person.

These circumstances are related not to substantiate the fact that we have pellagra with us at present, for that is undisputed, but they are resurrected for the purpose of demonstrating that the disease in one form or another has been with us unrecognized for a number of years. To establish or disprove this theory is an all-important point in the study of the history of the disease.

If there is such a thing as jumping at a conclusion I did so on August 7, 1909, but I did so with a clear knowledge of seven years' occurrences to guide me, and a hurried inspection of the hands and features of my patients that revealed twenty cases and opened the possibility of many more. That day I sent the Secretary of the State Board of Charities a telegram announcing twenty cases of pellagra in the institution, and forwarded letters of the same purport to the Governor and to the Secretary of the State Board of Health. Recognizing the fact that a positive diagnosis could only come from those familiar with the disease, I wrote to the Surgeon-Generals of the Army and of the Marine Hospital Service also, inviting their co-operation in the study of the disease.

The response was prompt from every source. The Governor called me up within an hour and next day sent a representative from the State Board of Health, and Dr. Lavinder came out from South Carolina by order of the Surgeon-General of his department, saw thirty or forty cases, diagnosed the disease and returned to his post. Captain Siler, of the Army Medical Corps, came soon after and was

joined later by Captain Nichols, and the two officers spent a month in a careful and systematic study of every phase of the disease, and the result of their labors is in print and will constitute a valuable addition to the literature of pellagra.

Simultaneous with their arrival came Dr. Buhlig, of the Northwestern Medical School of Chicago, at the request of the Illinois State Board of Health. We added such equipment to our laboratory as was indicated, and he had an assistant on the grounds for one month, during which time he himself made many visits. Distinguished men from all parts of the State came to study the disease, and a visit from the State Board of Charities, headed by Dr. Frank Billings, resulted in a recommendation to the Governor that a commission be appointed to investigate the disease and incidentally the food supply of all the State institutions of Illinois.

Upon this recommendation the Governor named a commission which, in point of intellectual ability and professional attainments, would be hard to duplicate in any State of the Union. The commission is just organizing, and it would be premature to speak of its work, but that new light will be shed upon the subject of pellagra no one conversant with the high character of the Board will for one moment doubt.

I aimed only to deal with the recognition of pellagra in Illinois, and in a conference of this magnitude, where the subject will be treated from every possible viewpoint, I have no right to digress. I will add, however, that I regard the spread of pellagra in the United States as a very grave matter. Seeing it develop in every ward of my institution and to the larger extent in the tent colonies for consumptives where the most nutritious diet of milk and eggs is given, seeing it develop in men and women of splendid physique as well as among those showing more marked mental and physical decline, I can only await with dread its appearance in the citizenship outside the institutions. Rumors of such cases have reached me, but not until Sunday, October 24th, was a clearly defined case brought to my attention. The victim is a well to do man of seventy, living the life of a retired farmer in a city of ten thousand population.

He was brought to the asylum by his family physician for diagnosis, and it was clearly and easily made. He has the typical marks of the disease, although at present experiencing but little distress. His case is most significant if it is the precursor of an invasion that seems to have found in the skin and alimentary canal of the American a fertile field for extension. I leave for others the presentation

of the various theories of its etiology, but desire to say that an amount of work along every line has been done in our institution since August 7th that would hardly seem possible, all of which will eventually reach the interested world.

This is the situation as it stands in Illinois today, and it requires no gift of prophecy to foresee that when solar influences become active next year, and with the interest of the profession thoroughly aroused in an endeavor to detect the disease, the center of interest will be in the Mississippi Valley, and very likely in the Peoria State Hospital. For that reason I propose that the next National Conference on pellagra be held in Peoria, Ill., about the middle of June, and that an association be formed here today for the further prosecution of the study of the disease. Illinois, as the greatest of the corn-producing States, cannot but feel an absorbing interest in a condition ascribed to its principal product, and I am here to assure every active worker every possible aid in his researches. By that time the knowledge of the disease will have spread to every State, and an assemblage of this character will bring information from the remotest section in the country.

To sum up the situation in the Peoria State Hospital, on November 1st I will say that since August 10th there have been officially recognized 130 well defined and diagnosed cases of pellagra, with one hundred additional cases on the wards with symptoms sufficiently pronounced to warrant their inclusion, but who for the present are suffering no inconvenience. They are being carefully charted and will form the basis of observation for next year.

Of the 130 cases, 75 were women and 55 men.

Of these, 30 women and 15 men, a total of 45, died since August 10th.

Post-morten examinations were held in 36 of these cases.

Of the living, the average age is 51 years.

Average number of years insane, 16.

Average period resident in this institution, 3 1-2 years.

Of the dead, the average age at death was 54 years.

Average length of time insane of those who died, 17 years.

Average period of residence in this institution of those who died was 4 years.

Of the living and dead, the average age was 51 years.

Average period insane of the living and dead, 15 years.

Average period of residence of the living and dead in this institution, 4 years.

Of the total cases, 12 were received from other asylums within the current year.

The death rate of cases actually transferred to the hospital for treatment has been 34 per cent.

The youngest pellagrin was 22 years old.

The oldest was 85 years old.

Of the total, 12 were epileptics and seven were in an advanced stage of tuberculosis.

The list contains but one colored person, a man, who survives with marked dry and thickened palmar tissue.

If to these are added the 100 living cases still on the wards, the mortality stands 20 per cent.

In conclusion, I trust that the conference will not take my confession too seriously. The early California pioneers looked for a long time with unconcern at the black sand in the creek bottom before they found that it contained gold, and many apples fell from the trees before Sir Isaac Newton discovered the law of gravitation.

Wherever the disease has been newly recognized there invariably came an admission that conditions somewhat similar had existed for years and had been overlooked, and in some instances institutional authorities recalled cases as far as thirty years back, therefore I feel that I owe no particular apology for the oversight; but do feel, now that we are face to face with the condition, that we should meet it energetically, intelligently, and always, so far as I am concerned at least, with the feeling that we are menaced with a national scourge.

ASPECTS OF THE PELLAGRA PROBLEM IN ILLINOIS

J. F. SILER
Captain Medical Corps, U. S. Army

Part I.—A Statistical Study of 100 Cases at the Peoria State Hospital.

HENRY J. NICHOLS
Captain Medical Corps, U. S. Army.

Part II.—Notes on the Epidemiology of Pellagra.

PART I.

A study of the literature on pellagra reveals the fact that not only is the etiology an unknown quantity, but the clinical picture of the disease is a most variable and changeable one. From personal observation and a study of summarized cases histories of the patients at Peoria, we have attempted to determine as to whether or not any constant symptoms and pathalogical findings may be noted. For statistical purposes we selected 100 cases for study, and the figures presented, with few exceptions, are based on this number of cases. As pellagra was officially recognized at Peoria about August 10th, in the midst of the period of acute exacerbation (May to November), it is entirely a matter of conjecture as to the extent of its prevalence in the Peoria State Hospital. About August 22d the entire institution, consisting of 2,140 inmates, was examined for pellagra, with the result that about 70 patients presented typical symptoms, and 150 additional patients were classified as typical or suspicious cases. Patients presenting the typical symptoms of an acute attack rapidly increased, and on the date of our departure, September 24th, it could conservatively be stated that at least 125 patients showed acute pellagrous symptoms.

Age of Pellagrous Patients—The average age of patients in the Peoria State Hospital was 47; the average age of pellagrous patients was 50; the average age of those who died in the institution was 57, while the average age of pellagrous patients who died was 55, only two years below the general average age. As to age decades:

From 20 to 29 years, 4 cases.
From 30 to 39 years, 18 cases.
From 40 to 49 years, 28 cases.
From 50 to 59 years, 29 cases.

From 60 to 69 years, 16 cases.
From 70 to 79 years, 3 cases.
From 80 to 89 years, 2 cases.

In 75 per cent. of the cases the patients were between 40 and 59, the youngest being 22, the oldest 85.

Previous Attacks of Pellagra—An effort was made to trace the disease through previous years. The members of the medical staff and selected nurses and attendants who had been connected with the institution for several years were closely questioned concerning the previous appearance of pellagra among these patients. The fact was easily established, in each case, on the testimony of 2, 3, 4 and occasionally 5 and 6 different medical officers and nurses, that pellagra had been prevalent in this institution for at least three years prior to the summer of 1909. Seventy-two per cent. of the cases under discussion had suffered from previous attacks during the summer months, the average number of attacks being two. Of those who had suffered from previous attacks, 25 per cent. gave a history of three previous attacks, 52 gave a history of two previous attacks, and 23 per cent. gave a history of one previous attack. During previous years these cases were thought to be cases of severe sunburn.

Duration of Insanity—The average duration of insanity in this series of cases is 13 years. Thirteen years' institutional life may have some bearing on the development of the disease. Pellagrous cases making up this series have averaged four years at the Peoria State Hospital. These two facts, average duration insanity and average stay at Peoria, would seem to indicate that the exciting cause of the disease is present within the institution. At the same time no nurses, attendants or employees have shown the disease.

Distribution as to Sex—Fifty-six of the cases were females; 44 males. This is in marked contrast to the finding in other sections.

Occupation—An attempt was made to classify the cases as to previous occupation, but owing to the incomplete records forwarded to the institution with the patients and the long duration of insanity, it was impossible in many cases to collect any facts worthy of record. Of those cases showing record of previous occupation there were 29 housewives, 13 farmers, 15 laborers, one photographer and one clerk.

Other Diseases—Thirty-six, or 36 per cent., of these cases showed other organic disease exclusive of amebic dysentery, which, although present among the patients, had not been definitely recognized. Among other organic diseases noted were 12 cases of epilepsy, 8

cases of pulmonary tuberculosis and 12 cases of organic heart lesions. These diseases apparently exist in pellagrous patients in about the same proportion as is found in the general population of the institution.

Results of Statistics Bearing on Previous Social Condition of the Patient—An attempt was made to collect data relative to these patients, as to institutional life; the use of corn-bread, corn mush, hominy or other corn products; the quality of corn used; the social status of the patients; and the occurrence, before commitment to institutions, of erythema, involving the backs of the hands, of summer diarrhœa and accompanying change in mental condition. For this purpose circular letters were sent to relatives and friends of the patients, and at the present time forty answers have been received. The points of possible interest brought out were about as follows:

1st. A large proportion of the patients had been inmates of almshouses and asylums for many years.

2d. A surprisingly small percentage of the patients had used corn products as an article of diet before entering institutions, and in none of the cases had corn products continuously been used as an article of diet.

3d. With two or three exceptions these patients were extremely poor and had lived under bad sanitary surroundings.

4th. In only two cases had an erythema been noted on the back of the hands, and as the hospital records show these patients to have been insance 16 and 23 years, respectively, it is altogether probable that these lesions had no connection with pellagra.

AS TO THE CLINICAL PICTURE.

1. *Cutaneous Symptoms*—The skin symptoms were constant and characteristic and the course was generally as follows: A bright red erythematous area appeared on the dorsum of both hands, usually simultaneously. Extension occurred within a few hours, and after a few days the color changed to a purplish red or reddish brown. At the end of the second week desquamation began, the epidermis peeling off in small, thin, bran-like scales. Desquamation was usually complete within a month of the first appearance of the lesion. The line of demarcation between the affected areas and the surrounding healthy skin was distinct and marked. The dorsum of the hands showed involvement in all cases. Extension of the erythema to the forearms occurred in 36 per cent. of the cases, and

in 17 per cent. of the cases erythematous areas appeared on other portions of the body. These erythematous areas usually appeared on the forehead, the eye-lids and neck. No characteristic lesions were noted on the feet, and in only two cases were such lesions observed on the legs. The palmar surface of the hands was not involved, except in two cases, in which slight extension occurred from the dorsum over the thenar eminence. The so-called characteristic erythematous areas on the vulva in the female were not noted. In a few of the more severe cases the vulva showed marked irritation and excoriation, but the lesions were in no respects characteristic of pellagra.

Bleb formation occurred in 10 per cent. of the cases. It was a noticeable fact that bleb formation was always associated with cases presenting most severe symptoms in other respects. In some cases vesicles only were noted, while in other cases the vesicles became confluent, forming one large bleb. In 66 per cent. of the cases in which bleb formation occurred, death resulted. This fact would seem to have an important bearing on the immediate prognosis.

Seborrhœa has been referred to as a characteristic finding. This condition was noted in a few of the most severe cases, but was by no means a constant or characteristic symptom.

2. *Digestive Tract Symptoms*—In order to arrive at any definite conclusions as to the significance which may be attributed to symptoms involving the mouth and intestinal tract, several factors must be considered.

a. The inmates of this institution are incapable of caring for their teeth, hence, personal care of the teeth is an unknown quantity. It naturally follows that particles of food cling to the teeth and become embedded between the gums and roots; decomposition of these food particles occurs, with resulting decay of the teeth, inflammatory reaction involving the gums and buccal mucous membrane, and the entrance into the stomach of these products of decomposition. These mouth conditions have been eliminated, so far as is possible, by the employment of a dentist. The following extract from the 1908 report of Dr. W. J. Weatherwax, dentist for the institution, is worthy of presentation: "Many mouths are a mass of decayed teeth and roots, surrounded with badly inflamed gums with pus exuding constantly from pockets."

b. The food supplied these inmates, while on a par with that supplied other institutions,—even better as compared with some institutions—and beyond criticism as to quality and mode of preparation,

is necessarily limited as to variety and component constituents. The resulting difficulty in assimilation with accompanying digestive disorders has a reflex bearing on any oral and intestinal symptoms which may be present.

Mouth—In 71 per cent. of the cases redness of the tongue or buccal mucous membrane was noted. In general terms it may be said that there were no distinct and characteristic mouth symptoms in mild cases. In cases of great severity, the mouth symptoms were marked and characteristic. The tongue was fiery red and edematous; the buccal mucous membrane was intensely inflamed. Ulcers were frequently noted throughout the mouth,—on the tongue and buccal mucosa,—and excessive secretion of saliva was of common occurrence.

In some cases this inflammatory reaction was so severe as to prevent protrusion of the tongue, and interfered greatly with the taking of food. This severe inflammatory reaction was of immediate grave prognostic import. Patients presenting such mouth symptoms seldom recovered from the acute exacerbation.

Diarrhœa and Dysentery—Diarrhœa was present in 85 per cent. of the cases, and in the milder cases was of short duration. In those cases presenting the most severe skin and mouth symptoms the diarrhœa was severe and persistent. As will be shown later, this diarrhœa may, to a very large extent, be attributed to factors other than pellagra, *per se*. Eighteen per cent. of these cases presented all the clinical symptoms of dysentery—frequent small stools, containing blood and mucous, and of very offensive odor. Of those patients who died or who were rapidly failing, 66 per cent. presented the clinical symptoms of dysentery. In the more severe cases it was very evident that some factor, other than the causative agents ordinarily associated with diarrhœa and dysentery, was present. These cases showed a profound intoxication, and physical failure was rapid and marked.

3. *Blood*—Hemoglobin averaged 81 per cent. The white cells averaged 9,904 and red cells 3,859,000, respectively, per cubic m. m. During the acute stage of the disease the blood picture was that of anemia. The color index was high in the acute cases. In 23 per cent. of the cases the white cells were normal; in 10 per cent. they were below the normal, and in 67 per cent. there was a leucocytosis of greater or less degree.

Blood Pressure—Nothing of interest or value was noted in this connection. The Stanton instrument was used, and the average blood pressure was 146.

Spinal Tenderness—Spinal tenderness was present in only 14 per cent. of the cases and apparently had no particular connection with the disease.

4. *Reflexes*—The patellar reflex was normal in only 20 per cent. of the cases. The remaining 80 per cent. showed a departure from the normal, 62 per cent. showing increased reflex, and 18 per cent. showing diminished or absent reflex. In 32 per cent. of the cases the plantar reflex was normal. The remaining 68 per cent. showed departure from the normal, 53 per cent. showing increase and 15 per cent. showing diminished reflex. The wrist, elbow and epigastric reflexes showed nothing worthy of note.

As some prognostic value has been attached to the Babinski reflex in connection with pellagra, it may be stated that this reflex was obtained in 15 per cent. of the cases, of whom only 20 per cent. died. As 80 per cent. of the cases showed abnormal patellar reflexes and 68 per cent. showed abnormal plantar reflexes, it would seem that the condition of these reflexes is of diagnostic importance.

5. *Mental Condition*—The question of pellagrous insanity in connection with these cases is entirely a matter of conjecture. It is of interest to note, however, that within the past two years 10 per cent. of the cases were committed to the institution from civil life. Four cases were adjudged insane two years ago, five cases one year ago and one case six months ago. Four of the cases in question are classified as dementia praecox, one as alcoholic insanity, one as involution psychosis, two as paresis, and the remainder as dementia unclassified. Of the total number of cases in this series 50 were classified as terminal dementia, 17 as dementia praecox, 5 as manic depressive, 3 as paranoia, 7 as imbeciles, and the remainder as various other types of insanity. In 57 per cent. of the cases no mental change was noted during the acute attack. The remaining 43 per cent. showed mental change, 10 per cent. becoming more irritable and excitable, while 33 per cent. showed increased depression or melancholia. None developed suicidal tendencies. Some of the cases in this series may be pellagrous insanity.

Eye Findings—Dr. Carroll B. Welton, of Peoria, examined the eyes of more than 50 per cent. of these cases, as well as an equal number of controls, and has very kindly permitted us to incorporate in this report his general conclusions, which are as follows:

"*Conclusions*—1. Paralysis of the eye muscles is found in the later stage of the disease in a small percentage of cases. Conjunctivitis is not an uncommon symptom. Early forming cataracts are frequently noted, and the metabolic nature of this disease is supported because this condition is generally considered as an altered state of the nutrition of the lens when occurring in normal individuals. Inflammation of the optic nerve and retina is observed in a relatively large percentage of cases. Common and most pronounced of all the eye changes is involvement of the choroid.

"2. In none of the cases presenting eye symptoms could the character of the changes be regarded as pathognomonic of pellagra.

"3. That the severity of the eye symptoms runs parallel with the severity of the general manifestations of the disease, and that the finding of marked eye changes adds to the gravity of the prognosis in pellagra and indicates, in a large percentage of cases, an early fatal termination."

Treament—The treatment of the condition was discouraging. Thyroid tablets were administered in a number of cases with no result. Fowler's solution, in increasing doses, was used without any noticeable effect. Atoxyl, in 5-grain doses, was used, one injection every seven days, and in only one or two cases could improvement be attributed directly to medication. Normal saline was used in the more severe cases with only temporary improvement. Bulgarian tablets were used in modifying the milk in the hope that improvement in intestinal symptoms might result. This diet had not been given extensive trial, and, while no definite conclusions were reached, it is believed to be worthy of further trial.

Mortality—Of the patients making up this series, 22 per cent. died, 10 per cent. were failing, 17 per cent. were improving and 51 per cent. had recovered from the acute attack. It may conservatively be estimated that the death rate from pellagra at Peoria ranges between 25 and 30 per cent.

SUMMARY.

1. A large percentage of the cases gave history of previous attacks.

2. A large proportion of the cases had been insane for many years and gave histories of long stay in institutions.

3. About one-third of the cases showed other organic disease, exclusive of dysentery.

4. All cases showed cutaneous lesions involving back of hands, and in a number of cases this was the only symptom noted.

5. Bleb formation occurred in a small percentage of the cases and a large percentage of those cases showing blebs died.

6. Symptoms involving the mouth and intestinal tract were characteristic in the more severe cases only.

7. The only reflexes of diagnostic value were the patellar and plantar reflexes. These reflexes, in a large percentage of cases, showed departure from the normal.

8. A small percentage of the cases were committed to the institution during the past two years and some of these cases may be pellagrous insanity.

PART II.

The failure of continental students of pellagra to agree on some one definite feature of a corn diet in relation to this disease and the scepticism of English writers of any form of the corn theory force American observers, in dealing with a disease, recently so widely recognized in this country, to undertake to work out the problem for themselves. This attitude applies to symptomatology and pathology as well as to etiology, because it is very evident that the most contradictory symptoms and lesions of concurrent diseases have been described as pellagrous. In fact, it applies to the very conception of the disease itself. Is pellagra an entity with manifestations in the skin, intestinal tract and nervous system, or is it a symptom complex developing under certain conditions of diet as a sequel in other diseases?

At Peoria a start was made by the microscopical examination of the stools and a remarkable state of affairs was at once revealed. Control examinations were then made of a number of non-pellagrous patients of the same general class. For comparison the results of examinations made on American troops in the Philippines under field conditions are also given.

	Inmates with Pellagra.	Non-Pellagrous Inmates.	Soldiers in P. I.
	88.	101	454.
Negative	14.7%	51.4%	48.8%
Amœbæ	37.5%	11.8%	16.2%
Flagellates (alone)	20.4%	11.8%	34.5%
Encysted Protozoa (alone)	27.2%	24.4%	
Per cent. of cases with Protozoa	85.3%	48.6%	51.2%

The only ova found were those of oxyuris in one case.

This condition was considered very unusual for a temperate climate, pellagra or no pellagra, and an examination of water was made. Amœbæ, flagellates, and fermenting organisms were found in the tap water at the asylum on each of the three examinations.

The water comes from two sources: First, the larger part ordinarily from an artesian well. Second, a small amount, 10 to 20 per cent., from the city water supply.

The artesian water is forced out by air pressure from a depth of 1,800 feet into a surface tank; it is then pumped to an elevated tank for distribution. The city water is pumped direct to the elevated tank.

The artesian water coming directly from the pump had something over 100 colonies to the c. c. flagellates, but no amœbæ or fermenters. The city water had about 10 colonies to the c. c., flagellates, but no amœbæ or fermenters. The surface tank had no amœbæ or fermenters. The trouble therefore lay in the pipes and elevated tank, as a result of some previous contamination, possibly from the surface tank, which is not thoroughly protected; possibly from some break in the pipe system.

Flagellates are not always considered pathogenic, but instances like the present are constantly multiplying, especially in the tropics, and more and more suspicion attaches to them. Five per cent. of all the people examined showed well marked evidence of active amœbæ dysentery present in the mucous and blood and very motile amœbæ. The amœbæ in the other cases were "resting."

On taking up autopsy work the chain of evidence in regard to the presence of an unusual amount of dysentery became complete. Eighteen autopsies were held from August 10th to September 20th. Twelve of these were done by ourselves, and of the 18, 12, or 66 per cent., showed well marked ulcers of the colon; two of the cases were as good specimens of amœbic colitis as can be seen in the tropics; one patient with pellagra died of peritonitis following a perforation of an amebic ulcer. These colons were characterized by either large irregular ulcers, thickening of the gut, or follicular abscesses. Of the remaining six cases two had tubercular ulcerations, one trichinosis, the others follicular enlargement of the lower ileum and colon. The mesenteric lymph glands were not materially enlarged.

The autopsy reports are given in full in another place, but no other organs showed any constant pathological change.

In looking over the autopsy record for the last two years it was noticed that in most cases the intestines were not examined; however, in twelve cases they were opened, and in eight of these mention is made of ulcerations of the colon. One case of multiple abscess of the liver is on record.

What bearing, if any, have these findings on pellagra? They may have an important bearing as a predisposing factor in rendering the mucosa of the colon more permeable to toxins and is interesting to note that Neusser refers to old and fresh ulcers as a finding in the intestines in pellagra, and another author speaks of perforation as an occasional cause of death.

The findings given above may also prove to be worth considering in connection with the seasonal occurrence of the disease.

No satisfactory reason is known which would apply to this country except greater amount of diarrhœal disease in the summer months. This is well recognized at Peoria and almost all the patients gain weight with the beginning of the cold weather. We know that protozoa are more active during warm weather. Is it not possible that these protozoa during the summer months produce conditions in the colon which are more favorable to the elaboration and absorption of a specific toxin of pellagra?

The matter of predisposition is an essential one as far as the patients at Peoria are concerned, because, in the first place, the inmates are the poorest class of humanity in Illinois, and most of the pellagra developed among the worst of this bad class. In one-half of the cases sufficient cause of death was found autopsy and in one-third of the living cases well marked organic diseases other than senility and dysentery were present. Almost all the cases belong to the untidy class. Out of 2,100 patients 20 per cent. are constantly untidy and 5 per cent. occasionally so. In many of these cases untidiness is undoubtedly synonym for dysentery.

Granting for a moment that a protozoal infection of the colon is a predisposing cause of the majority of the cases at Peoria, what is the additional fact, or which gives the skin symptoms, stomatitis and general debility? The parasitic theory of any disease is the most attractive one and there seems to be a strong undercurrent of feeling in favor of it in this country, but nothing was seen to substantiate such an impression in regard to cultures from the blood, spinal fluid or organs at autopsy. Cultures were made after approved methods (see appended chart) from these various sources in fifteen cases and no growths of any kind were obtained and nothing unusual

was seen in fresh or stained blood films. The whole picture of the disease is that of an intoxication; the gradation from the slightest taint of the most marked cases, and the absence of any constant finding at autopsy except in the intestinal tract point in this direction. In this connection it should be noted that while we have tabulated one hundred cases, many more undoubted cases kept cropping out, and still more suspects remained. It looked as if some common cause were at work on the whole population, and the most susceptible were affected. This leads to the question of diet and the overwhelming evidence of some form of corn theory among those most concerned cannot be lightly cast aside. The possibilities are as follows:

1st. Good corn is injurious in large quantities. This theory can hardly be used to explain the trouble at Peoria, because the purchases for 1908 show only thirty-eight pounds per year per patient received, or a little over one ounce per day. As a matter of fact some classes of patients receive rather more, but not exceeding two ounces per day in a liberal diet. It has been estimated that about 16 ounces of white rice per day are necessary to produce beriberi. If corn holds the same relation to the disease a much larger amount would have been necessary at Peoria.

2d. Spoiled corn is responsible: This is not the case as far as can be told by the senses. The corn used was inspected on the 'change and at the mill, and it is No. 2, the best that is on the market. Nothing in the way of a "food scandal" could be unearthed. The corn products do not have to be transported far, and if any poisons develop they cannot be told by ordinary means.

3d. Toxins may be developed in the intestines by fungi or bacteria on a corn diet. In regard to moulds, several trials were made but no moulds were found that survive the heat of cooking. Of course, this does not exclude them, but it makes them seem less likely as a cause. On the other hand, a spore-bearing bacterium was repeatedly found in corn-meal, which survived steaming for two hours, and this seems to offer possibilities.

In order to learn something of parasites on corn, a trip was made to the University of Illinois, and it was found that only in recent years has any definite work been done on this subject, and that much remains to be done, especially in regard to bacteria.

The most common cause for disease of ears on the stalk has been found to be a kind of diplodia, which has been stated by one author to be on the increase in recent years, and along with aspergillus, penicillium, etc., has been advanced as a cause of pellagra. The

Department of Botany has agreed to furnish us cultures of the known moulds and bacteria and work will be done with these.

The most promising field seems to be along the line of an intoxication produced by toxins of corn products in a damaged intestine, and this will be followed up. Several instances have been heard of, of a toxic action for animals, first of an excessive corn diet, also of a diet of corn gluten infected with moulds; and there are no doubt several kinds of disease connected in some way with corn, but pellagra must be due to one, not to several kinds of corn poisoning.

The outstanding fact about corn raising in the last fifteen years is that the shelled corn is marketed four to eight weeks earlier than it used to be, that the time of weathering and drying on the stalk is cut short and that more trouble is experienced in handling the corn and preventing it from going bad. This fact, with the considerable increase of the use of corn products in the past few years may prove to be of significance.

The recommendations respectfully submitted for the Peoria State Hospital are:

1st. Cleaning out the pipes of the cold water supply; better protection of the surface tank; regular examinations of the water for bacteria and protozoa.

2d. Examination of stools of possible cases of dysentery, especially among the untidy, and appropriate treatment of dysentery cases.

3d. Examination of corn from the diet of the more debilitated patients.

SUMMARY.

1. The pellagrous patients at the Peoria State Hospital show a very high percentage of protozoal infections of the colon.

2. A study of the patients and diet points to some endogenous intoxication.

3. The protozoa may play an important predisposing part in the seasonal production and absorption of an endogenous toxin.

DISCUSSION ON THE PAPERS OF DRS. ZELLER, SILER AND NICHOLS

Dr. L. J. Pollock, Dunning, Illinois: The discovery of pellagra in the State of Illinois is indicative of its widely spread distribution in the United States. The importance of its recognition in other States than those of the South is self-evident.

The first case occurring in the Cook County Institutions was noted in August, 1908, when the diagnosis was as yet not made. With the subsequent occurrence of three other cases presenting the same symptomatology, course and fatal outcome, and not being familiar with the occurrence of pellagra in the United States, we came to the conclusion that we were dealing with a clearly defined disease with which we were unfamiliar. A clue as to its nature was offered to us in the description of pellagrous insanity in Bianchi's Psychiatry. From the onset the next case was thought to follow closely the description of Italian pellagra, but it was not until the inquiry of the South Carolina State Board of Health was addressed to us that the diagnosis of pellagra was definitely made. The first public notice of the occurrence of pellagra in the Cook County Institutions followed the confirmation of our diagnosis by Dr. C. H. Lavinder on July 18, 1909.

The devious route at which we came to the conclusion that we were dealing with pellagra proclaims the necessity for widely distributing literature concerning the nature and prevalence of this condition throughout the United States. This importance is further emphasized by the fact that pellagra has existed in the Illinois institutions for a number of years before its recognition, Dr. Podstata, Superintendent of the Northern Hospital for Insane, recalling cases occurring in his service several years ago.

A description of pellagra is superfluous following the numerous reports upon this subject, but the various modes of onset and multiplicity of symptoms justify a brief analysis of the cases arising at the Cook County Institutions.

The entire number was 26. Females 13; all insane; males, 13; 8 insane, 2 in the poor house and 2 in the hospital for tuberculosis.

The duration of the fatal cases averaged thirty-four days. The duration in the remaining cases averaged forty days.

The nativity was as follows: United States, 7; England, 1; Ireland, 9; Bulgaria, 1; Germany, 5; Denmark, 1; Austria, 1; unknown, 1.

Of those foreign, the Bulgarian was in this country six months, the others ranged from eight to forty years. They had been confined in the institution from four months to twelve years.

The psychoses at the time of admission were as follows: Dementia paralytica, 4; alcoholic, 5; dementia precox, 5; paranoia, 3; acute confusional, 1; melancholia, 1; senile dementia, 3.

The symptomatology has been in general fairly uniform. The

cases had variable onsets, some with malaise, mental depression and indifference; others with anorexia, nausea and vomiting, increasing weakness, dizziness and staggering gait; all presenting a dermatitis, usually symmetrical in distribution. In some, however, one extremity became affected before the other. The lesion commenced in the majority of cases as a hardened erythematous condition, becoming pigmented, fissuring and finally desquamating, leaving an atrophic skin behind; in the others the onset of the dermititis was sudden, with the formation of blebs, which, when burst, left the underlying portion of the skin moist, pink and glazed. The exposed portions of the body were usually attacked, but in some few cases the ankles and knees and in one case the buttocks were affected; one case is prominent on account of the wide distribution of the dermatitis and the large involvement of the anterior surface of the arm. In this case there was a dermatitis of the anterior and posterior surface of the lower one-third of the forearm, the dorsum of the hands, a symmetrical area on the anterior surfaces of both elbows, both knees, the popliteal spaces and ankles. The mucous membranes showed changes, in the mouth as a severe stomatitis, with numerous ulcers and heaped up epithelium; in some cases a conjunctivitis, and in the majority of women affected a vaginitis was present. The gastro-intestinal system showed stomatitis, denuded tongue, marked ptyalism; in some cases anorexia and refusal to eat, vomiting and severe diarrhœa, associated with tenderness over the abdomen. The stools were lienteric and contained small quantities of blood. The nervous system showed weakness, staggering gait, dizziness, exaggerated knee jerks, followed by their disappearance, choreiform movements and in one case symptoms of meningitis, as shown by marked spasticity, a double knee and ankle clonus, Gordon Oppenheim and Kernig's sign, the patient dying of respiratory failure. The mental state has been one of depression, occurring upon the former mental condition, bordering upon melancholia with crying, refusal to eat, etc. In one case, with the subsidence of the skin lesion, the patient entered into an excited, clouded, delirious condition in which she has since remained.

The course in those cases not dying during the acute exacerbation has been one of uniformly progressing cachexia and weakness, and despite the disappearance of the gastrointestinal lesions and skin disturbance, the patients progressively became weaker and died.

Of those cases which died, numbering 14, 1 died of apoplectiform convulsions of dementia paralytica; one in a low muttering delirium

with choreiform movements; one of respiratory failure; two of tuberculosis; one of carcinoma of the stomach; and the others of exhaustion.

The medicinal treatment has consisted of the hypodermic administration of arsenic salts, with no appreciable effect.

The pathology as seen in the post-mortem examinations upon four cases showed nothing distinctive. Besides the findings of cachexia, some fatty degeneration of the liver, kidney and heart, injection of the large intestines, prominent Peyer's patches, showing shaven beard appearance near the ileo-cecal valve and enlargement of the mesenteric glands were present. In the nervous system some edema of the meninges and microscopically chromatolysis of the anterior horncells of the cord.

Dr. C. H. Lavinder, Washington, D. C.: I had the pleasure of going to Peoria, Illinois, when pellagra first developed there. So far as the symptomatology of the disease in Peoria is concerned, I think it is very wise for all of us to tabulate symptoms, for it is the accepted opinion that pellagra varies in various sections of the country and in various seasons of the same sections of the country; that is to say, some particular phenomena will be prominent in one season or section which may not appear in other seasons or sections.

So far as Captain Nichols' paper is concerned, working in Columbia with Dr. Babcock, I myself have found protozoa in the stools of pellagrins, but not so frequently as he has found them. I think it is an interesting observation. The plagellated organisms which I saw are considered non-pathogenic.

Dr. J. W. Mobley, Milledgeville, Georgia: I wish to make a few remarks in connection with Dr. Nichols' paper as regards the frequent complication of enteric ulcer of some character associated with pellagra.

In looking over the pathological record at the Georgia State Sanitarium, covering possibly 100 cases, more or less, away back as far as 1897, according to the descriptions of the post-mortem examinations made, there were cases in which there was pigmentation of the hands and feet, skin abrasions, and stomatitis. Of course, these cases were not considered at that time pellagrous in character. I made many of these post-mortems myself, and the fact prevailed that ulcers frequently appeared about the ileum in those cases which now, I have every reason to believe, were pellagrous. Furthermore, there

was an increase of intestinal parasites. Dr. Willetts, recent pathologist to the Georgia State Sanitarium, in 500 unselected cases for examination, found in the feces about 57 per cent. of infections with some form of intestinal parasite, and it is noteworthy that of these 500 cases 35 were pellagrous. Of the pellagrous cases, there were about 40 per cent. infections with intestinal parasites; the most important were infections from the strongoloides, and uncinaria, both being bloodsuckers, and especially strongoloides, which was found in 20 per cent. of the cases, and which is the hardest parasite to dislodge from the intestinal mucosa. I have been connected with this institution twelve years, and in going back over the records I can recall these cases as pellagrous quite frequently. Just at this point I want to speak of two particular conditions, which are almost the same, and yet there is a little difference in them. It is in this relation that we are often perplexed to know whether we have a nervous disease primary to pellagra, or whether the pellagra is primary, and the nervous symptoms, being trophic in character, are secondary. About five months ago a young lady was admitted to our institution, who gave a history of epilepsy, with organic brain disease, dating from the age of 3 years. She had convulsions. These convulsions were frequent, and after about three months it was noted that there was some stupor. She was not profoundly stupid. While she was epileptic—and these patients are usually dull mentally—yet her expression was bright. She had some scanning speech. The reflexes were all exaggerated. In a general way, she had all the symptoms of cerebro-spinal sclerosis. She became somewhat stuperous, finally developed stomatitis with an aphthous deposit around the margins of the tongue. The inflammation extended, after which she became more stupid. She was moved to the infirmary, and developed a mild diarrhœa. This case represents the active type of intoxication psychosis to which I wish to call your attention, and this woman died in four weeks after her removal to the infirmary. She had a temperature the first week which was below normal. The second week the temperature became a little elevated, while in the third week it rose to 105. The skin eruption began to appear at about the end of the third week. It was not pronounced in character, but simply a brownish red pigmentation about the hands and about the mouth. She then developed all the symptoms apparently of spinal trouble, dying almost in a condition of leptomeningitis.

Here we have a case of progressive organic disease of the brain and spinal cord concluding with such symptoms as give us every

reason to suspect an acute and profound intoxication of pellagrous origin. On the contrary, it would not be entirely out of place to regard the stomatitis as coincident, and the trophic disturbance about the hands and feet as secondary to the organic nervous disease, in which pellagra played no part in the etiology.

CASE NO. 1:

Miss L., white female; age 30; admitted March 4, 1909; native of Indiana; resident of Georgia one and one-half years. Family and personal history free from syphilis, insanity, pellagra and tuberculosis, though patient had some remote physical signs of tuberculosis on entrance examination; very well nourished; gait spastic; marked inco-ordination; some intention tremors; speech scanning; marked mental reduction. Convulsion since child three years old. Patella reflex exaggerated; Babinski and Gordon absent; some clonus. Patient imperfectly oriented as to time, place or person; attention poor; some anesthesia and atrophy of lower extremity. Voluntary grasp quite deficient. While the patient's mentality was very limited, the expression was cheerful and eye rather bright. The first known pellagrous symptoms developed about four weeks before death; there was some stupor, with increased saliva, the patient was disposed at this time to hold food in the mouth—a mild stomatitis gradually came on, some apparent increase in the tremor. The sore mouth increased with aphthous deposits about the margins of the tongue. Deep reflexes generally increased; no hallucinosis. Diarrhœa set in one week from first symptom—five to ten offensive stools daily; acid in reaction; dark brown color; temperature 98 to 99 F. during first week. This represents the morbid picture for about two weeks, when all symptoms increased in activity with temperature 103 F. A dark reddish pigmentation developed about the dorsal surfaces of both hands, with a few discrete areas about the mouth. The temperature gradually rose to 105 F.; pulse 120—the entire morbid cycle covering something over three weeks, when the patient died in a spastic state quite similar to lepto-meningitis. This case also represents, in its relation to insanity, an acute intoxication psychosis, with a fulminating conclusion. First heading mental classification; Acute Intoxication Psychosis.

CASE NO. 2:

Mrs. H., white female; age 23 years; native Carolinian; resident in country up to several years ago, since which time she has lived in city; admitted into sanitarium February 4, 1909. Family and per-

sonal history negative as to insanity, tuberculosis, syphilis, sprue or pellagra. Since marriage the patient has been operated on for appendicitis and has shown some hysterical tendency from time to time. The first evidence of mental disturbance was shown about one week before admission to State Sanitarium, when the patient expressed some apprehensive hallucinosis, with slight mental confusion. She was supposed to be suffering from neurasthenia, and was sent to a general hospital. Her condition grew steadily worse, so she was committed to the State Sanitarium. At time of reception patient was poorly nourished; mental confusion marked; disoriented as to time, place and person. Attention very poor; insight lost. By the second day after admission patient had developed a mild delirium, and further tests to determine the psychic reaction were inapplicable. She was now in a state of active hallucinosis, with a psycho-motor reaction indicative of fear and impending danger. The gait was spastic; patella reflex exaggerated; Babinski inconstant; ankle clonus marked in both. Some apparent hyperasthesia. There was no evidence of skin disturbance up to this time. There was a slight stomatitis, with whitish deposits around margin of tongue. Stools liquid and while infrequent at first, an uncontrollable diarrhœa finally developed. Temperature for first week 97 to 99 F.; pulse 90 on average. Sore mouth gradually grew worse with all symptoms increasing in intensity; temperature during this exacerbation goes to 104 F. Following this there was a remission of acute character of all symptoms, the patient dying after about six weeks from exhaustion. A reddish brown pigmentation appeared on dorsal surfaces of hands about the fourth week of the disease; some discoloration about the mouth. The last phase of the disease presenting the physical signs of amyotrophic lateral sclerosis.

The mental aspect of the disease is indicative of a profound Infective Exhaustion Psychosis; or the second classification we have already referred to.

Dr. J. W. Babcock, Columbia, South Carolina: I have not the honor and pleasure of a personal acquaintance with Dr. Zeller, but I think Dr. Zeller, like all asylum doctors, including myself, is here absolutely in the interest of truth. I do not think Dr. Zeller or myself represents any peculiar school, as to the theory of the development of pellagra. We are all here to learn the truth, and to confer together in the interest of truth. This subject has been forced upon the attention of the public through the often maligned asylum and

the asylum doctor, and as far back as twenty-five years ago we were asked by so great a light as Weir Mitchell what contribution had the asylum doctors made towards the progress of medical science? So I want to ask this assembly in which the minority is largely composed of asylum men to recognize that out of these asylums comes this momentous question. (Applause.)

Again, in behalf of the asylum doctor, and it has been my pleasure to serve as a private in the ranks with men for twenty-five years, whose position is like nothing else on earth, I will say that there are some men whose prime characteristic is moral courage. In all my experience with asylum doctors, I have known of no one of them who has shown the high degree of moral courage that Dr. Zeller has in reading his paper from this platform this afternoon, and if I may presume on this occasion to thank Dr. Zeller personally and in behalf of asylum doctors throughout the length and breadth of the United States, then I want to return our humble thanks to him for standing here in South Carolina and reading us that magnificent paper. (Applause.) I know many asylum men, but I know not a single one in America or in Europe who would come before an audience like this and stand before the people of the United States and proclaim in the manner he has done here this afternoon what he believes to be the truth. (Applause.)

Dr. Julius C. Sosnowski, Charleston, South Carolina: I was struck with the ages of the patients mentioned by Dr. Zeller, Dr. Nichols and by Dr. Siler. The average age seems to be 50 for the older patients, and the youngest in the thirties. While I have seen very few cases of pellagra, yet I recall twelve that came under my observation in Charleston, and of this number six were below 14 years of age; one was 18; four were between 20 and 40, and one was above 40. Six of these patients were white and six colored. The ages of these patients would seem to be at variance from the experience of those who have already spoken.

Dr. M. B. Young, Rock Hill, South Carolina: Speaking of the ages of patients afflicted with pellagra, I recall one case of a little child, two years and five months old, in which I made a diagnosis of this disease, which was confirmed by my professional colleagues, who saw the case in consultation. This child developed a rash on the backs of the hands and on the feet. The child was accustomed to playing with scouring soap, and the mother thought the dermatitis

was produced by the child lathering itself with this soap. She did not call my attention to the rash at the time I first saw the child. The child went on for a month or two, when the red rash disappeared, and the skin became crusty and began to peel off. The child developed temperature. Very little attention was paid to the rash, which appeared on the hands. I dismissed the case for a while, until I was sent for again, when the child developed diarrhœa. The stools were of a muco-bloody character. This continued for one or two days until the stools became very bloody. The child was demented, and it sometimes required two or three in the room to hold it in bed. The child became so weak that it could not get out of bed. At about this time I had an acute attack of malaria and left the case in the hands of another practitioner. Not having read what Dr. Nichols has said about amœbæ at the time, I did not think of them, although Dr. Miller, who saw the case, and myself, spent hours in looking for amœbæ in other cases that were known at the time to be pellagrous.

Dr. H. E. Menage, New Orleans, Louisiana: We have had in one of the wards of our hospital the case of a little girl in whom the disease was typical. The child lived for a week after we first saw it. There was a previous history of two years' duration of the disease. The interesting features about the case are the pathologic findings in the brain. The child was idiotic and cried constantly.

Upon opening the skull cavity the brain presented a peculiar appearance. The anterior convolution was large and edematous. The posterior convolution behind the fissure of Rolando was small and vermiform, in that it looked like a bundle of worms. On either side of the middle of the main comissure and just back of the fissure of Rolando, bilaterally and symmetrically, there were two large cysts filled with a fluid and encapsulated with rather thick walls.

I thought I would mention this case because it is not reported in the paper which I shall read for Dr. Dyer.

Dr. John N. Thomas, Pineville, Louisiana: I want to ask Dr. Zeller what part corn bread played in his dietary at the institution in Peoria, and how much corn bread he gave his patients.

Dr. Zeller: Corn bread constitutes a very insignificant portion of our diet. About two ounces a day is given on an average to all patients, so that corn is not a great factor in our diet.

DR. THOMAS (resuming): My experience is almost similar to that of Dr. Zeller's, except that at our dinner meal the only bread used is made from corn meal and the patients are given it liberally.

Since the papers and investigations of Drs. Babcock and Lavinder, I have been so thoroughly convinced that corn bread plays a very important part in the causation of this disease, that I have eliminated it entirely from our list of eatables at the Louisiana Hospital for Insane.

The cases presented for examination seem to be mostly chronic cases, and judging from the pictures shown and the cases we have seen, none of them seem to be mild, but are chronic cases that have existed for three or four years. If it is possible it would be well to exhibit a few mild cases.

As to the water supply and suspicion of bacterial infection of the Insane Hospital at Peoria, I wish to say that we receive our water supply from an artesian well over 1,100 feet deep, and the water has been passed upon by a chemist as being absolutely pure, so that we have eliminated that feature as a possible cause of the disease. Our food supply is liberal in amount, and we can ascribe the disease to nothing else but bad corn meal.

I have seen cases of pellagra develop in patients who have never been off the ward at all, and I am convinced that the disease is either due to impure corn meal or to an infection of some kind.

DR. GEORGE A. ZELLER, Peoria, Illinois: The water supply of our institution is derived from the city mains of Peoria. It enters a clean cistern or reservoir from which it is pumped into circulation. The anomaly of the situation is that there are 300 employees who drink this water all the year around, and have done so since the institution was founded, yet there has never been a case of dysentery in any employee of the institution. There has been no case of diarrhœa in our institution among the employees. When we come to take up the question of the water supply, and remember that our employees are healthy and vigorous and are absolutely free from any intestinal disease traceable to the water, and then add to that the 1,800 or 2,000 patients in the institution who have no diarrhœa the year around, these things of themselves invalidate somewhat the theory of a polluted water supply. It is true, amœbæ have been found in the water, but I have some ideas about that which I will not give you at this time. Dr. Buhlig made several analysis of our water and also found amœbæ and flagellates, but he says that such a condition

is not unusual, and I rather gained the impression from him that Chicago water does not taste good unless it has a few amœbæ in it. I would like to hear from Dr. Buhlig on the subject.

Dr. I. W. Faison, Charlotte, North Carolina: In the first place I want to sanction and endorse what Dr. Babcock has said in behalf of Dr. Zeller. I want to say to him that as a Northern man he is almost good enough to come South to live. (Laughter.) The cases of pellagra that have come under my observation have been acute. Those I have seen have had no skin lesions at all, and I am fully persuaded that these cases can occur and do occur as pellagra without any skin lesions whatever.

I recall the case of an old maid (I do not care to give her name), who was taken sick three or four years ago. She remained under my care and observation without a diagnosis of pellagra having been made until the spring of this year. She only had a nervous indigestion, and nervous trouble, or weakness, without any manifestations of the disease which would enable me to make a diagnosis of pellagra. She had an acute stomatitis, with a very red flannel-like tongue. There were blebs over the mucous membrane of the lips and tongue in her case. In the other cases I have seen the tongue was a clean red, but in this case there was an immense crop of blebs. She had considerable pain from the condition about the mouth. Salivation was terrific, and the odor which came from her mouth was almost unbearable. She had intense diarrhœa; the mucous membrane of the rectum was greatly inflamed, and she had what I might call a fiery red mucous membrane all over the vulva and vagina. With this stomatitis and salivation, as much as three quarts of saliva came from her mouth in 24 hours. From a woman who had weighed 98 or 95 pounds she ran down to 80 pounds. She became very weak and had to be confined to bed. At present, to all intents and purposes, this woman is in better condition than she has been for the past ten years. She has increased in weight from 80 pounds to 110, weighing more than she ever weighed before in her life. The stomatitis was painful, and nothing that I ever did to her afforded any relief except a solution of argyrol, 10 grains to the ounce. I washed out her month with peroxide of hydrogen, followed by boracic acid solution, and by argyrol every three hours. The argyrol in this case relieved the pain in her mouth like magic. In a few days the blebs began to shrink. There was no pus. They fell off like old vaccinated scars, leaving a healthy mucous membrane. This

woman had false teeth, and yet under the plate that carried the false teeth redness did not appear. When we took out the plate and examined the condition of the mouth we found an absolutely normal color of the mucous membrane; but behind the plate and inside of the cheek the mucous membrane in different portions of the mouth showed this fiery redness. In addition to the use of argyrol, I gave her hypodermically every day two-thirds of a grain of atoxyl and 1-30 gr. strychnine three times a day. I kept this up for three weeks, and with it cut out the use of all corn bread. She showed remarkable improvement.

I came here last year persuaded that corn was the cause of pellagra. I left very much in doubt on this point. I stand here today and say that my opinion is that corn products furnishes the cause; next year may be so changed that corn is not the cause of this disease. Of course, I reserve the right to change my mind as to the cause of this disease. In my opinion there is some condition in corn meal or in corn bread which acts as the exciting cause of pellagra, and what that condition is we do not know, and when these men across the water, who have studied the disease for years and years, say that the disease is produced by damaged corn or maize, we should not be easily led away from that idea.

In the treatment of the case I have referred to I neglected to say that I flushed out daily the colon with normal salt solution, and I must say it was one of the best remedies I used. I followed the treatment I have outlined for four or five weeks, when the redness of the mouth disappeared; the blebs gave way; the patient's appetite returned, and the woman felt so good that I woke up to the fact that I had gone far enough and was afraid to use atoxyl further. Accordingly, I stopped its use, but in ten days after I stopped its use the mouth became inflamed again. I again began the use of atoxyl, and in five days improvement began and continued. I kept her on it. She then went to the mountain country, is now able to walk about, her cheeks are taking on the glow of youth, and she is a healthy woman today. I do not know whether she is cured or not, and I told her that we would have to wait until next spring and see what is going to happen, and that I could not tell at present whether she would be a well woman or not.

Now, gentlemen, I am going away from this conference before you change my opinion again that the product of corn is not the cause of pellagra. I do not believe it is communicable. I do not believe that it is contagious; therefore, I am not afraid of it in these two channels.

SOME DIFFERENTIAL POINTS IN THE SKIN LESIONS OF PELLAGRA—REPORT OF A CASE WITH REMOVAL OF SYMPTOMS

ISADORE DYER, PH. B., M. D.

Professor on Diseases of the Skin, Medical Department, Tulane University of Louisiana.

NEW ORLEANS, LA.

There should be some clear definition of the symptomatology of the cutaneous evidences of pellagra so that the diagnosis may be made by any observant physician. At present the varied reports of cases, many incomplete, lead to confusion in diagnosis, and perhaps may mislead to the report of cases as pellagra which are something else.

When Dr. Searcy was studying his pellagra cases at Mt. Vernon Asylum in Alabama he came to New Orleans and showed me photographs and specimens which I could not recognize as fulfilling the essentials for a diagnosis of classic pellagra. The mass of his cases have, however, since then established the correctness and value of his observations.

Since the agitation of the pellagra question in the South I have seen several cases which might have been mistaken for pellagra, and most of these went on to fatal terminations.

Altogether, I have had the opportunity of observation in four cases of pellagra, two in my own practice. I have had two other cases of undoubted pellagra under my advice—one in Mississippi and one in Louisiana. In no two of these six cases were the skin evidences just alike, yet all were cases of comparatively recent development, and all in their first attack.

The cases seen by me had certain points in common, but were sufficiently dissimilar to suggest the probability that the skin evidences of pellagra are more apt to be the direct reflections of the associated etiologic factors than a separate, integral symptom, and that the severity and the type of the lesions are significantly proportionate to general systemic involvement. I have some photographs sent me by Dr. J. D. Donald, of Hattiesburg, Miss., taken of the hand of a case of undoubted pellagra. His case died* after an attack of a few weeks' duration, but developing fulminating eruptions and disturbances of all membranes, especially mucous membranes of mouth, genitals and adjacent organs. The photographs are excellent

*Report of Dr. Donald's case related in Case II.

delineations of pellagra—*when you know that they were taken of a pellagrin's hand.* They would as readily be taken for photographs of a case of blastomycosis, the papillary character of most of the eruption being quite apparent. Yet the beginning and the course of this eruption on the hands was as typical as it could be in a classic case of pellagra.

My first patient (Case III, *infra*) with pellagra presented an entirely different eruption. The hands were affected on the dorsal surfaces, particularly associated with the orifices of the hair follicles; the joint areas were softer and less involved. While the entire dorsal surfaces of the hands were covered, the areas between the joints were especially thick and rough, made up of closely aggregated papules, pin-head sized, uniform, and so rough as to be almost spinous to the sense of touch. This eruption of the hands extended in long triangles up the extensor surfaces of the forearms and the lower third of the arm, the base of the triangle being at the wrists and the apex just above the olecranon process. At no point after leaving the wrist was the triangle wider than two inches, and the tapering was almost geometrical in contour.

The coloring in the eruption was peculiar—the yellow, tan color of bran. A like eruption was on the legs, but here the worst eruption was just below the knee, tapering in a triangular form to the ankles, where it stopped. This case had typical "rough skin" with fine scales on the surface.

Except for the configuration, the absence of itching, and the associated symptoms, a diagnosis of pityriasis rubra pilaris would have been more than probable.

When first seen this patient had a severe stomatitis, anorexia, diarrhœa (with frequently bloody stools), the genitalia were engorged, raw, excoriated, painful, and the menstrual periods had been attended with unusual dearth of flow.

Hebetude, emaciation and insomnia nights were marked symptoms.

My second patient (Case IV, *infra*) was a man of 49 years of age, giving the history of a laparotomy for appendicitis some six weeks before the eruption appeared on his hands. He had no eruption or other symptom save that which was present on his hands.

This showed as a bilateral and symmetrical eruption covering the dorsal surfaces of the hands, even to the edges of the finger nails. The eruption extended up on the wrists and encircled the wrists. The eruption on the wrists was marginated by a waving band of infiltration, smooth, elevated, dull red in color, and at the border considera-

bly lighter than the rest of the affected area on the wrists. All of the eruption on the wrists was swollen but unbroken. On the backs of the hands, however, and on the fingers, even along the linear aspects of these, there were many small vesicles, so superficial as to break easily, and in places had already crusted. Where the crusts had dried some time, the areas of the eruption presented distinct papillomatous elevations closely packed, rough on top, scaling and a deep yellow in color. At no time, so the patient stated, had the vesicles been large.

This case presents the picture of a classic pellagrous skin on the hands. It is easily differentiated from vesicular eczema by the persistence of the vesicles, the development of papillary areas and by the marginate, erythematous, *elevated and infiltrated border,* all of the latter evidences testifying to a deep seated affection, beginning deep and not a mere catarrhal process started in the mucous layer.

Now, each of these three cases present types of pellagra with resemblances to other affections. Each began with an erythematous process, at one stage having nothing especial to distinguish it from an erythema of ordinary type and of simple origin.

As the group of erythema multiforme has been studied its variants have increased considerably, including many diseases of the skin of exudative origin and due to as many different causes. We find the erythema of caloric type, caused by too much heat, cold, or sunshine when too direct, exactly similar erythemas starting from the ingestion of certain drugs, or even certain foods.

Reflex conditions in the habit of the individual may bring on simple or grave exudative erythemas, the borderline between these and destructive neuroses being hard to define at times. Toxins from food and even the injection of serums or vaccines in susceptible individuals may profoundly affect the skin, even causing areas of hemorrhage and of destructive sloughings, if the hemorrhage is diffuse.

As the degree of this or these antagonistic substances may affect the individual, so the results may appear, locally or generally, involving the skin more or less profoundly at the same time.

This idea alone can satisfy my own unrest at the varieties of pellagra which are being described. So many of Searcy's cases were bullous in type. The beautiful illustration which accompanies the pellagra article by Rist in the *Pratique Dermatologique* (*Tome* 3), pictures a dermatitis of foliaceous form with vesicles undeveloped. Others frame a description of hands which are keratosed and fissured.

With each of these forms, however, there are related enough con-

comitant symptoms to fix the diagnosis of pellagra. I believe it is wrong, then, to state that the erythema is *the* characteric symptom of the disease, and particularly when, as in some instances, the erythema has given place to hyperplastic changes in the skin.

The symptom-complex in pellagra should be arrived at by associated evidences, if possible.

Just as I am satisfied that I might have to prove the diagnosis of any one of my cases, just so I am certain that there may be cases arising during the present campaign against the disease in which I might have the right and reason to question the diagnosis. Most other diseases of the skin have more exact characteristics, for the reason that they are usually more specific in their etiology and pathology and the variant in symptoms of local nature is not as elastic or irregular as it would appear to be in pellagra.

I now wish to report briefly the few cases which have come under my care, either directly or indirectly, in order that they may be made of record, and that a further memorandum may be made of the treatment followed, as this seems to differ somewhat from accepted methods.

Case I.—Miss S., Alabama. First seen June 23, 1908. Patient related that the condition had appeared early in March, the first symptom being a sore mouth, as she expressed it, "like salivation." The eruption on the hands appeared on the 3d of May, now limited only to the hands, forearms and neck. The type of eruption was distinctly erythematous without scaling, a dull, brickdust red in color, having the appearance of sunburn. Eruption was bilateral and symmetrical on the extensors of both forearms, the backs of the hands and on both sides of the neck. The patient related that she had grippe in February and had not been well since.

The following blood examination was made, which seems to bear very little on the case:

Examination of Specimen of Blood Smear.

4,540,000 red blood cells to cu. m. m.
4,120 leucocytes to the cu. m. m.
No plasmodia malariæ found.

The patient was not seen again, but the physician referring her to me wrote subsequently that she had entirely recovered, the eruption having then disappeared under the treatment advised, which consisted in regular doses of quinine sulphate three times a day, two grains at the dose, and salicylate of soda at the same dosage.

Case II.—Mrs. X. (Dr. Donald's case). I wish to quote Dr. Donald's description of this case as given in his letters to me for the chief reason that no alteration of his narrative of the case could in any way increase the value of his observations so excellently given:

"The case as I know it presents the following history, viz.: Female, age 34, married, mother of one child, 11 years old. Had had laceration of cervix and pelvis; has had several operations for their repair; has never been very well since the child was born. First seen by me about five weeks since (April 1, 1909). At that time complained principally of a rundown feeling, loss of appetite and general weakness; no special pain. Rather thin, pale and cachectic look; tongue slick, divested of epithelium and red. Complained of general burning in stomach and bowels. Uterus enlarged, soft and bled on slight manipulation. Was put on tonic treatment but continued to decline for three weeks. Burning grew worse about this time; dark red erythematous spots appeared on back of hands, across the metacarpophalangeal joints and spread rapidly over entire dorsal surface of hands; is very annoying, burning and painful. Spreads to anterior surface of wrists, one week later begins to desiccate and desquamate. The latter is now very marked and extensive; line of demarcation from sound tissue very marked. Tongue and buccal mucous membrane at this time very much inflamed and covered with small ulcers very thickly. Profuse salivation, constant nausea; no diarrhœa but constant irritation and burning about anus and vulva. Temperature within the last ten days varies from 99 1-2 degrees a. m. to 102 degrees p. m. No mental symptoms thus far, but physical prostration very marked.

"Treatment at first was Gooddell's chlorides, then 1-3 grain doses argent nit. before meals with lac bismo and maltopepsine after meals. Now 1-8 drop doses of carbolic acid with 1 oz. doses Phillip's milk of magnesia; soothing applications to hands; 1-150 grain doses of atropine sulph. for salivation; all of which has done absolutely no good.

"I find that the husband suffered from a similar trouble for three years, but of a much milder type. He has now been well about 18 months, with the exception that his mind is not near so active as formerly.

"My diagnosis is pellagra; any light you may give me will be very

much appreciated. Have had the hands photographed and will send you a copy if you wish.

"Trusting to hear from you at once, yours most respectfully.

(Signed) "J. D. DONALD."

N. B. (I suggested the administration of quinine in large doses, but the suggestion came too late as the subsequent details will show. —Dyer.)

"April 27, 1909.

"I enclose herewith photos of hands as per promise; should have preferred to have had them colored, but could not get it properly done. My patient continued to go to the bad rapidly; the desquamation of hands was complete, extending into the palms; the eruption around anus and vulva, extensive and destructive, extending well into the vagina. Delirium set in four days since, rapidly increasing until this A. M. at 5:30 o'clock when death closed the scene.

"Nothing ever seemed to benefit her in the least; on the other hand, actually all medicine seemed to aggravate the disease.

"I trust I shall not again meet with a similar case. I thank you very much for your letter and the interest you manifested in the case. I feel that I would not fully have discharged my duty to the profession nor to humanity if I should not publish the case."

CASE III. Mrs. T., Louisiana. First seen June 17, 1909. Age 25. Two months ago eruption began on hand, two months after child birth. Eruption began with "chapping of both hands"; now involves both hands, forearms, as well as the mucous membranes of mouth, throat, genitals; the bladder as well affected. Nausea constantly present without reference to injestion of food.

Examination of the patient showed her to be emaciated, depressed, apathetic and indifferent to most everything about her. No specific nervous symptoms.

She was promptly placed on large doses of quinine (ten grains A. M. and P. M.) for seven days, which was administered in the form of the hydrobromate, as patient expressed a fear of idiosyncrasy with the drug. In addition to the quinine the following prescription was given after meals:

Rx. Liquor sodii arsenitis, 2 drops;
Tinct. nucis vomicæ, 10 drops;
Elixir lactat. pepsin, 1 drachm.

For the first two weeks of treatment the patient improved very little; strychnin had to be administered from time to time and her dietary had to be reduced to liquid food. The eruption on the hands and arms, and that on the legs (described in main text of this paper) materially improved, losing its inflammatory character and drying to the appearance of sand paper on the skin. The mucous membranes of the mouth and genitals, however, kept raw and seemed to refuse to heal under any treatment at first. Daily starch baths with permanganate mouth wash and douches finally proved effective, and at the end of about three weeks the general symptoms improved and the patient was convalescent.

When last seen, the first week in October, the patient had no evidences of the disease whatsoever; had grown stout, her appetite had returned and she expressed herself as feeling free of any distressing symptom of any sort. On October 26 a report from the home of the patient stated that she was "still improving; no new symptoms."

The treatment with quinine was maintained throughout in this patient and stopped only on her visit to me in October, and the following prescription ordered to be taken for six or eight weeks without interruption:

Rx. Liqour sodii arsenitis, 4 drachms;
Tinct. nucis vomicæ, 1 oz.;
Tinct. cinchonæ, 6 oz.;
Elixir simplicis, q. s. 12 oz.
M. et Sig: Teaspoonful after meals in water.

Case IV. Mrs. McK., Mississippi. Age 42. This patient was seen by my associate, Dr. Henry E. Menage, on September 25, 1909. He reported the case as typical of classic pellagra with a history of one and a half months' duration for the eruption. Patient, however, had shown progressing debility for four months, with bad memory. The eruption was described as xerodermic and occurring on the hands, forearms and over the olecranons; some on the ears. A blood examination was made by Dr. C. C. Bass, which is interesting, as showing a positive Wasserman, as follows:

Hemoglobin.. .. ..46%
Red Cells.. 3,135,000
Leucocytes.. 8,700
Small mononuclears.. .. .. 19%

Large mononuclears. 3%
Polymorphonuclears. 78%
Wasserman's reaction positive with both lecithin and liver extract as antigen.

This patient was treated with atoxyl, but this was discontinued and the quinine and arsenic, nux vomica, &c., was given instead. Irregular reports were received from the patient, but stating that her condition had improved.

Case V. Mr. B., Mississippi. First seen October 7, 1909. History of recovery from laparotomy for appendicitis six weeks ago. Eruption present not over three weeks; began with redness and swelling. Not restricted in dietary since discharged after operation. Does not eat corn products habitually. General and family history good. Married, with children. General appearance good; complexion clear; no sign of emaciation; no nervous symptoms whatever and has had none. Above average intelligence. Clearly understands the seriousness of his condition.

The eruption on both hands covered the dorsal surfaces of fingers, body of hand, and also whole of the wrists; the latter are encircled by the eruption which here appears as an infiltrated erythema, deep dull red in color with a yellow tinge. The borders of the erythema are very much elevated above the level of the rest of the eruption. No break in the skin on the wrists. On the hands and fingers a marked high grade inflammation in process. Vesicles plentiful and close together, especially broken in the centre of the hand with weeping. The vesicles over the fingers seem to be most recent and here they are most numerous. The part of the hands next to the wrist is covered with marked keratinization associated with papillary growths. Some scaling. The whole area of the eruption is characterized by the yellow pigmentation and both hands are considerably swollen. Not much pain nor much itching; generally uncomfortable.

Treatment was begun at once with instructions to the patient to take quinine hydrobromate in ten grain doses twice a day for three days; then five grain doses three times a day for three days; then five grains twice a day for a week. In addition, the patient was given a prescription calling for the following:

Rx. Liquor sodii arsenitis, 2 1-2 drachms;
Tinct. nucis vomicæ, 1 oz.;
Tinct. cinchonæ, 2 oz.;
Elixir calisayæ, qs. 6 oz.

A local application of a protective ointment was ordered.

Under date of October 21, the patient for the first time writes that the eruption has dried and except for the harshness and thickened skin and the yellow color he would think that he was nearly well.

In addition to medication this patient was instructed to add to his ordinary diet the juice of one or two oranges, or of one or two lemons each day. He was ordered to eliminate corn products and to add to his dietary or to increase the amount of rice and lentils.

Patient was seen October 27 and presented a good general appearance. The eruption had entirely dried, leaving keratinized areas over dorsal surfaces of fingers, backs of hands and wrists being simply reddened—but without any lesions. Quinine continued in 2 grain doses A. M. and P. M., and the arsenic compound was also continued.

This case is one of the six referred to in which the treatment has not been conducted long enough to know what the result will be, but the improvement under the quinine was sufficiently rapid to argue that it is a therapeutic agent of material value in the treatment of these cases.

OBSERVATIONS ON THE TREATMENT OF PELLAGRA.

Since the first patient with pellagra came under my care I have realized that the treatment has been purely speculative and that whatever good results have been obtained have only arrived from treatment aimed at a correction of the status of the circulating blood in the victim of the disease. No great stress has been laid by any one using arsenical preparations in claiming that these had any reactionary effect on the nervous system or that this was the object. More recent laboratory experiments with derived serums have all pointed to the need of antagonistic principles in the blood and not through the nervous system.

My own limited experience with the disease has emphasized the one point that so long as the originating cause of pellagra is unknown we may speculate as to the factor producing it, but the fact remains that it presents many symptoms which argue a disease due to a toxic substance and symptoms which are like those found in other diseases in which the cause is known.

For years I have treated all types of toxic erythema where the specific cause was not determined with quinine and salicylic acid salts. The success arrived at made me give quinine in the first

case and I have continued to use this as the mainstay in each case of pellagra that I have had to treat, or for which I have advised treatment. In each case the symptoms were promptly controlled with quinine (given usually as the hydrobromate) in good sized doses and by keeping up the quinine continuously. In two cases the symptoms have disappeared entirely. In the last two cases the treatment has not been followed long enough to establish a definite report, but each case has improved enough to make the prognosis favorable.

I have no argument to make for quinine as I have used it with empyric judgment and have continued its use because the results have been good—so far.

PATHOLOGY OF PELLAGRA

H. F. HARRIS, M. D.

Secretary State Board of Health
ATLANTA, GA.

There are few if any diseases characterized by perceptible organic lesions the pathological anatomy of which is so difficult to arrive at as that of pellagra.

This is to be accounted for first and foremost by the extreme chronicity of the disease. At this point it may perhaps be well to say that some of the earlier American writers on this subject, and those to whom the credit is largely due of calling attention to the wide prevalence of the malady, generally fell into the error of describing the characteristic exacerbations that come from time to time in the course of this affection as being "acute pellagra." As these reports were usually made from asylums there is little room to doubt that probably most, if not all, of the cases referred to occurred in persons already victims of pellagrous insanity. Certain it is that in a large number of instances of the disease that I myself have seen, both in and out of asylums, in not a single instance could there be any doubt as to its chronic nature. Still another difficulty lies in the fact that these patients rarely die in the earlier stages of the disease. At such times the true character of the affection from which they are beginning to suffer is very seldom diagnosticated, and even where this is done the patient is taken off by some intercurrent malady and the morbid anatomy and histology of the two diseases is almost necessarily more or less confounded. Since the introduction of modern methods of histologic examination there is not in the whole literature, so far as I am aware, an account of a thorough post-mortem examination in an uncomplicated case of pellagra in the earlier stages. Another difficulty has been that the grosser changes are found very inconstantly in most of the internal organs, and are relatively of little importance—it being in the central nervous system, where the alterations are of a microscopical character, that we have to look for the true seat of this affection. As it is only within recent years that our methods have been so developed that many of these alterations can be made out, it follows that the descriptions of the morbid histology given by the earlier writers is of comparatively little importance. In the absence of such methods

of examination one can well understand and sympathize with the despairing statement of Bertherand who said "from an excess of fatality pellagra has no causation, no treatment and no morbid anatomy."

Up to the present time all recorded post-mortem examinations with thorough studies of the tissues have been made without exception on old pellagrous subjects, and we are therefore constrained to regard as being typical of this affection those alterations discovered in the terminal stages of the malady. I would have no hesitation, however, in predicting that the future will show that the initial changes are in the central nervous system.

However, before describing these alterations attention will be given to certain changes in the other viscera that are of clinical importance, and still others will be mentioned on account of their theoretical interest.

Skin—A knowledge of the skin lesions in pellagra dates from Casal's first observations which began about the year 1735, although his monograph on the subject was not published until 1762. In the meantime Thierry had gained access to Casal's manuscripts, and published, in January, 1755, an account of the writings of the latter on this subject. The alteration begins, as first shown by Reymond in 1889, with considerable suddenness, developing during the course of 24 hours after a prodromal period of greater or less length. Merck has recently asserted that there is often a preliminary rash, occurring as discreet maculæ, that last from a few days to a few weeks. The erythema begins on the back of the hands, and at the bases of the fingers. It is at first a livid red, and after a few days becomes covered with scales and shed-off epithelium; this hyperkeratosis continues throughout the course of the eruption, and is exceedingly characteristic. The tissues are swollen as a consequence of the increase of both blood and serum in the derma, and as a result the normal elevations and depressions of the skin become more marked than usual, giving to the back of the hand a wrinkled appearance; the contrast between the youthful face and the aged characteristics of the hand are most striking in some subjects. This lesion extends gradually until in most cases it covers the entire backs of the hands and may reach up to a greater or less height along the forearm and even to the shoulders. It does not often extend to the tips of the fingers, but may do so. After several recurrences the backs of the hands become pigmented and in some cases the skin becomes permanently thinned.

Similar lesions are observed on the backs of the feet, though by no means so common as in the location already described; they always occur with or following the eruption on the hands. In that most excellent monograph of Valdes that writer refers to the frequency of the occurrence of the lesions on the backs of the feet in Yucatan, and it would appear to me from the descriptions I have read of the disease as it occurs there that it is more frequent here than in Europe. The erythema may spread to the ankles and legs.

In quite a number of cases the lesion is observed on the face, beginning usually on the bridge of the nose and gradually extending over its entire surface and down on the cheeks, and in extreme instances may reach to the chin and lips, and spread itself finally over the entire countenance. It is more frequent in men than women. The scalp remains normal. The eruption may be in small distinct spots, though it is usually confluent. In some cases it occurs also on the neck and extends down the sternum, giving rise to the so-called "Casal's necktie."

More rarely still other parts of the body may be affected, the change being observed on the elbows, on the arm and on the skin, covering the popliteal space, on the scrotum, around the anus and in the perineal region. In women the vagina may be inflamed and later ulcerated. In a rapidly fatal case in a white man, following the eruption on the hands, I recently observed spots scattered over the various parts of the body of an irregular form, sharply circumscribed, and pigmented to such a degree that they appeared almost black; they were not preceded by an erythema. Similar discolorations occurred in the perineal region, on the scrotum and around the anus. In the severer forms, particularly the backs of the hands, sometimes exhibit small bladder-like elevations filled with serum, which later burst, leaving superficial ulcers that heal very slowly; occasionally similar lesions are found on the backs of the feet and even on other parts of the body. It is said that in rare instances the erythema may cover the entire body. In the mildest forms of the disease no skin lesions occur.

On microscopic examination, as first shown by Babes and Sion, sections of the hyperemic skin exhibit a slight serous exudate, with a few leucocytes, and peculiar homogeneous, metachromatic masses of what appears to be coagulated albumin; the sweat glands contain metachromatic granulations. The small nerves show practically no change.

In the stage of desquamation the changes are much more pro-

nounced. There is hyperkeratosis with shedding off of the corneous layer of the epiderma; the inner epithelium layers contain much yellow pigment. The papillæ contain numerous lymphocytes and plasma-cells, with quite a remarkable absence of mast-cells in all cases that I myself have examined. The sweat glands are hypertrophied, and the sebaceous glands are dilated and often contain bacteria. The skin is thickened as a result of the increased blood supply, and from the presence of swollen, degenerated, elastic fibers and the peculiar hyalin albuminous substance already referred to.

Where ulceration occurs there is complete absence of the epithelium layer, with more or less loss of substance on the surface of the derma. The elastic and connective tissues that form the bulk of the latter structure undergo degenerative changes on the surface, and there are found in the diseased structures polymorphonuclear leucocytes, a considerable serous exudate, and numerous bacteria. Somewhat lower down plasma and lymphoid cells are quite numerous.

Tongue—The tongue undergoes marked changes in pellagra. In the earlier stages the epithelium shows much the same alteration that is observed in the epiderm in the affected areas on the skin. At a later time the epithelial cells shed off around the edges of the tongue, and this may progress until the entire structure appears bare, but a thin epithelium layer may be still demonstrated by means of the microscope. Numerous deep furrows often appear on the back of the tongue as the disease progresses, and its tissues become red. Ulceration is then apt to occur,—first around the edges of the tongue, and in some cases at a later time on any part of its surface. Microscopically these changes are practically similar to those occurring in the skin.

Cheeks and Gums—Similar alterations are found on the gums and on the buccal mucous membrane, and at certain stages, in many cases in the pharynx. As the disease progresses the back of the pharynx assumes a deep red color, sharply circumscribed and symmetrical, this discoloration may be seen advancing forward over the surface of the soft palate in some instances.

Stomach—The mucosa of the stomach is often found pale, and its walls dilated as a consequence of atrophy of its muscular coat; in some cases its surface is quite red in the pyloric region.

Intestines—Similar alterations are found in the intestines. Anemia or hyperemia are particularly frequent in the jejunum, and ulcers are apt to occur in this situation, and even more often in

the ileum. Similar lesions are occasionally found in the large intestine. Not uncommonly the walls of the gut are thinned. The alteration last mentioned is of some historic interest, as the Italian Labus claimed in 1846 that it was characteristic, and it was only after this view had been contradicted by Morelli, and later by a commission appointed by the Congress of Geneva, in 1847, that it was finally established that this lesion is inconstant, and by no means the anatomical criterion of pellagra as had been claimed.

Mesenteric Glands—These structures have been occasionally found hypertrophied.

Spleen—The spleen is usually diminished, occasionally hypertrophied. Metastic foci are sometimes found in this organ.

Pancreas—Occasionally this organ has been found atrophied.

Liver—The liver is usually atrophied, though it is sometimes increased in size. In five of my post-mortems the organ weighed only once as much as 1010 grams. Cirrhosis is now and then found. Microscopically the cells of the peripheral portion of lobules are frequently quite fatty, though this alteration is so frequently observed under other circumstances that it scarcely has any significance. The central vein of the lobule has been oftentimes found dilated.

Kidneys—The kidneys are usually decreased in size, but are frequently found to be normal. Cysts in the cortical portions are frequent as a consequence of the scar formation in these viscera. Microscopically they often show the characteristic changes of interstitial nephritis, with which all are familiar, and which therefore it is unnecessary to detail here. In some instances the only alteration has been found to be fatty changes in the epithelial lining of the tubules.

Adrenals—The adrenals are normal.

Lungs—It is rather curious that tuberculosis is rarely found in the lungs of the pellagrous, but hyperemia, œdemia and emphysema are occasionally encountered, and pleurisy with effusion is not unknown; all of these changes are evidently in the nature of complications.

Heart—The heart is often slightly atrophied, and has been found fatty and the fibers pigmented in some cases; this is evidently secondary, and of no significance. The pericardium is usually normal.

Oseous System—The bones are often friable.

Musculature—The muscles are usually atrophied, but are sometimes normal.

Brain—Writers, such as Strambio, Fanzago, Leghano, Verga, Labus, Mardi, Carraro, Fantometti, Rizzi, Gorno, Girelli, Biscia, Frank, Bayle, Lallemand, Meckel, particularly Roussel, and others, seem without exception to have found nothing further wrong with the brain than more or less œdema of the pia-arachnoid, along with thickening, or thinning, or adhesions in some cases. In 113 post-mortems Lombroso found, in addition to the change first mentioned, atrophy of the brain in 11 cases with occasional hardening of its tissues; in 18 out of 28 cases the brain weighed less than normal, but, on the other hand, was increased in this particular in 7 instances.

By far the most interesting communication that had up to that time been made on the changes in the brain was an article by Babes and Sion in 1899. They showed the presence in the nerve cells, particularly in the large chromophilic cells in the cortex, the presence of unmistakable degenerative changes. The tigroid bodies lose their power of staining with basic dyes, and the cell becomes swollen and vacuolated. The nuclei are frequently pushed to one side and lose their power of taking basic stains, and present swollen nucleoli; the pigment in these cells is also dislocated, and instead of being around the nucleus lies scattered throughout the cell-body. The processes of the cells often appear to be broken off, and seem swollen. The pericellular lymph-spaces are dilated, and the walls of these cavities are frequently lined by yellow pigment. In the brain tissue small collections of lymphoid cells are frequently encountered, and the neuroglia cells in the vicinity of the blood vessels are swollen. These alterations have been in the main confirmed by Marinesco, Rossi, Richette and Grimaldi, and by myself in this country, and are of great interest and importance. In addition to the changes mentioned, I would remark that in all of my cases the small vessels of the brain seemed unusually filled with blood, and the privascular lymph spaces are quite uniformly dilated. There were no collections of lymphoid cells anywhere in the tissues. The nerve cells showing degenerative changes usually measured less than the normal ones, and always contain a greater or less amount of acidophilic protoplasm. The cells suffer to a varying degree in different parts of the brain. Perhon and Papinian have demonstrated, as might have been expected, that the neuro fibrils of the cells show degenerative changes.

Alterations similar to those found in the cerebrum have been observed by me in the nerve cells of Purkinje. The cell protoplasm loses its affinity for basic stains, and the nucleus undergoes a sim-

ilar change. The cells contain a finely granular protoplasm that takes acid stains, and in many of the cells only the nucleolus retains its power of absorbing basic dyes. The nucleus does not seem to be dislocated in these cells so often as in the pyramidal cells. In all of my cases it was quite evident that many cells of Purkinje had undergone degeneration and entirely disappeared. In one instance this change was most striking; in this case the molecular and granule layers were in many places separated by microscopic spaces that probably existed during life, and probably have the same significance as the dilated lymph spaces. These cerebeller alterations probably explain the ataxic forms of the malady. So far as I am aware no one has previously observed these alterations in the cerebellum.

Spinal Cord—Changes in the nerve cells of the spinal cord are practically in every way identical with those occurring in the brain; they were well described by Babes and Sion in the paper already referred to, and have since been confirmed by all who have studied the subject. They show every stage of degeneration from slight loss of chromophilic substance to practical destruction of the cell-body. In 1883 and 1884 Tonnini described degenerative changes in the lateral columns of the cord, and somewhat later, in 1890, Belmondo demonstrated the frequent occurrences of corpora amylacæ in both the gray and white substance, and the presence of changes leading to obliteration of the central canal. Along with this there was increase in the neuroglia fibers, and atrophy of many of the nerves. In 1893 Tuczec published an excellent monograph on the histology of the central nervous system in this disease. In eight necropsies he found combined scleroses of the posterior and postero-lateral columns of the cord six times, and in two instances a similar change in the lateral columns alone; he confirmed the frequency of the obliteration of the central canal described by Belmondo. He also called attention to the curious fact that portions of the gray substance in the cord are frequently found apparently detached from their normal situations, and even lying as isolated bodies in the surrounding white substance; under such circumstances the cord is more or less deformed in appearance. In four out of five post-mortems I have found similar scleroses, and in every instance pronounced changes in the ganglin cells similar to those described by Babes and Sion. I have also in four out of five cases found the central canal obliterated, though in no instance throughout its entire course; the changes are most pronounced in the lower cervical and dorsal regions.

In one instance I have encountered a typical "meningo-myelitis acuta," such as described by Belmondo. Although the post-mortem was made only a few hours after death, the cord was very soft and on miscroscopic examination was found to present alterations of a most pronounced kind. The myelin sheathes of the nerve fibers showed marked degenerative changes, and the nerve cells of the gray substance exhibited to a high degree the alterations already described. Corpora amyalacea were specially abundant throughout both the gray and white substances.

Spinal and Sympathetic Ganglia—The ganglion cells of these structures exhibit changes similar to those found in the central nervous system, though they are not as a rule so marked.

Eyes—From the results of the work of Beitti it is not improbable that circulatory changes are frequent in the central nervous system in pellagra, as he has shown that in quite a proportion of the cases the retina is ænemic, and in about the same percentage the opposite state of hyperemia occurs.

Peripheral Nerves—It has been claimed by some that the alterations have been found in the peripheral nerves, but this remains without confirmation.

PELLAGRA—ITS ETIOLOGY, PATHOLOGY, DIAGNOSIS AND TREATMENT

C. W. G. ROHRER, M. D.

Medical Assistant to the State Board of Health of Maryland.

Baltimore, Md., October 29, 1909.

Definition—Pellagra is a specific infectious disease, due to a parasitic fungus, namely, the *aspergillus fumigatus.* Locally, pellagra is characterized by an erythematous, desquamative inflammation of the skin; and, generally, the disease is characterized by the formation of tumor-like masses or granulomata in various parts of the body. Constitutionally, the disease is complicated by digestive and neurotic derangement.

The disease may be either acute or chronic. A majority of the cases, however, are of chronic duration.

In the State of Maryland but three cases, two by Dr. Wm. S. Thayer, of the Johns Hopkins University, and one by the present writer, have been reported up to the present time.

Classification—Pellagra should be classified along with tubercle, lupus, syphilis, glanders and farcy, leprosy, actinomycosis and rhinoscleroma, as one of the infective granulomata. The abundant presence of tumor-like bodies, especially in the kidneys, lungs, brain, stomach, intestines and spleen, makes the present writer feel justified in vouchsafing this assertion. The superficial portions of the lungs and brain, especially the walls of the blood-vessels, show them most abundantly. These small, nodular lesions, when examined by the microscope, resemble very closely a tubercle with the giant-cells removed from its centre, or a syphilitic gumma with the giant-cells eliminated from its periphery.

Occurrence in Animals—Recently M. Müller, in the *Journal of Comparative Pathology and Therapeutics,* described several outbreaks of an enzoötic character occurring in lower Alsace-Lorraine, and affecting horses, cattle and sheep. The disease, it is stated, was caused by feeding with musty fodder. The principal symptoms consisted in myopathic paresis or paralysis, and in the oxen and sheep in excessive salivation. The pulse and respirations were only increased in old-standing cases. There was no fever and the sen-

sorium did not appear to be affected. Occasionally animals recovered after a very long period of convalescence.

The so-called cornstalk disease, prevalent among cattle in the West, is probably allied to pellagra. "Blind staggers," and many doubtful cases of cerebro-spinal meningitis in horses, are believed by the present writer to be due to pellagra.

The disease has been produced experimentally in dogs and in chickens.

Etiology—The causes of pellagra are predisposing and exciting. The principal predisposing cause is the eating of improperly cured maize or Indian corn. Chronic diarrhœal and dysenteric disorders, especially of an ulcerative type, have also been recently advanced as possible predisposing causes. Impure drinking water, it is alleged, may bring about these intestinal lesions, and thus open up avenues of entrance for infection with pellagra.

The use of corn products as a predisposing cause was well established in the fatal case which this writer lately studied. After the disease had become plainly evident, the patient stated that she could no longer eat corn-bread, because it "went right through her."

As no ulcers were found at autopsy in either the stomach or in the intestines, this writer believes their presence to be accidental or due to other causes.

The exciting cause of pellagra was formerly believed to be a parasitic fungus, *sporisorium maidis,* which produced a diseased condition of Indian corn. Today it is pretty generally conceded that pellagra is caused by the *aspergillus fumigatus.* Some of the fall cases may be due to the *aspergillus flavescens.*

Several observers have found bacteria in the circulating blood of pellagrous patients, and ascribed to them a possible etiological significance. During life no bacteria were found in the circulating blood of the one fatal case recently studied by the present writer. After death numerous bacterial emboli were found in the various viscera and organs, notably in the liver and in the lungs. These bacteria were looked upon as secondary invaders, and not as causative factors.

According to observations made by the present writer, the *aspergillus fumigatus* grows not only upon corn, but also upon peas, beans, and other podded or leguminous plants. It also grows upon buckwheat beaten to the ground by heavy rainstorms.

During a wet season leguminous plants, such as peas and beans, are liable to rest upon the damp ground and thus form a nidus or

hotbed for the growth of the *aspergillus fumigatus*. Buckwheat is sometimes similarly affected. An ear of corn, however, is the most frequent habitat of the now conspicuous *aspergillus fumigatus*. The warmth and moisture generated by an ear of corn during an unusually wet season, or within the husk of an ear of corn harvested a little too green, furnish ideal conditions for the propagation and growth of the *aspergillus fumigatus*.

Another factor hitherto overlooked in the causation of pellagra is the harvesting of corn by machinery. The present writer believes the corn harvester has had much to do with the fairly wide prevalence of pellagra in recent years. To this new departure in agricultural methods should be attributed at least two-thirds of the cases of pellagra. The "corn harvester," therefore, can be classified as one of the "predisposing causes" of pellagra.

Corn cut with a harvester is usually bound tightly into small bundles, and hence cannot properly dry and cure. Occasionally it lies upon the damp ground several days before it is placed in the shock. Harvested while damp or a little too green also helps engender conditions favorable for the growth of the *aspergillus fumigatus*.

Corn harvested in the old way by hand is not bound into bundles, and is placed more loosely in the shock. The atmospheric air, as well as the heat and light of the sun, has free access to the ripening ears of corn, and as a rule they cure properly.

Pathology—As has already been stated, the present writer believes that pellagra should be classified with the infective granulomata. When examined by the microscope these small tumor-like nodules or granulomata are seen to be composed of a necrotic centre surrounded by spindle-shaped cells and small round cells, very much like a tubercle or a gumma minus giant-cells.

These granulomata are most abundant in the walls of the superficial blood-vessels of the lungs, especially those of the upper lobe of the right lung. Next to the lungs rank the superficial blood-vessels of the brain, thus furnishing a pathological basis for the distressing mental symptoms apparent in the last stages of the disease. Granulomata are also found in the stomach, intestines, spleen, kidney and uterus.

Another feature is the intense congestion noticeable in practically all the tissues and organs. An extreme degree of gastroptosis and enteroptosis was also pointed out at the autopsy. The liver showed chronic passive congestion, with numerous bacterial emboli in its

smaller vessels. The stomach at its lower fourth is encircled by many varicose veins. Attached to several of these veins is a polypoid growth. The kidneys contain several small cysts, and show a condition of chronic diffuse nephritis. Numerous necrotic areas are also seen. The cortex is narrow, measuring but one-eighth of an inch. Numerous granulomatous masses are found in the walls of the uterus. There is a mucopurulent discharge from the internal os. The fundus of the uterus is filled with blood and pus. Hence it would seem that the toxic products elaborated during the growth of the *aspergillus fumigatus* have a special predilection for the female uterus, similarly to those generated by the fungus causing ergot of rye, namely, *claviceps purpurea.* The heart weighed but 5½ ounces, and its interior was filled with white clot. The proportion of the weight of the heart to the weight of the body was as 1 to 233, the patient having been reduced to eighty pounds in weight. The heart also showed the condition known to the Germans as "drop heart." With the exception of slight degeneration of the posterior columns of the cervical portion, the spinal cord shows no conspicuous change. The blood-vessels of the brain contain numerous granuloma-like masses. The brain weighed nearly 44 ounces. The skin shows degeneration of its epidermic layers. The deeper of these have been transformed into keratin; the superficial layers have become gangrenous. The cutis vera or true skin shows little or no involvement.

Diagnosis—The diagnosis of pellagra rests upon the history of the case and the characteristic symptomatology. The one fatal case which eventually came under my observation had suffered many diagnoses and much treatment, all, however, of no avail. The last diagnosis made before my advent into the case was "chronic eczema."

A history of the use of corn products as a staple article of diet is very suggestive. My fatal case, as her family informed me, had eaten corn-bread every day of her life, both summer and winter.

The characteristic triad of symptoms are:

1. Gastro-intestinal.
2. Erythematous.
3. Nervous.

The gastro-intestinal derangement is usually the initial symptom of pellagra. Dyspepsia, diarrhœa and stomatitis are the cardinal symptoms referable to the alimentary canal. My patient had complained greatly of her stomach for three or four years prior to the

appearance of the skin lesions. She always drank hot water before meals—the conventional "hot water cure for dyspepsia." About the same time she had to desist from eating corn-bread, owing to an obstinate diarrhœa which it caused.

In my patient the skin symptoms began with unfailing regularity toward the last of April. The skin lesions were first apparent in April, 1907. They began as "small, broad, scattered bumps." The patient and family thought it was poison oak. One of the neighbors also diagnosed it as such and prescribed a soda water wash; later, she prescribed local applications of hog's lard. The skin lesions first appeared on the dorsal surfaces of both wrists. About a week afterwards these areas "turned red as flannel." From the wrists the erythema extended to the backs of the hands and fingers. About two weeks after the disease had become apparent upon the wrists the feet were affected. The dorsal surfaces of both feet were first attacked, and then the ankles. Red spots also appeared upon both knees. Several days before the feet became affected, the classical red band appeared upon the forehead. The erythema, characterized by its fiery redness, always appeared toward the end of April, and lasted from three to four weeks. At the expiration of that time it disappeared entirely.

The second attack, occurring in April, 1908, was more severe and prolonged. The erythema again entirely disappeared in about four or five weeks. In April of the present year (1909) the disease recurred for a third time. This third and last attack was more severe than either of the others. The erythema faded slightly, but symmetrical gangrene of both feet resulted. She grew gradually worse and died August 20th, 1909. The nervous symptoms were not markedly manifest until a few weeks prior to my patient's death. She was delirious at times, and always thought somebody was going to kill her. My patient did not have any convulsions, but at times was in a stupor. My patient suffered but little pain. The skin from the diseased areas could be cut or torn without entailing any suffering. She was very sensitive, however, to heat and cold. For about a year and a half, so the members of her family stated, she was getting thin in flesh, hollow-eyed, and of a swarthy complexion.

Treatment—The treatment of pellagra, like that of other infectious diseases, may be either prophylactic or curative. Prophylaxis, however, is the more important. Right here, and in the following words, I wish to sound the keynote of this Conference, *upon proper prophylactic measures depends the suppression of pellagra.*

The essential prophylactic step can be summed up in the one terse sentence—discontinue the use of spoiled maize or Indian corn. It were better perhaps to institute measures to prevent corn from becoming musty or spoiled. The proper curing of corn is a *sine qua non* in the prevention of pellagra. This can scarcely be hoped where corn is cut by machinery. Leguminous plants and buckwheat, which have lain upon the damp ground, should not be used for human food.

The curative or medical treatment of pellagra is notoriously unsatisfactory. In brief, it is a feeble attempt to combat the symptoms as they arise. The question of diet is all-important. Avoidance of the suspected cereal and the substitution of other good, easily assimilable articles of food is the one thing needful. Among drugs ferruginous tonics and arsenical preparations are indicated. The stock diarrhœal mixtures are advocated for the gastro-intestinal irritation. Opiates may be called for, if the tenesmus be great.

My patient received one-half ounce of whiskey and one-fortieth of a grain of strychnia sulphate every four hours. Her appetite was poor, and about the only thing she relished was ice cream. Five-minim doses of atoxyl were given hypodermically every four hours, but without any appreciable results.

Dr. Wm. S. Thayer, of the Johns Hopkins University, apparently cured one of his cases of pellagra by the use of thyroid extract. He gave three 2-grain doses of desiccated thyroids a day for several weeks, and then decreased the dose to four or five grains daily, continuing the same for several months.

SUMMARY AND CONCLUSIONS.

1. But three cases of pellagra have been reported in Maryland up to the present time. Two of these were reported by Dr. Thayer, and the third by this writer. Thanks are due my able chief, Dr. Marshall Langton Price, Secretary to the State Board of Health of Maryland, for an opportunity to study the prevalence of pellagra in Maryland.

2. Pellagra is an infectious disease, produced by the *aspergillus fumigatus*. The *aspergillus fumigatus* is not very tenacious of life. Not infrequently it is overgrown by the common blue-green mould (penicilium crustaceum), or much less frequently it is overgrown by the common white cottony mold (mucor mucedo); hence the confusion in regard to the etiological factor. The *aspergillus*

flavescens, to which the so-called fall cases of pellagra are attributed, is probably a slightly modified *aspergillus fumigatus.*

3. Personally I believe the appellation "fall attack" of pellagra to be a misnomer. It is merely a recrudescence. In the summer there is a temporary abatement of the symptoms due to the use of fresh vegetables and fruits. Upon the resumption of dry foods in the fall of the year, there is a flaring up of the spring attack.

4. Pathologically, pellagra should be classified with the infective granulomata.

5. The following is additional evidence that pellagra is due to a fungus: The skin lesions in pellagra are similar to those produced by an allied fungus, namely, *claviceps purpurea,* the organism which excites the growth of ergot of rye.

6. It was definitely proven that musty corn was the cause of my fatal case of pellagra. To prevent pellagra, corn-meal should be made from properly cured corn. Also, avoid eating leguminous foods(peas and beans), and buckwheat, the pods of which have lain on the damp ground. The *aspergillus fumigatus* sometimes grows upon these articles of food, but the rôle which they play in the causation of pellagra is a minor one.

7. Meteorological conditions, especially in the great corn belt, have much to do with the prevalence of pellagra. Many cases are apt to follow a wet season, while comparatively few will develop after a dry season. A wet season furnishes ideal conditions for the growth of the *aspergillus fumigatus.* The past season has been a remarkably dry one, hence we need not expect a new outbreak of pellagra for some time to come.

8. The harvesting of corn by machinery, that is, the use of the corn harvester, sometimes called the corn binder, is responsible for the alarming prevalence of pellagra in recent years. The corn is bound into bundles and cannot ripen or mature properly, owing to exclusion of the air and sun; a favorable environment is thus afforded for the growth of the *aspergillus fumigatus.*

PELLAGRA IN YUCATAN

GEO. F. GAUMER, M. D.

IZAMAL, YUCATAN, MEXICO.

Pellagra is a non-contagious, tropho-neurosquamous erythema, due to a specific cause.

History—Although isolated cases of this disease may have existed in Yucatan at an earlier date, yet it was not until 1884 that it became epidemic.

In 1882 the locusts or grasshoppers invaded the State in such numbers that they destroyed every cultivated plant, and were especially destructive to the Indian corn or maize.

Corn being the only cereal used in Yucatan for bread, famine seemed inevitable until the merchants began to import corn from the United States. This importation of corn continued until 1891, when the country had recovered from the devastations of the locusts. The imported corn was brought from New York in the bottoms of vessels as ballast and from careless handling and bad storage it was often rendered unfit for food. During the voyage this corn often got damp and even wet, and by the fermentation caused by heat and humidity a peculiar kind of fungus is developed which has been called "Sporisorium maidis." The constant eating of corn affected with this fungus produces a vitiated state of the blood which leads to the slow development of pellagra.

Among the better classes the disease seldom made its appearance. Whether this was due to the fact that, having the facilities, their food was better cooked, or because, having the means to purchase the little corn produced in the State, they were but small consumers of the imported article, it was the middle and lower classes who, from reduced circumstances, were obliged to purchase the cheapest corn in the market, that suffered most from the ravages of the disease.

While pellagra had been known for many generations in Italy, Germany and other European countries, yet it had probably never before made its appearance as an epidemic in any part of America; and yet, there is no good reason why sporadic cases might not have appeared occasionally wherever corn was used as an article of diet.

From 1891 to 1901 Yucatan produced sufficient corn for home consumption, and new cases of pellagra were no longer to be found,

while the old cases ran their course and nearly all those attacked in former years died from the effects of the disease.

From 1901 to 1907 the corn crops were almost total failures and corn was again imported in greater quantities than ever before. Most of the corn came from the United States, Mobile and New Orleans being the chief sources of supply, the remainder coming via Vera Cruz from the interiors of Mexico, or nearly the same distance, by water.

Pellagra again became epidemic, but was not then confined to the middle and lower classes, as in the former invasion. The wealthy hemp owners, on account of the exorbitant prices paid for hemp, found it was more profitable to import than to raise corn for home consumption, thus compelling even well-to-do people to consume the imported article, as the home product was no longer sufficient for the wealthy families. Pellagra then spread alike among the rich and poor, until, by the close of 1907, about 10 per cent. of the inhabitants were victims of the disease, and at the present writing not less than 8 per cent. of the adult population have pellagra.

Prodromes.—Lassitude, occular phenomena, vertigo, headache, general weakness and occasional digestive disturbances.

FIRST STAGE.

Symptoms—A patient upon first consulting a physician complains of a sensation of heat in the mouth, throat and stomach, upon the exhalation of the breath; taste is impaired, there is anorexia and frequently ptyalism with a broad, flabby tongue irregularly marked by red blotches, a peculiar formication in the extremities which often extends very gradually to the whole of the body. About this time small, smooth and very lustrous specks make their appearance on the dorsal aspect of the hands and feet. These specks, when first noticed by the patient, are no longer than a pin head, but rapidly become more numerous until uniting they form lustrous patches which are checkered off in little squares, and these are separated by fine lines, thus giving the cutis a scaly appearance, which is better seen by drawing the skin together with thumb and finger. This often covers the whole of the body, but is generally confined to the extremities, chest and back. The skin takes on a senile appearance and the itching becomes almost intolerable, and if scratching be resorted to for relief the burning that follows is unendurable.

About this time the strength begins to fail, and the patient walks with a heaviness and a peculiarity in his step that he does not under-

stand. He can no longer find his way with closed eyes. The reflexes become greatly exaggerated and his movements are incoordinate. His sleep is much disturbed by hallucinations and strange dreams. During his waking hours he examines himself minutely and with frequency, and soon begins to carry on a constant, though inaudible, conversation with himself or some imaginary companion.

In conversation with other persons it soon becomes evident that the mind wanders. Before completing one subject another is begun. Meaningless words are thrown in and as important ones left out. The patient's description of his disease becomes long and tedious, and he often ascribes as a cause of his present condition some insignificant disease or injury that befell him in his youth, and ends up with the assurance that some person who, knowing of the circumstances, has taken advantage of the opportunity to bewitch him.

SECOND STAGE

As time goes on the physical sufferings become greater and greater, the skin wrinkles more, the appetite fails and this is due to the perversion of the special senses of taste and smell, so that the patient looks upon the most savory dishes, all kinds of drinks and even his medicines as filthy substances calculated to aggravate his sufferings if not to kill him. Hunger increases the mental derangements. Strength fails until the patient is confined to his chair or bed. The mind becomes more deranged as the itching and burning continue to rack his nervous system, so that he often seeks to put an end to his sufferings by committing suicide.

The bowels at first were often constipated, but as the disease advances a diarrhœa sets in, which is accompanied by a progressive emaciation, until the patient is reduced to a mere skeleton, or, in some cases, it passes into a dystentery, which, running a rapid course, soon terminates fatally.

THIRD STAGE.

Inasmuch as pellagra is a disease in which every symptom from its first appearance advances progressively to a fatal termination the third stage is but the first and second in a state of progression in which the mental and physical phenomena predominate. Fear of impending danger often makes the patient want to flee from home and friends, and to take refuge in the forest, but, upon finding himself devoid of strength and the power of locomotion, his fright

increases, and he strives to escape from an imaginary bondage, Friend are no longer trusted, but are treated as enemies.

All former symptoms are progressively intensified until the patient loses control of the mind and complete dementia generally occurs near the termination of the disease.

All psychosis are of the melancholic type and tend to make the patient cowardly, rarely becoming aggressive.

Weeks, months and sometimes years are spent in this stage, during which time the patient gradually grows weaker until he is unable to swallow food or drink, to utter an audible word or to voluntarily move a single muscle; thus the spark of life is slowly but surely extinguished.

FORMS OF PELLAGRA.

In a practice of twenty-five years I have been in the habit of recognizing three forms of disease known and treated here as pellagra. These I have classified according to their cause as pellagra, pseudo-pellagra and pelagia.

Pellagra is a disease whose origin can always be traced to the ingestion of spoiled corn.

Pseudo-pellagra: A disease whose origin can always be traced in the use of alcoholic liquors.

Prior to 1900 nearly all of the liquor consumed in Yucatan was distilled in the State, and came from sugar cane or bee's honey, and up to that date pseudo-pellagra was quite unknown.

Since 1900, for various reasons, Yucatan has ceased to produce the liquors consumed by its inhabitants, and they are now concocted from alcohol distilled from corn in the interior of Mexico, and essential oils skillfully prepared by experts in New York.

Since the artificial drink has been substituted for the pure distillate, pseudo-pellagra and other well marked derangements of the human organism have become quite common.

Pelagia: Has no constitutional symptoms and is purely a local condition due to exposure to the direct rays of the sun and the inclemencies of the weather.

Any two or all three of these conditions may, and often do, exist in a patient at the same time.

In pseudo-pellagra, as in pelagia, removing the cause cures the disease; although in most cases pigmentation remains through life.

Pellagra, in its primary stages, is often curable by the application of the proper remedies, and by leaving off the use of corn. After mental phenomena have made their appearance the disease becomes

incurable; although by judicious treatment the course of the disease may be retarded and life prolonged.

In Yucatan pellagra is not influenced by reasons as indicated by Dr. Lavinder in his "Pellagra a Precis," nor have I ever been able to detect any increase of temperature in connection with the various stages of pellagra. Ulcers and ulcerative processes form no part of pellagra, but when they do occur as complications they are very obstinate and difficult to cure.

Desquamation never takes place in the earlier stages of pellagra, but in case of long standing the constant scratching causes the cuticle to break up in the form of small, white, thin scales, which are thrown off in considerable numbers. The Indian doctor makes his diagnosis by drawing his fingernail rapidly across the affected skin. If a white line of scales is left in its wake, the disease is pellagra.

TREATMENT.

The first thing to be done is to put the system in the very best possible condition for the assimilation of food and for the elimination of the disease. This must be done according to special indications in each individual case, but the remedies that have given the best results are rauwolfia heterophila in small doses long continued. With the fluid extract of this plant many recent cases can be cured, and with it the disease can be very much retarded in all cases.

Arsenite of patassium alone, or in combination with the rauwolfia is a valuable remedy in many cases. Opium may be used to control the diarrhœa, though it is liable to aggravate the nervous symptoms, in which case the fluid extract of coccolobamiferi, being an astringent tonic, is to be preferred in all such cases, and is much more reliable.

As special tonics aristolochia lireoipes, salvia serotina and hydrastis canadensis are to be preferred.

The severer nervous symptoms are controlled by salanun torvum and piscidia erythrina. Insomnia finds a remedy in passiflora ciliata and incarnator.

Edema and heart troubles may be best controlled by the use of Cereus zrandiftorus.

The fluid extract of the above remedies are to be preferred.

In individual cases, and when indicated, good results may be obtained from the use of sulphide of calcium, carbonate of lithia, arsenite of copper, tincture of the chlorid of iron, acid solution of

iron, sulphate of strychnia, thuja occidentalis, echinacæ, tagetes patrela, phytolacca mexicana and quinin.

Many other remedies may be used to meet special indications.

As external remedies few have any effect, but the best results have been obtained from the use of the sulphide of soda in baths, and ointments of arsenic, carbolic acid or tagetes patula will often remove all external signs of the disease.

The hygienic measures to be employed are frequently bathing with water at a temperature most agreeable to the patient. Exercise in the open air with change of scenery and surroundings, and frequent changes of clothing. Leave off the use of corn as an article of diet, and select a healthful and nutritious diet of meats, fresh vegetables and fruits.

DIFFERENTIAL DIAGNOSIS.

PELLAGRA.	PSEUDO-PELLAGRA.	PELAGIA.
In the beginning of the disease patient complains of a sensation of heat in the mouth, throat and stomach on expiration.	Normal.	Normal.
Sense of taste impaired; there is anorexia and ptyalism.	Normal. Sometimes present.	Normal.
Tongue broad, flabby and irregularly marked by red blotches.	Uniformly red.	Normal.
Bowels constipated, followed by diarrhœa and sometimes dysentery.	Diarrhœa sometimes present.	Normal.
Cuticle assumes a scaly appearance; scales lustrous, thin and not detachable until disease is far advanced—seldom pigmented; only affects the cuticle.	Dorsal aspect of all affected parts become erythematous, assumes a dark color and are pigmented progressively; scales thick and detachable—epidermis and part of true skin affected.	All exposed parts become erythematous, assume a dark color and covered by large, thick laminated scales, detachable. Skin either œdematous or hypertrophied.
Pruritus and burning deep-seated, aggravated by scratching. Affected differently by sun and shade.	Superficial, aggravated by sun's rays.	Slight, but the burning is intensely aggravated by sun's rays.
Muscular weakness marked and progressive.	The same.	Normal.

PELLAGRA.	PSEUDO-PELLAGRA.	PELAGIA.
Vertigo, occipital headache, insomnia, neuralgias and cramps.	If present can be traced to alcohol.	Normal.
Occular phenomena generally present.	If present alcoholic.	Normal.
The gait is usually paralytic, occasionally paralytic spastic and progressively ataxic.	Usual symptoms due to alcohol.	Normal.
Mental phenomina progressive from slightest perturbation to complete dementia.	Alcoholic if any.	Normal.
The pellagrin avoids company, seeks solitude, is distrustful, melancholic, avoids conversation, prefers the dark, wants no assistance.	Seeks company, avoids solitude, is confiding, is cheerful and talkative. Avoids the dark, wants help.	Normal. Indifferent.
Epileptiform movements rythmic and often continued to death.	Not rythmic.	No movements.
Pellagra is not limited to season, age, sex nor condition in life.	Limited to alcohol users.	To persons past middle life, who have been much exposed to sun rays.
Attributable to the use of spoiled corn.	To the use of alcohol.	To exposers to the sun's rays.
The pellagrin does not fear death, generally unconscious at death.	Fears death and is generally conscious at death.	Indifferent to death.

PELLAGRA IN JAMAICA

D. J. WILLIAMS, M. D.

Medical Superintendent, The Asylum

KINGSTON, JAMAICA.

The existence of pellagra was recognized in our wards about 12 years ago, but as then it was unknown in the West Indies, the correctness of the diagnosis was questioned, and the erythematous condition of the exposed limbs attributed to "sun-burn."

The condition always appearing in the feeble, listless and anergic inmates, it was believed an error in diagnosis was made, but as time went on and similar objective signs of disease appeared in cases not exposed to the direct rays of the sun, we were forced to admit the former diagnosis of pellagra was correct, and this has been confirmed in more recent times.

Four or five years ago, the disease was very prevalent in our wards; four per cent. of the inmates—male and female, in about equal numbers—were attacked, in a population of 1,050. With generous diet, rest in bed and tonics the majority improved temporarily, others made no improvement, but suffered from chronic diarrhœa, progressive weakness and emaciation until death ended the scene.

Owing to the mental obfuscation of these inmates it was impossible to obtain a reliable statement of their subjective symptoms, one patient only—a chronic maniac—complained of intense pain in the erythematous patches on the exposed limbs and nape of the neck. She recovered shortly afterwards and hitherto there has been no recurrence of symptoms.

With the object of ascertaining the truth that damaged or diseased maize was directly responsible for the disease, I excluded corn-meal and every form of corn from the inmates' diet for twelve months, but as I found that cases admitted into our wards after the exclusion of the corn-meal from the diet suffered from the disease, I am not prepared to admit that maize or Indian corn is the only cause of this condition; in fact, I wish to record my opinion that Indian corn—damaged or otherwise—is not the sole cause of pellagra. On the strength of this opinion I have again restored corn-meal as an article of diet here.

Dr. D. M. Sandwith, of London, made the following remarks on last year's report of this asylum:

"The severe drought and shortage of food stuffs mentioned on page 14 are important because they are likely to cause an increase of pellagra, as has happened under similar circumstances in Italy, Egypt and elsewhere.

"The measure mentioned on page 49 of substituting bread for maize rations is likely to be useful in treating pellagrous patients, provided maize flour is not used in making the bread.

"Atoxyl or Soamin should be employed in treating pellagra, as is now being done in Roumania, Egypt and the Southern United States, where the disease has lately been discovered.

"It is possible that some of the deaths reported on page 55 as being due to chronic enteritis and dysentery are really caused by pellagra, which is by no means confined to lunatic asylums."

Such, in a few words, is the history of pellagra in Jamaica, or rather in the Jamaica Lunatic Asylum, for I am not aware the disease has been met with elsewhere in the island.

October 21, 1909.

PSILOSIS PIGMENTOSA IN BARBADOS

C. G. MANNING, M. R. C. S. ENG., L. R. C. P.

Medical Superintendent Asylum

BRIDGETOWN, BARBADOS.

We have had a disease here for the last 15 years, or thereabouts, which the general run of medical men in the Colony call pellagra. I do not know what has cropped up in South Carolina, but I am sure that what we have here is not pellagra, and my reasons for saying that are the following:

I. Pellagra is said to be caused by ergotized maize or some other disease in the food we eat. If that proposition is to stand, we would naturally conclude that in an asylum like the one I am in charge of (400 beds) where the maize and other food is cooked in the same kitchen and served alike to patients and attendants, this disease would spread alike to attendants and patients.

Now, as a matter of fact, we have never had an attendant have this disease, and I will add we have never seen it in patients of the better class, patients, in short, who take a pride in bathing frequently and in keeping themselves clean.

II. The dark discoloration and squamous appearance of the skin does not appear over face, chest and back, nor is it a true pigmentation in any sense of the term.

It is scurvy crust which appears in ninety-nine cases out of a hundred on the elbows, knees, ankles, hands and feet, but specially on point of elbows and knuckles; very rarely on the chest, back, etc.

III. It often appears and disappears without leaving a mark, and reappears again at varying distances of time, but it is not a true pigmentation, white like a tattoo mark, and never disappears.

The disease (which we have in Barbados, which I have named Psilosis Pigmentosa), has the following characteristics: It is essentially a disease of poverty, hunger and dirt. The patient first complains of disinclination to take his food. Soon he positively refuses solid food or highly seasoned food of any kind. On examining the mouth, tongue and pharynx the cause is quite apparent. The tongue will be found stripped of its epithelial covering. At first this is most apparent at the tip and along the edges. It will be found that mucous membrane of the cheeks and fauces looks red and irritated. Patients prefer milk and this is the best nourishment for them,

because it is the least irritating and most nutritious. Very soon attacks of diarrhœa set in and often prove intractible. Scurvy patches of dark color will be found over the points of the elbow, knees and knuckles. These generally are the localities first attacked; later on the feet and hands become affected, and if the patient wears slippers or shoes the part of the foot which is covered is never attacked. The patient rapidly loses flesh and this is followed by intense anæmia, the brain, like the other organs of the body, is badly nourished and cerebral anæmia causes the patient to become silly and half-witted, sometimes mouthing and garrulous, and it is on account of these cerebral symptoms that the patients are certified as insane and dumped down in our asylum to save further trouble on the part of the parochial authorities. Under regular feeding and steady treatment these patients do wonders, the scurvy patches disappear and as the patients become less anæmic and so the cerebral symptoms clear up and they become perfectly sane and rational. They often go on quite well for years, but there is a tendency for attacks to recur, and again with suitable treatment all the symptoms clear up and the patients appear perfectly well and will put on flesh. But the disease does not always pursue this satisfactory course. If the patient is admitted after the above preliminary symptoms have become chronic and have not been treated, the disease pursues its headlong course and defies all the skill and attention that we are capable of affording. The diarrhœa becomes intractable and frequent vomiting is a most troublesome symptom, sometimes the ejecta from the stomach contains streaks of blood, sometimes clots are thrown up. The diarrhœa is also blood stained and in one case I have seen true melana, dark tarry stools passed before the patient's death. The smell of the evacuation is overpowering and there is a sickening odor never to be forgotten. The patient goes to a skeleton, suffers intense pain right through the alimentary canal from the mouth to the anus, and dies simply a skeleton with the skin stretched over it.

The patches on ankles, dorsum of feet, etc., if the patient is white, soon have a purple blotched appearance, and, in the report which I wrote for our Government, I described these as wine-stained patches, because they presented the exact appearance of having been steeped in claret. They are indeed a kind of purpura, they form blebs, break down and the epithelium strips off, and this is why I call the disease psilosis, from bare, "stripped." Gangrene invades the discolored area, and the cutis vera is the next to separate and come away "in

strips." The muscles follow suit and even the tendons hang in shreds about the parts that break down so rapidly. The buttocks soon have discolored areas, over the trechanters, sacrum, etc., and after a deep burrowing gangrene sets in over the glutei muscles, horribly offensive sloughs separate bit by bit and the patient sinks exhausted, a miserable and pitiable object, and death is indeed a welcome visitor.

Follow this body into the dead house and what do we find?

Not a particle of fat in the localities where it is usually found in fair quantity, even in abundance, the knife goes through the thoracic walls exactly as it would through parchment. The muscular walls are atrophied beyond recognition.

There is not a drop of blood anywhere to be seen, the lungs are ashen gray, almost white, contain a little frothy mucus and there might be a little serous fluid in the pleura, but no blood. The heart is pale, flabby and contains a little straw-colored serum in the pericordial pouch; the valves are probably in fair working order, the muscular walls thin and pale; if there is any blood found in it, it appears in the shape of a straw-colored gelatinous mass, showing that death was slow and tedious.

The intestines are empty and all of the organs of the abdomen testify to the absence of red blood cells. The stomach is studded over with light red petechiæ and they are no doubt the cause of the blood-stained vomit and the melona, and there has been a slow and steady depletion going on, such as occurs in cases of anchylestomosis. There is often a thick, ropy mucus covering the stomach and can be scraped off with the knife, leaving the potechiæ bare and red; sometimes these are stellate in appearance from the congestion of the vessels leading to these petechiæ.

The intestines are empty, bloodless, and now and then have a bluish appearance on slicing them open with a scissors: the muscular and mucus coats are gone and long strips, sometimes two or three inches long, appear in the alvine evacuation. If one is put under the tap and tries to wash them they burst here, there and everywhere, the erosion of the walls leave very little to maintain their continuity. The large intestines often have the appearance of a gut attacked by dysentery. There are ulcerous patches and congested areas quite sufficient to account for the bloody appearance of the stools. The kidneys are congested and often have a little blood in the caluces and sometimes a drop or two of pus.

The spleen is small and atrophied, the pancreas is fairly normal in

appearance. The liver ænemic and of a pale nutmeg color and appearance, due, no doubt, to anemia.

The brain is absolutely bloodless, with slight excess of fluid in the ventricles and hollow places.

Treatment—When I first took charge of the asylum in this Colony I must confess that there appeared very little hope of doing anything for these poor sufferers, but as I went on patiently studying the progress of the disease to its ultimate and fatal end I came to the conclusion that the quality of the food had nothing whatever to do with causing it. I soon began strongly to suspect that it might be caused by insufficient quantity, for all the cases I have seen have been absolute paupers, poverty-stricken and dirty in the last degree, and also by being badly cooked, as this Colony is bare of forests and fire wood is exceedingly scarce all over the island.

I am of the opinion that it is due to a fungus, and as soon as I discovered that patients began to be attacked with this disease who had been in the asylum, in some cases for thirty years, and who suddenly contracted this disease, and died from it, I thought that this fungus was not transmitted in the food, but in the clothing. It occurred to me that I would boil foul linen and clothing used and worn by these cases, and there has been a complete arrest of the spread of the disease to the old residents in the asylum, and, furthermore, there has been a marked amelioration in the symptoms of those admitted with the disease since they have been frequently bathed and supplied with clean and sterilized clothing, and, indeed, so hopeful am I in the disease that I am now writing a second report on this disease for our Government.

Pointing out the success of my efforts in this direction, I was first induced to try this method by observing that without a single exception those cases with discolored patches on their feet always stop short and exactly corresponding to the part of the foot covered by a shoe or slipper.

Internal Treatment—I have found nothing to compare with 1-40 grain bichloride of mercury three times a day and long continued. And milk and nothing else but milk. When they begin to recover, Scott's Emulsion has given good results, but it must be started with small doses and gradually increased and, of course, it is discontinued if it upsets the stomach.

From the success attending my efforts in our asylum I am strongly of the opinion that this disease, if properly handled in the earlier stages, will in course of time become a matter of history.

I trust that these notes, hurriedly strung together, might be of some service to you, and if they appear in your transactions, would it be asking too much to request you to send me a copy?

I should have been pleased to have sent you some water-color drawings of these morbid appearances in this disease, but I have had no time to do so as your meeting is booked for the 3rd and 4th of November.

PELLAGROUS INSANITY AMONG THE ARABS IN EGYPT.

DR. A. MARIE

VILLEJUIF, PARIS, FRANCE.

Pellagrous insanity is becoming almost unknown in France. It does manifest itself there, however, in the form of hereditary insanity, as the recent investigation of Regis has shown. The asylums of Pan and Montpellier are almost the only ones where this affection persists in the statistical tables. It is because maidism (pellagra) is only possible where maize is eaten. To be poisoned by the ferments of spoiled corn, it is necessary for this cereal to form part of the prevalent dietary, which perhaps with us only exceptionally and in regions remote, isolated and poverty-stricken. Such is not the case in Spain, where maidism persists in certain regions, and affects 20 per cent. of the population.

In Italy, in spite of the valiant struggle led by Lombroso all his life, the regions of Bergamo, Brescia, Venice and Padua still number 30 to 50 pellagrins to 1,000 inhabitants. Treviso, Vicenza, Cromona and Pisa 10 to 20 per 1,000. At Milan I was able several months ago to examine a number of insane patients of this kind. They estimated the total number of pellagrins in all Italy at 72,000.

In the Orient maidism rages in Roumania, Servia, Bosnia, Macedonia, Albania and in Turkish territory as well as in Greece.

The admirable labors of Babes and Marinesco in Roumania upon the pathology of the nervous system from this intoxication have supplemented those of the Italian school to which we owe the measures for social prophylaxis (the law of July 21st, 1902,) originating from the Anti-Pellagrous Congress of Bologna, Padua and Milan.

In Egypt pellagra reigns as in other Turkish countries where corn is largely used. There one may say that almost all the fellaheen are in some degree touched by the pellagrous poison. At the hospital of Kasr-et-Nil, at Cairo, in ten years more than 1,000 pellagrins have been treated. Each year of this number of cases forty are complicated with insanity, and are consequently committed to the asylum of Abbassia, where I was able to study them.

Outside of the hospitals, the study of pellagra in the country population has been undertaken by Dr. Sanwith (Egyptian Congress, page 485). He thinks that on a general average more than 36 per

cent. of the Egyptian peasantry are affected. In districts the least poverty stricken the proportion may fall to 15 per cent., but elsewhere it rises above 62 per cent.

In lower Egypt the average would be highest even with young women. The considerable still-birth rate would not exist but for this plague.

In upper Egypt the greater dryness and the use of millet as food lessens the danger, and probably also the countries remote from the seacoast use less imported maize, which is more dangerous because of the ship's hold and defects of transportation.

At the asylums which we visited the statement of admissions due to pellagra from 1896 to 1906 was:

	Men.	Women.	Total.
1896	9	2	11
1897	10	13	23
1898	10	29	39
1899	19	14	33
1900	8	27	35
1901	27	10	37
1902	23	10	33
1903	41	11	52
1904	38	15	53
1905	54	11	65
1906	46	13	59
			440

The number of pellagrous insane, as it appears, is continually increasing, and only those, however, who clearly bear the physical stigmata are enumerated, but the hereditary pellagrins do not always present these stigmata thus clearly.

The erythema varies in appearance according to the season; although its topography remains the same. From racial characteristics it acquires certain relative pigmentary peculiarities. It is thus that old cicatrices instead of contracting by a darker color as with the white race manifest themselves, on the contrary, by a grey tint clearer in the men of color in consequence of a sqaumous thickening of a thick and dry epidermis.

The climate and the Arabian costume direct also certain peculiarities to the localization of this erythema. It is thus that the short and wide sleeves make the (pellagrous) glove more extended and the

grandoura opened wide upon the breast causes the erythema to extend from the neck to the sternal region.

Upon the legs the boot of erythema often reaches above the knee and the bare foot is attacked just as the hand by the solar localization. One may observe either the increase of pigmentation at the beginning or the final depigmentation by a dystrophic state of the epidermis which becomes like parchment, dry and scaly. Frequent scars from previous ulcerations further alter the appearance of the integument at the most exposed positions.

The period of ulceration is sometimes preceded by a phase of desquamation by shreds of pigmented epidermis which breaks and forms fissures. In the negro, especially upon the face, the eruption can assume the appearance of grains of millet pigmented and hard upon the forehead, the neck, the cheeks and the periphery of the lips.

We shall not dwell either upon the well known physical stigmata of the integument with their seasonal rhythms or upon the dominating visceral troubles which accompany them (diverse gastro-intestinal disorders, gastritis, diarrhœa, etc.).

We shall devote our space to mental and nervous peculiarities, the manifestations of which are generally consecutive upon the preceding signs; although they may precede them or in certain cases be substituted for them.

The mental state of the patients is generally characterized by an initial phase of irritable weakness, by an apathy with physical and mental depression and diverse phobias, sitophobia is frequent and coincides with the gastro-intestinal troubles, gastralgia, cramps, nausea, state, alternating constipation and diarrhœa.

Mutism joins sitophobia, the patients become wild and apprehensive, isolating themselves and seeking dark corners (photophobia and painful cutaneous sensitiveness to heat).

Mental torpor is accompanied by amnesia and spasms, developing even vertigo and epileptiform convulsions or their psychic equivalents. In the first rank of these latter mention should be made of walking automatism, a frequent cause of innocent suicides by precipitation or submersion in the canals of the Nile. Self-accusation or preoccupation with hypochondriacal or nosophobia thoughts are not rarely associated with vague ideas of prosecution. Stupor is mixed with automatic exaltation and diverse flights (fugues) with or without dreamy confusional states.

Sitophobia can arise either from dysphagia or anorexia and apathy may border upon catatonia. It may sometimes give place to sito-

mania in a later stage, for pellagrous insanity frequently assumes the chronic form with or without the addition of remissions. This is understood, for the patients after improving relapse into the same condition as when first attacked as a result of eating spoiled corn. Since the same causes reproduce the same effects, relapses are frequent and also chronic conditions. Besides complications from other factors producing psychoses are not rare.

As an example for Egypt, we may cite the association of the pellagrous intoxication with that of hashish. We may cite the possible combination of diverse infections, of malaria and syphilis in particular, and the parasitism evidence in Egypt of ankylostomiasis which we have found very wide-spread among the Egyptian insane as in the native population in general.

For the aforesaid specific infection, I have already described general paralysis as frequent among the Arabic Egyptians. Does pellagrous pseudo-general-paralysis exist among them, or is it a simple combination of specific general paralysis upon a soil prepared by the pellagrous intoxication?

This question is not new, and it is the French school which solved it during the resounding Academic debates stirred up by Baillarger in 1847.[1]

The examination of pellagrous insane Arabs of the asylum at Abbassia shows that if true pellagra is almost constant among them as among the population from which they come, there are alongside of the pellagrins who have become insane—the insanity being a consequence of the pellagra and in direct casual relation with it. They draw from their pellagrous origin certain typical characters connected with physical stigmata.

A certain number of insane paralytics coincide with pellagra and confirm the opinion of Baillarger that the final paralytic phase of pellagra can represent an identical state clinically and pathologically with general paralysis of the insane. These cases do not invalidate at all the other form of general paralysis from which they are distinct, and which I have described as being in relation to syphilis. The two forms can, however, be combined, that is to say, one may observe among the Arabs general paralytics who are at once both syphilitic and pellagrous.

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[1]The question arose upon a plan of investigation proposed by Roussel to the Minister of Commerce,—the investigation to be made in Spain into the causation and manifestations of pellagra, which was then frequent in Southwest France. Mr. Gilbert, a member of the Academy, expressed an opinion contrary to the theory of its origin from Indian corn, supporting his belief by cases observed by him in Paris at the Hospital St. Louis. It was a question probably of hereditary pellagra analagous to the case reported by Regis in a patient having never eaten corn spoiled or otherwise, but a daughter and granddaughter of true and insane pellagrins.

PELLAGRA IN EGYPT

Hospital for the Insane

ABBASSIA, CAIRO, EGYPT.

The statistics on enclosed sheet show how formidable this disease is as a cause of insanity in this country. There are, doubtless, many thousands of the poorer fellaheen suffering from this disease in the provinces. In the towns where syphilis and hasheesh are rampant and little maize is used, pellagra is uncommon, but its place as a cause of insanity is taken by the above agents, and we have the hasheesh intoxications and general paralysis in considerable numbers.

Pellagra is very common among children in Egypt; apparently this is not your experience in America. I have seen scores of children from 10 to 15 dwarfed, cachectic, anæmic and displaying splendid black pellagrous rashes, and usually insane. The mental symptoms are modified by the age, and the children are mischievous and restless, besides being depressed and deluded. Some of them look like little mummies. I think men suffer more frequently from pellagra than women in Egypt, but this is only an opinion. I have no figures to back it.

In this asylum no maize is used in any form. All flour is carefully analyzed and the diet is kept absolutely free from maize. Yet pellagrous patients resident here for years and thus abstaining from maize for long periods again develop pellagrous rashes here with acute physical symptoms, sometimes resulting in death.

The following are notes of a few of such cases, but scores of similar cases are recorded in our books.

1. I. S. S., Case Book XVI, 27. Admitted 18th November, 1903. In June, 1907, he developed a black charred-looking rash of neck with diarrhœa and all the symptoms of acute typho-pellagra, and died.

2. S. A. H., Case Book XVII, 233. Admitted 14th March, 1908. On 8th May, 1909, he developed a pellagrous rash with diarrhœa and acute prostration and died on 15th May, 1909. (See photographs I and II, taken on May 8, 1909.)

3. M. D., Case Book XVII, 225. Admitted 5th March, 1908. On 3d December, 1908, pellagrous rash reappeared.

It is noteworthy that the rash and the diarrhœa occur simultaneously. The rash is therefore not a casual symptom produced by the

exposure of a pellagrous person to the sun's rays, but a sign of a general degenerative process affecting the whole organism.

Nearly all cases of acute typho-pellagra have a very black indurated rash, and there seems to be a correspondence between the darkness of the rash and the acuteness of the general symptoms.

Nearly every case of pellagra suffers from bilharzia disease. Very many from anchylostoma also, and very many from extreme favus of the scalp. I have always considered that these parasites have fixed themselves upon a decaying organism; but there is something to be said for the possibility that these parasitic diseases account for some of the symptoms of pellagra (anæmic, emaciation, etc.). We should like to know whether in America you find these parasitic diseases so frequently (or almost invariably) associated with pellagra?

STATISTICS.

Egyptian Government Lunatic Asylum, September 25, 1909.

Year.	Annual Admissions of Cases of Pellagrous Insanity.			Annual Deaths from Pellagra in Asylum.		
	Male	Female	Total	Male	Female	Total
1901	23	10	33	6	4	10
1902	22	9	31	6	2	8
1903	41	11	52	9	1	10
1904	38	15	53	3	3	6
1905	54	11	65	13	4	17
1906	91	33	124	9	1	10
1907	69	20	89	7	2	9
1908	71	17	88	6	2	8

The above has been extracted from my annual reports. The great increase in the pellagrous admissions in recent years is due to *more careful examination and search* for pellagrous symptoms. No doubt, nearly as many pellagrous cases were admitted in 1901 as in 1908, but their pellagrous character was not demonstrated. Lately every case has been watched and few escape diagnosis now.

Note—The above figures refer to cases of insanity *due to pellagra,* not to cases showing pellagrous symptoms *merely.* Thus, we admit numbers of congenital imbeciles, precocious dements, epileptics, senile dements, etc., who have signs of pellagra, but the pellagra seems a *coincident* illness, *not* a cause of the insanity, so their diagnosis would appear under other headings.

I send some *old reports.* If you will look up "Pellagra" in their contents you will find many remarks about pellagra in Egypt, which, of course, are at your disposal.

PELLAGRA IN EGYPT

R. G. WHITE, M. B.

Director Serum Institute

ABBASIA, CAIRO, EGYPT.

The following preliminary note, with suggestions as to the means best suited to take up the study of pellagra in Egypt, was submitted to Mr. Graham, Director-General Public Health Department, Cairo, in July, 1909. It was not written for publication, but was read at the conference through the courtesy of Mr. Graham and Dr. White, and is now published with their consent upon special request.

The notes are the result of some observations made during the early months of this year, and are not in any sense intended as an official report upon an important subject now being urged upon the Egyptian Government.

To the Director-General, Department of Public Health.

Sir: I have the honor to submit to you a report of the investigations so far carried out on the occurrence of pellagra among the Fellaheen.

These investigations have been devoted:

1. To a study of the cases as seen at the Lunatic Asylum, Abbasia.
2. To visits to the villages and homes of the patients.
3. To a minute examination of well marked cases in Zagazig, and (latterly) in Benha.
4. To carrying out certain experiments.

With regard to No. 1, Dr. Wernock very kindly gave access to the cases and the books of the Asylum. He also rendered valuable assistance in the way of suggestions and books of reference.

On his suggestion, Sharkin—the province giving the greatest returns of such cases to the Asylum—was chosen as the most suitable place in which to begin.

At the Asylum, also, where a room was set aside for the purpose, some pathological work has been done, and material collected from post-mortems on typical cases.

Experiments are being performed on the effects of inoculation of cerebro-spinal fluid obtained in such a manner. Experiments on cerebro-spinal fluid obtained by Lumbar punctum still remain to be performed.

With regard to No. 2, the methods of drying, storing and cooking

durra have been noted in the different Markaz towns, as well as in several villages of Sharkin; samples of bread and durra were taken for further investigation, either in the way of chemical analysis or for experiments on animals.

At the same time patients were examined and suitable ones chosen for further examination in the hospital. A similar procedure is proposed for Ghalioub.

With regard to No. 3, for purposes of investigation, patients were brought to hospital in order to be under closer observation. In all, sixteen cases have been so examined—of these, only two remained.

In Benha Hospital, where a start has been made on similar lines, there are now five cases under observation.

With regard to the Zagazig cases, for various reasons, ten were found unsuitable for investigation and were discharged cured, either with or without treatment other than hospital diet and hygiene. The remaining six had their diet or treatment varied, in order to observe any change in their condition.

One boy ten years of age (see photo.) had no treatment other than three months' residence in hospital—in which time his weight increased by four kilos, and diarrhœa practically disappeared. (He had no medicinal treatment.) His rash completely disappeared, although on admission he was one of the most marked cases. He has been discharged, but his further progress must be watched. He has to report himself regularly.

Five cases who had shown improvement on the ordinary hospital diet were at varying times, for varying periods, put on durra bread instead of the ordinary wheaten bread of the hospital. The durra bread used was made from durra obtained from a native house, and was prepared from time to time in the usual way by a native woman. The changes under these conditions were in some instances marked, being of the nature of a toxemia. There was noted return of diarrhœa, loss of weight, dull and listless appearance, with clayey color of skin. There was no return of the rash, however, possibly neither the length of time nor the conditions favored its return. This is a point worthy of further investigation.

Some cases showed distinct improvement on soamin. No conclusion, however, can be drawn, as others showed a similar improvement without any medicinal treatment.

It would be an interesting further stage of the investigation to live in one of the villages largely affected as Minet-el-Gamh. Patients

could be treated there whilst living in their homes, so that the effects of hospital diet and hygiene could thus be eliminated.

One case, aged 25, ( ), deserves special attention. This patient was for the first eleven days of his stay in hospital on hospital diet, from which maize is totally excluded. He improved in condition, and was put on durra bread for three weeks; but his symptoms became so marked that at the end of that time the hospital diet had to be renewed. During the three weeks of durra bread he lost 2.3 kilos in weight; he has now been two months on hospital diet, and has put on 5.2 kilos. During the period when he was having durra bread, he fell off markedly in condition—his skin became dark; but no rash appeared, and the diarrhoea increased. He also developed delusions, but they have now disappeared, and his mental and physical conditions have much improved.

Another case, age 13, ( ), while still on durra bread, continued to increase in weight; but his other conditions were marked—namely, dull and depressed state, with toxemic symptoms. After the durra bread was stopped he became much brighter in appearance, and his color began gradually to improve.

A point worthy of note, and one which markedly complicated the clinical picture in the cases observed, was the fact that all the cases, especially under observation in the Zagazig Hospital, suffered from bilhargia; whilst all, with three exceptions, suffered also from anchylostoma. This point is of great interest, and may possibly have some bearing on the fact that patients fall a victim to the disease whilst their brothers and sisters, living under similar conditions, escaped.

With regard to No. 4, namely, experiments, much remains to be done.

On the hypothesis that decomposing durra has to do with the causation of pellagra, animals were fed on such durra, whilst others were fed on healthy durra—as controls.

In the case of chickens, there seemed at first to be a stunting of the growth of those fed on the diseased grain; whilst others, from the same nest, thrived on good grain. However, after a time the former birds seemed to get accustomed to the damaged grain, possibly due to the establishment of a tolerance for the toxine (if much be present), or the grain was less damaged than formerly, so that the birds seemed able to thrive on it.

In the case of rabbits under similar conditions, two that were fed on damaged maize showed a distinct difference from the controls

that were fed on good grain, the difference in weight in one case being 80 grammes. The difference in their coats was marked—in the case of the former they were turned and staring, whilst the latter were smooth and normal.

Bread made from damaged grain is being analyzed to see, if possible, if some extract can be found of the nature of an alkaloid. Also the moulds are being identified so that, if possible, they can be artificially grown, and so found pure for purposes of further experiment and investigation.

It is impossible, at such an early stage of an investigation of this nature, to draw any definite conclusions.

Of the many theories put forward as the cause of pellagra, the maize one, from the great support it has received, has been given first place. At the same time attention has been paid to other theories (*e. g.*, the protozoan one), and blood films have from time to time been examined.

Judging from the work so far done, one feels only justified in saying that at least damaged maize, as found in some of the villages, and presumably used for food purposes by the poorer fellaheen, seems to cause a condition of the nature of a toxemia. Whether this is due to some poison elaborated by a fungus growth in the durra grain or to the fungus itself, only further research can decide.

Judging from the experience so far gained, the following would seem to be the best lines upon which to continue this research.

1. That a continued minute examination be made of typical cases, over a lengthened period of time, at the homes of the patients as well as in the hospital.

2. That the different provinces be visited, both in Upper and Lower Egypt, so that the differences in prevalence may be compared with the different conditions—either as to the nature of the durra or its process of drying, storing and cooking.

3. That the attention of Markaz doctors be drawn to the subject, so that through their barbers an estimate be made of the prevalence of the condition in Egypt. This has already been done in the case of Sharkia—the numbers, however, remain to be checked.

4. That further blood examinations, also experimental tests and chemical analyses, be carried out.

5. That an extended study, over a lengthened period, at all times of the year be made, so that the difference in incidence and various stages of the condition be noted.

Many difficulties have had to be overcome owing to the reticence of the natives, their ignorance of the disease as such, and their fear of any interference.

At first great difficulty was experienced in obtaining true histories of the cases and of the duration of the illness. Later, when the confidence of the villagers had been gained, this improved somewhat. It is hoped that the return of the patients cured to their villages will strengthen their confidence and make it easier for further investigation.

THE GYNECOLOGICAL, OBSTETRICAL AND SURGICAL ASPECTS OF PELLAGRA—A PRELIMINARY STUDY

ELEANORA B. SAUNDERS, M. D.

Assistant Physician, State Hospital for the Insane

COLUMBIA, S. C.

When we realize that the Italians have been studying pellagra for one hundred and seventy years, it seems impossible to advance any new theories or ideas about this disease. Still certain aspects of the problem have forced themselves upon my attention during the last two years so that I beg to ask your consideration of some gynecological, obstetrical and surgical phases of our newly recognized malady. I wish to report briefly a series of cases in some of which pellagra was not only not recognized, but did not manifest itself cutaneously till after surgical intervention; also others associated with menorrhagia and post partum hemorrhage and some cases developing pellagra after parturition.

Sir Henry Holland, writing upon "The Pellagra in Lombardy" in 1817, says: "In females, the menstruation is generally continued without irregularity," but such has not been the fact in our cases, as the majority suffer from amenorrhea or menorrhagia. The amenorrhoea is probably due to the anemia secondary to the disease. In these cases normal menstruation does not return until either the patients are on the road to recovery or after they are fully restored.

Patients suffering from menorrhagia are relieved of that condition by a recession of the pellagrous symptoms. In young girls and unmarried women, amenorrhea is the more common, while menorrhagia and metrorrhagia are more often the rule in multiparous women. The flow may occur at the regular periods, irregularly or almost continuously, but amenorrhea is most frequent in our patients. Rather than read a long and possibly wearisome list of histories, I have endeavored to summarize in the briefest abstracts the twenty-four cases that form the basis of my paper. These observations are based upon the systematic study of white females only.

CASE 1—

Girls eighteen years old, typical syndrome of eruption, stomatitis, diarrhea, depression, amenorrhea for months, slow recovery, (not an asylum case).

CASE 2—

Single, twenty-six years of age, history of several annual attacks of pellagra, amenorrhea throughout every attack, very red tongue and mouth, great fear of water, pellagrous dermatitis. Later prolonged profuse menstruation. In this case the menstrual abnormalities were entirely due to pellagra. Recovered. (Private patient).

CASE 3—

Married, forty-two years of age, ten pregnancies. During the last pregnancy had eruption, fiery tongue, depression. After normal labor as the doctor was leaving the house, the nurse exclaimed, "The patient is flooding." A very severe post-partum hemorrhage followed with a resulting anemia of thirteen months' duration. Recovered. (Private case).

CASE 4—

Twenty-six years of age, single. Became depressed, had eruption and menorrhagia which was relieved for a time by currettage. Finally menorrhagia developed with delirium, convulsions and death from typhoid pellagra. (Treated at home.)

CASE 5—

Twenty-four years of age, single, very nervous, "run down" and weak; suffered from menstrual disturbances, including marked metrorraghia. Had uterine operation without benefit. Eruption developed later and was followed by marked depression, necessitating admission to State Hospital. On admission stomatitis, diarrhea and eruption. Improved mentally. Discharged.

There is often a very annoying pruritus with or without vaginal discharge. These patients complain bitterly of the intolerable itching and burning, regardless of the degree of inflammation. The vulvar mucous membrane often shows the same "stippled" appearance as the tongue. Small hemorrhages, such as are observed under the epidermis of the palms and soles have been seen under the vaginal and vulvar mucous membrane and adjacent skin.

All writers have emphasized as the most striking pellagra symptom the development of dermatitis on the exposed surfaces of the body and ascribed this erythema to the sun's rays. This most obvious but really least important symptom probably accounts for the fact that pellagra has been recognized in American dermatologies

while omitted from other text books. It is only recently that some attention has been given to the evolution of pellagrous inflammation upon portions of the body not subjected to the sun's influence; such as bony prominences and folds of the joints. Here the cause of the hard and rough skin has been assigned by some to pressure. (Sandwith.)

Description of these lesions upon unexposed surfaces are rare, so that I take the liberty of quoting at length from a recent article by Nicolas and Jambon of Lyons, France. (Annales de Dermatologie et de syphylographie, 1908.) "Pellagrous vulvitis has not been described up to the present by writers with the frequency it deserves. Brault of Algiers, however, has described it in several of his cases. When we ourselves saw it for the first time, we considered it common intertrigo due to the lack of bathing. But the establishment of similar lesions in other cases and the reading of Brault's observations, made us study this symptom with greater care. It manifested itself as an erythema, very acute, slightly painful, giving sensations of smarting, burning and occupying the whole vulvar region as well as the perineal, the anal fold and the internal surfaces of the labia majora, internal and external, are erythematous patches. These lesions are oozing and take on a pseudo-membranous appearance. The erythema lessens upon the labia minora and ceases completely at the vestibular vaginal commissure. The same condition exists at the anal commissure, the mucous membrance of which does not participate in the inflammation. The invaded regions are the site of an infiltration sufficiently marked and then maceration of the epidermis rapidly develops. A fetid odor is given off. The prognosis of this erythema is essentially chronic like that of the whole malady. The lesions appear at about the same time as the skin and buccal manifestations. They do not exist without those manifestations, but they may be absent."

My own observations upon cases have led me to the following conclusions;-some of which verify those of Brault and of Nicolas and Jambon. From the vaginal mucous membrane there is a thick exudate identical in appearance with the viscid secretion so often seen in the mouths of pellagrins and frequently not only suggesting but really regarded as salivation (pyalism).

There is also often a very profuse exudate from the vaginal mucous membrane, which being acrid causes a maceration of the skin. In other cases a distinct excoriation or erosion and in some a complete denudation develops. I, too, was disposed to believe it due to

untidiness in patients and from very acid or alkaline urine, but examination shows the urine to be bland. Here, I may mention that as is the case in Italy I have frequently found the urine of pellagrins ammoniacal. On the skin about the perineum are areas varying in size from very small spots involving almost all the inner side of the thighs, extending far around to the gluteal regions and reaching in some cases almost to the knees. This area is always very red, swollen and oozes constantly a profuse sero-sanguinous fluid. This appearance is usually seen in the so-called "wet" cases where the skin of the hands and other parts is in the same bullous condition. These stigmata of pellagra upon the unexposed parts of the body are due, of course, in the first place to the virulence of the unknown poison, since they are not seen in mild cases. Secondarily, the inflammatory exudate produces maceration which in turn further extends the zone of inflammation. Here also, pressure may be an element in these cases. I may mention in passing that colored women appear to be more prone to these moist pellagrous lesions than white women. This applies to private cases as well as to asylum pellagrins. Also the extent of the dermatitis and the degree of pigmentation seem to have a prognostic value or a direct relationship to the subsequent liability to the development of more serious mental symptoms and of typhoid pellagra.

The vaginal mucous membrane of these cases shows the same red inflamed condition, and here too is seen a peculiar mottled appearance as of small hemorrhages under the membrane. Cases examined at the State Hospital show the vaginal mucous membrane to be inflamed in equal degree with that of the buccal and other mucous membranes.

Very decided pain is felt in the region of the ovaries at the menstrual period and irregularly. Autopsies show a red congested ovary, the whole body of the ovary being involved. Pregnancy by its debilitating effects, has its weight here and objective pellagra has at least been hastened or aggravated by this great physical strain. Frequent pregnancies and prolonged lactation by lowering vital resistance may alter the character of the case. "The considerable rate of still births," says Marie, writing of pellagra among the Egyptian Arabs, "would not exist without this great malady." In another place Marie says, basing his remarks upon Lombroso's observations: "In hereditary pellagrous women are noticed atrophy of the breasts, irregularity or absence of the catemenia, which is sometimes followed by a painful metrorrhagia. Calderini notes dysuria

in 57 per cent. of the females pellagrins, amenorrhea of 50 per cent.; leucorrhea in 50 per cent. and abortion in 17 per cent. Many cases suffer from subjective sensations of uterine inflammation or displacements without objective signs. A robust country woman in the hospital at Verona, thirty-one years old, the mother of five children, complained of violent pain on urination and a sensation of weight in the uterus "as if it were made of lead," with burning at the cervix uteri. She would not tolerate a speculum for examination but was treated a long while for metritis. Finally she began to complain of epigrástric swelling, burning on the back, alternate sensations of hot and cold and had diplopia; later she developed a melancholic mutism without adequate cause. It was then thought that perhaps the uterine symptoms might be of a pellagrous nature. A careful examination with the speculum, however, showed no trace of metritis. It turned out to be a case of unrecognized pellgra."

Other authorities assert that "the female sex, as we have seen, is decidedly more disposed to pellagra, and this arises probably from the fact, that especially the nervous system of women is easily affected, and that they are more overburdened than the men, particularly in the country population of Roumania by work and child bearing. Indeed the greatest frequency of pellagra in women begins only after the 20th year and continues up to the 40th, accordingly, in the time of the sexual life of the women. In general most of the pellagrins are found to be within this period of years, although many children are pellagrous. This is evidently due to the fact that the rural population in more under a strain in these years 20 to 40, while their diet is not sufficient for the amount of the work done." (Babes and Sion.)

Case 6—

Married ten years, 28 years old, first pregnancy ending about twelve months ago. Three weeks after child-birth she suffered with sore mouth, but had nervous symptoms before confinement. Stomatitis, diarrhœa excitement, later eruption on hands, which grew worse. Patient lost ground physically and mentally, became suicidal and consequently was admitted to asylum. Has run a typical course and has been very suicidal, and at present all symptoms are better except the mental. Still under observation.

Prolonged lactation by enervating influence may cause a depression amounting to resistive melancholia. Such cases becoming pellagrous usually complain of "getting run down," and are thin and

nervous. Diarrhea sets in and the eruption completes the picture. In time it is to be hoped that we shall learn to diagnose, or at least to suspect the presence of pellagra in debilitated and nervous women without waiting for the appearance of the eruption.

CASE 7—

A decided blonde, age 32 years, second child three years of age, prolonged lactation during the summer of 1908, was much debilitated and "run down." Eruption appeared later, but she continued to nurse the child until admitted to asylum in April, 1909. Since admission she has shown great mental instability, is moody, emotional, and at times very noisy, has bad diarrhea and eruption, which is exceptional in that it extended far above the elbows. (Is still under treatment).

Frequent child-bearing by sapping the strength of these nervous patients seems to play its part as a predisposing cause in the development of the pellagra syndrome. Already Strambio about 1790, as noted by Babes and Sion, had observed that gestation gave occasion to the breaking out of pellagra. He also observed cases in which pellagra appeared only during gestation and lactation, and then receded.

CASE 8—

Married, age 49, history of many and very rapid pregnancies, always run down, had eruption several years. Very depressed. Upon admission to asylum had dermatitis and diarrhoea. Abject depression marked this case. She suffered from menorrhagia. Finally she made a complete recovery. Discharged.

Surgical operations acting as trauma develop latent pellagra and lowering the resistance aggravate or hasten its course. In the early cases, we have all the train of symptoms: backache, pelvic distress, head-ache, bearing down pains, weakness, lassitude, emaciation, ovarian and menstrual disturbances, which are seen in pelvic disease, for, as Manton well says, mental symptoms are present in gynecological cases. When the eruption is not present, the unsuspecting doctor or consultant is most likely to pronounce the case pelvic and treat the symptoms as pointing to a primary gynecological trouble without thinking of its being a part of the pellagrous symptomatology. Here, too, the mental depression is regarded as merely a symptom of the pelvic disease and operation or special treatment is advised. The consultant neglects to ask for a history of the eruption for one

year or maybe for four or five years, and at the same time there is perhaps a very red tongue, swollen and indented with stomatitis which he overlooks or diagnoses and treats as ptyalism and disregards the severe wasting diarrhoea with increased knee jerks, pupillary anomalies, marked atheroma, enlarged epitrochlears dorsal pain and "nervous dyspepsia." I am compelled to say that cases of so-called post-operative insanity admitted to this asylum point to this conclusion, for in the spring and summer of 1908, we had, as we do every year many such cases which showed all these symptoms of early pellagra and gave histories of former eruptions and diarrhoea. To cite an unusual occurrence but striking coincidence, in one week we admitted five cases of pellagra which had been operated on for gynecological trouble within the preceding two months and all of these at the time of admission had or developed within a month, the typical objective pellagrous stigmata. Three of these five died and two went home, recovered.

The uterine hemorrhage, irregular or continuous, accompanied or not by a vaginal discharge, especially in women of advanced years, will alarm the doctor, and even though there be other symptoms pointing to pellagra, he immediately diagnoses cancer and strongly urges the family to have an hysterectomy done. Such pellagrins have been subjected to operations for cancer in this country and abroad. The gastric disturbances call the attention to the stomach, and ulcer or cancer is thought of at once. When an exploratory operation is done, the stomach is seen to be in the same inflamed condition as the mouth and other mucous membranes. Even the kidney does not escape surgical invasion. The absorption of fat accompanying the emaciation may be the cause of a so-called floating kidney and perhaps, too, it will partially adjust itself and waiting for a time till the patient has recovered from pellagra, can in many cases be put off and watched closely for alarming symptoms of any kind due to the condition of the kidney.

The point that I wish to make is to suggest that hereafter as the recognized pellagra zone widens, when confronted with a nervous or mental case in a woman for whose relief their intervention is sought, surgeons and gynecologists must reckon with the pellagra syndrome and at least temporize in trying to relieve the pellagrous symptoms and thus prepare the patients for the surgical ordeal before resorting to an operation. When we fully realize how unequal is the struggle against pellagra alone, how can we subject already

enfeebled patients to the ordeal of a capital operation to be followed by the combined effects of pellagra and trauma?

The following cases illustrate my contention. These cases, one and all, had had surgical operations to relieve pelvic symptoms and were brought to the Asylum very soon after these operations had failed to relieve the symptoms delineated.

CASE 9—

Single, twenty-four years old when admitted to the asylum. Prolonged ill health accompanied by ovarian symptoms for which ovariotomy was done, soon followed by pellagrous dermatitis with refusal of food, diarrhoea, mutism, ideas of poison, exhaustion, emaciation, death.

CASE 10—

Married, aged 43 years. Very acute ovarian affection, ovariotomy followed by pellagrous rash, stomatitis, diarrhoea, very excited, admitted to asylum, died after three weeks of delirium from typhoid pellagra. Temperature 107 before death.

CASE 11—

Single, age 25, always suffered from ill-health and dysmenorrhea. Developed dementia precox, ovariotomy, pupura, admitted to the asylum, diarrhoea, catalepsy, resistive, refusal of food, emaciation, sudden death.

CASE 12—

Single, 24 years of age, mother insane with history of unrecognized pellagra. Ovariotomy for "female trouble," after which she was admitted to asylum and in a few days showed a pellagrous eruption, stomatitis, diarrhoea. After months of treatment for the mental trouble, was discharged recovered.

CASE 13—

Married, twenty-six, ovariotomy for a depression of many months' duration but had also dermatitis, diarrhoea, great emaciation and lassitude, mutism, greatly apprehensive. Admitted to the asylum very prostrated, being an apparently hopeless case. Recovery after many months' residence in asylum.

CASE 14—

Married, 27 years of age, female trouble, prolonged lactation, depression, uterine operation, partial recovery but soon relapsed, depression, noisy at times, rapid emaciation, dermatitis, diarrhoea, admitted to the asylum, eruption still present, catatonic, spastic, mutism, dilated pupils. Still under treatment.

CASE 15—

Married, 23 years of age, female trouble, weak, nervous. Hysterectomy followed by excitement. Admitted to the asylum with diarrhoea, refusal of food, dermatitis, great emaciation, suicidal, death from exhaustion.

CASE 16—

Married, forty-one, repeated gynecological operations for "nervousness and female trouble," loss of appetite, petulant disposition, diarrhoea, indecency. Committed to asylum, soon developed stomatitis, eruption and emaciation. Removed to private asylum. Died from pellagra.

CASE 17—

Married, fifty-eight, severe pains in ovarian region, nervousness, ovariotomy, excited, admitted to asylum, emaciation, eruption, delusions of poison, death.

CASE 18—

Single, twenty-one, history of nervousness and flooding. Operation. No improvement. Another operation was advised, but was refused. Recovered from menorrhagia. Suffering from amenorrhea and mental symptoms, was admitted to asylum, a typical pellagrin. Recovered.

CASE 19—

Married, forty years old, diarrhoea, digestive disturbances, rash, depression, ovariotomy, relapse for two years, exhaustion, death. (Never admitted to asylum).

CASE 20—

Married, twenty-nine years old, eruption, stomatitis, diarrhoea, digestive disturbances, stomach contents suggested ulcer. Exploratory operation showed an inflamed and a very red stomach. One week after operation red rash on hands and blebs, followed by desquamation. Recovered. (Mental depression not sufficient to call for asylum treatment).

CASE 21—

Married, thirty-two years old, diarrhoea, dilated pupils, depression, emaciation, operation for floating kidney. Symptoms of depression, nervousness and not benefited mentally by operation, admitted to asylum with diarrhoea and typical stomatitis and eruption. Still under treatment.

CASE 22—

Single, thirty-two years old, "female trouble," hypochondriacal, nervous, ovariotomy, emaciation, digestive disturbances, persistent vomiting and diarrhoea, admitted to asylum for treatment in almost dying condition, carphologia, severe diarrhoea prostration with large moist rales. Prolonged course. Recovery.

CASE 23—

Single, twenty-four years old, after months of declining health, ovariotomy was done to try to relieve her trouble. Emaciated, rigid, very excited, stomatitis, rapid development of eruption, very wild delirium. Death in a few weeks after admission to asylum from typhoid pellagra.

CASE 24—

Married, forty-four years old, hysterectomy for hemorrhage and discharge accompanied by cachexia and declining health, excited, restless. Admitted to the asylum, very sclerotic, varicose veins, large simple goitre, diarrhoea, eruption, death.

To sum up my observations and reading upon the aspects of pellagra embraced in this paper: Of the twenty-four cases reported, four are still under treatment, 10 recovered, 10 died.

GENERAL—In America and Roumania the female sex is more liable to pellagra, the period of greatest incidence being the 20th to 40th year. Pregnant women suffering from pellagra are liable to abortion (17 per cent.) to give birth to still-born infants and at delivery to post-partum hemorrhage. Gestation and lactation, especially when frequent, predispose to pellagra. Parturition is often an exciting cause for the outbreak of the dermatitis. Amenorrhea and leucorrhea occur in 50 per cent. of the cases and dysuria in 57 per cent. Unmarried female pellagrins are more subject to amenorrhea. Multoparous pellagrins are liable to menorrhagia, and present symptoms suggesting cancer. Their subjective symptoms may point to diseases of the pelvic organs and require careful examination for their exclusion.

Not uncommon are vulvitis, vulvo-vaginitis, cervical erosions, endo-cervicitis and endometritis, ovarian neuralgia and inflammation, maceration and denudation of adjacent skin upon the thigh and in perineal and anal region, especially in "wet" cases.

SURGICAL—A surgical operation may bring out latent pellagra. Diseases of kidney are simulated and may be primary or secondary.

Stomach symptoms are often so severe as to require attention and rigid diagnostic methods. Care should be exercised to prevent needless surgical or other treatment.

Other subjective symptoms may annoy the patient to such an extent as to demand treatment. Pellagrins often complain of symptoms suggestive of hemorrhoids when it is really proctitis, a part of the general inflammation of intestinal mucose and adjoining epidermis.

In essence, pellagra may be a tropho-neurosis, but in women the pelvic organs are especially subject to the invasion of the unknown poison, is a fact demanding wider recognition as well as further study.

Of course, ours is the dark side. In our asylum life we see a few failures among many brilliant surgical successes, but the above surely point to the fact that in the initial stages, especially where the symptoms are not very well marked and where there is much pointing to pellagra as well as to other diseases, a very careful differential diagnosis should be made and care given to other than the pelvic symptoms of these cases. If possible, relieve the primary disorder, pellagra, before resorting to radical operations, which at best cannot cure pellagra, and will probably only increase the already lethal tendency. Not much will be lost in waiting for a time, at the expiration of which the symptoms may have been relieved, or the pellagra erythema now so necessary for diagnosis have appeared in full effloresence.

Cases of pellagra on account of their debilitated condition are prone to other diseases and are subject to "incidentals" which do require surgical intervention. These, of course, demand and should have prompt attention. But after granting all this, I am forced to believe that the majority of such cases should be treated not as having a primary organic pelvic disease, but as suffering from functional or symptomatic disorders, and, furthermore, that the gynecological, obstetrical and surgical aspects of pellagra are factors which not only the general practitioner must consider, but with which specialists of several kinds who live in the "pellagra zone" must hereafter reckon for the real welfare of their patients.

PELLAGRA—ITS RELATION TO INSANITY AND CERTAIN NERVOUS DISEASES

J. W. MOBLEY, M. D.

MILLEDGEVILLE, GA.

Much has been written in the last two years on pellagra, and especially pellagra in the South.

The daily newspapers have given considerable attention to the subject, and agitation of this malady has arisen from many other sources since its general recognition by the medical profession in the South.

The purport of this paper is not so much to deal with the history of pellagra, but to present the disease, as the writer sees it, in its many sided clinical aspects. At the very outset, we are drawn into contention with a serious problem. There is no further question as to the existence of pellagra throughout the South.

It is here in its true and allied forms, confronting us with such an awful clinical picture as to carry death and despair in its very name. The alienist looks back with regrets upon the mortality of this scourge in hospitals for the insane. He is made to reflect with deep concern for the future of his race, when he observes the powerful impress of this disease upon the mind and nervous system. The general practitioner is not so much called upon to consider pellagra in its relation to insanity. On the alienist rests the moral duty of warning his people against an infection which, once firmly rooted, will manifest its baneful influence forever upon the human family.

Discouraging enough would it be if each individual case represented a morbid entity unto itself. The picture becomes more serious, however, when we contemplate the role which pellagra plays in the chain of heredity. Its poisoning effects are not spent alone upon the individual subject, but like tuberculosis, it transmits a morbid inheritance to the recipients of its toxines.

I desire here, gentlemen, to deeply impress upon you the evil which this disease must represent as a vicious factor in our social system. What would the world give today if it could recall the propagation of idiots? What a stain could be removed from medical science if the life-history and management of tuberculosis might be reviewed. Many already are the deteriorating forces which prevail against our

race. Shall we sit idly by and allow the ravages of this insidious monster to sap, unbridled, the vitality of our future generations?

Upon this distinguished conference rests the responsibility of inaugurating measures looking to the suppression and control of this rapidly increasing malady.

PREVALENCE.

The report of the Public Health and Marine Hospital Service for 1909 will show that Dr. Gray, of New York, and Dr. Tyler, of Massachusetts, reported two probable cases of pellagra forty-five (45) years ago. I am informed that both of these gentlemen were alienists, and you are here reminded again of the splendid opportunity offered physicians to hospitals for the insane to observe these cases. It is, indeed, hard to account for the stuporous lapse of time in which forty years or more passed away without the record of a single case in the United States. Harris, of Georgia, and Shewell, of Chicago, finally reported a case each in 1902. Following the last two mentioned cases, there was another lapse of comment upon the disease until 1906-1909, when there seems to have been an epidemic of observations as well as cases reported.

Probably to Babcock, of South Carolina, is due the greatest credit for the most thorough report of cases in the South, and a complete bibliography of the disease in this country.

No doubt, pellagra has been steadily on the increase in the United States since 1860. The impression prevailing that it was a disease indigenous to foreign countries, not much vigilance was given to its detection.

It is true that many clinical conditions may have been confused with pellagra, more especially certain nervous diseases attended by secondary trophic symptoms, dependent upon faulty innervation, but such are not sufficient excuses to warrant so great an oversight on the part of the medical profession.

ETIOLOGY.

The cause of pellagra will probably be found in both general and specific factors. Just to what extent, if at all, they operate together, we are unable to say. The various clinical aspects of the disease, however, would justify us in considering its etiology under several headings.

First.—Is pellagra produced only by a specific organism, and its toxines?

In considering a specific factor as the sole cause of pellagra, we are brought to face with many conditions which speak both for and against this etiology. The labors of the very best men in this country and abroad have been surrounded with doubts and practical inconsistencies, but have ultimately, and, with some degree of reluctance, converged into the opinion expressed by the famous Italian professor, Dr. Lombroso—that a fungus growing upon maize produces a toxine which causes the disease.

Other specific factors are shown in the protozoan theory, which so closely allies syphilis with pellagra as to make their clinical picture at times quite similar. The peculiar cell observed by Dr. Cross, of Georgia, with a chlorophyle-like content is on the side of a specific infection. He describes these cells as about the size of hookworm eggs, either round or oval in shape. He suggests that they may have been altered epithelial cells, but their morphology seemed to be unchanged.

The specific fungus (aspergelli) which grows upon corn will also grow on cheese, and may account for the disease in many instances, where the history of a corn free menu is obtained. The confusion and tonic muscular state, known as "blind staggers," incident to horses that are supposed to have eaten molded corn, indicates a clinical phenomenon of vicious infection and specific origin. Brown and Low report a case as having occurred in a shopwoman from the frequent taking of raw oatmeal and rice. Maize seems to have played no part in the production of this case.

Much more evidence might be cited in favor of a specific etiology, but as this theory does not prevail unanimously in this country, and more particularly in the South, we will proceed to other factors yet to be considered.

Secondly.—Is the disease caused by the toxine of a specific organism, operating only in the presence of a peculiar morbid predisposition, racial peculiarity? Or does any latent or active pathological state favor its development?

In studying the causative factors of pellagra from this viewpoint, we are brought to face with inherited pathological tendencies which cover a broad field in general medicine. Are certain individuals immune, or are all classes susceptible to the infection? The clinical behavior of the diesase opens this query, but I shall not attempt to discuss it here. Certainly an immunity does not appear to develop

by virtue of previous attacks, as competent observation will point to the conclusion of an incurable malady. The negro female in Georgia seems to be the selective host. The explanation of the apparent proclivity for this race is untenable unless explained in the deteriorating phenomena which marks so great a decline in the generic purity of this class since their so-called freedom. Apropos of this idea—impressive are the facts that disclose the alarming increase of syphilis, tuberculosis and insanity in the negro race of Georgia since their emancipation. Dr. T. O. Powell, former Superintendent of the State Hospital, in an able paper, shows that there were only forty-four (44) insane negroes in the State of Georgia up to 1860. Previous to this time they enjoyed splendid health, and tuberculosis and insanity were exceedingly rare among their race. These forty-four insane negroes occurred in a population of 465,698, representing approximately one insane to every 10,584 of negro inhabitants. The succeeding ten years show an increase of insanity in the negro to one in every 4,225, with a colored population of 545,142. The census of 1890 gives the total negro population in Georgia at 858,815—the number of colored insane at 910; advancing the ratio in thirty years from one to every 10,584 to one to every 943 of negro inhabitants.

Georgia may be accepted as a fair index to the social, physical and mental condition of the negro in the South. Syphilis and tuberculosis, fraught with their chain of moral and physical deterioration, have progressed *parri-passu* with insanity in the Southern States since 1864, and should be considered here, especially, in relation to pellagra. These diseases, so infrequent in the days of slavery, are now so common as to represent the principal chronic maladies from which the negro suffers. Interesting will it be to note here that the post-mortem records of the pathological laboratory of the State Hospital for Insane of Georgia will show that out of one hundred unselected necropsys about ten (10) per cent. of patients dying probably of pellagra, manifested lesions also of localized or general tuberculosis. If, on the contrary, we presume that death in these cases was caused from tuberculosis, then a like per cent. exhibited symptoms and morbid changes suggestive of pellagra. The association of the two diseases are, to the author, too frequent to pass unnoticed. The microscopic findings further show areas of syphilitic necrosis, together with the gross tubercular changes in some of these cases.

The anatomical diagnosis of the majority of these cases appearing on the records as tubercular enteritis.

Considering the fact that pellagra is a chronic, progressive disease;

considering the fact of its frequent association with syphilis and tuberculosis; and considering the relationship of syphilis to certain chronic nervous diseases, as paresis and tabes—are we not justified in suspecting the existence of a morbid and etiological equivalent in the three conditions?

The basic principle of perfect health is pure blood and an untainted physical make-up, and while each individual may possess an independent reaction index both in health and disease, yet, the impress of chronic morbid factors, operating through generations, must finally be expressed in a reduction of the health standard, in the scale of human life.

The influence of crossing in the negro has brought as its product an hybrid type, with a weakened vital resistance. The most potent etiological factor in the production of insanity is insanity itself; the same may be said of tuberculosis. The scientists tell us that neither of these diseases are transmitted as such; but no student of anthropology would attempt to deny their destructive effect upon the moral and organic continuity of the human race.

As an hereditary factor, pellagra possibly possesses, along with insanity and certain other chronic organic diseases, an etiological equivalent in its own production.

Among other contributing causes in the production of pellagra, I feel compelled to mention certain intestinal parasites. Dr. Willets, Pathologist to the Georgia State Sanitarium, has shown that out of five hundred unselected insane negro females, thirty-five had pellagra. The fecal examinations of the entire number (500) disclosed the fact that 50 per cent. were infected with some form of intestinal parasite, the percentage and name of each respective parasite being given in the table below:

Number Negroes Examined.	Infections.	Ascaris.	Trichuris.	Strongyloides.	Uncinaria	Hymenolepis.
500	50%	28.2	40.2	18.2	11.8	0.2
Pellagra.						
35	40%	8.57	17.14	25.71	20.0	2.86

The thirty-five pellagra cases, of course, are included in the total number examined; that is, five hundred.

You will observe from the table that the highest per cent. of infection in the pellagra cases is from strongyloides and uncinaria. These parasites are both blood suckers, and are quite hard to dis-

lodge from the intestinal mucosa. Whether their clinical significance extends beyond this scope, I am unable to say.

I do not regard pauperism, *per se,* of any value in the production of pellagra, aside from the unfavorable conditions of habitation; neither has the effect of the sun rays been satisfactorily analyzed. The question of contagion received some support through the peculiar development of the disease among recent admits to hospitals for the insane.

Dr. N. P. Walker, of the Georgia State Sanitarium, reports that out of eighty-nine (89) cases of pellagra, among the insane colored females, only nineteen or twenty-one per cent. were infected with the disease at the time of admission. The remaining seventy (70) cases exhibited the first known symptoms of pellagra after admission.

The table below shows approximately the time of first known symptoms after admission, in the colored female cases referred to:

Months	Months	Months
One to four	Five to eight	7
27	11	Nine to twelve
Unknown	One to two years	One case ten years
4	20	after admission.

This table may be accepted as representing fairly correctly the status of the (80) cases, more or less, of pellagra occurring among the white patients of the Georgia State Sanitarium in the last two years.

In my opinion, the records of all hospitals for the insane in the United States will show that 50 to 75 per cent. of pellagrous cases exhibit the first known definite symptoms of the disease after admission. Of course, allowance will have to be made for those cases of the manic—depressive group, with a marked inherited instability—and where the psychic disturbance is one of the very early symptoms of pellagra. It is of interest to note, in contrast to the frequency of pellagra in the colored female in Georgia, the negro male appears seldom to suffer from the disease.

However, so far as can be ascertained, there has never been a case of pellagra to develop among the nurses, white or colored, while employed as such in the Georgia State Sanitarium.

Third.—Certain pseudo-pellagrous states arising from a general toxæmia, resulting from a mixed infection of pathogenic bacteria, is only mentioned because of the many clinical conditions which seem to portray a picture of this character. More notable is this condition among the insane, and especially, such psychoses as

come under the dementia præcox type. These patients will sit in katatonic stupor with the buccal cavity full of offensive saliva. They will gradually develop a stomatitis with a chain of nervous symptoms and morbid anatomy almost identical with classic pellagra. From my observation, the digestive disturbance with diarrhœa usually precedes the local epidermal changes, in these cases, and the picture, as a clinical unity, points primarily to a toxic infection arising either from a neglected oral hygiene or a poisoned saliva.

Dr. John A. Fordyce, of New York, in a very excellent paper, has shown the very close relationship of mucous membrane affections to certain diseases of the skin. Many pseudo-pellagrous states are quite significant in this connection, as good observers have shown that catarrhal inflammations of the skin may interchange with those of the mucous membranes,—the one being active, while the other may be in abeyance. Pertinent is the theory in this connection that certain nervous diseases may have their morbid expression in cutaneous manifestations.

The physiologists tell us that the nervous system, the epidermis and the epidermic tissues, with the mucous membrane of the mouth and rectum, all have their histogenesis in the same embryonic membrane, that is, the epiblast.

Now, admitting the fact that the pathognomonic symptoms of pellagra are shown in a triad of morbid conditions visible through either one or all of these systems—are we in a position to deny that a general toxæmia, beginning in the oral cavity, might not be confounded with many of the pseudo-pellagrous states now existing throughout the country. Certainly with the physicians in Georgia, there is a disposition to assign to the pseudo-pellagrous group such morbid pictures as sustain a clinical relationship to chronic syphilis and tuberculosis. Especially is this true where syphilis and tuberculosis are associated with insanity, and the clinical picture is one of a wasting disease with such nervous symptoms as might be present in pellagra.

Take the novice and carry him through the colored female department of the Georgia State Sanitarium, and you will hear the spontaneous expression from him,—Syphilis! Tuberculosis! in reference to many of the so-called pellagra cases.

DIAGNOSIS—PSYCHIC PHENOMENA.

I trust, for lack of time, I may be pardoned for embracing under this general heading the remaining phases of pellagra which I desire to consider, though some do not properly belong here.

The disease, as a clinical problem, has attracted attention for many years in the Georgia State Sanitarium. It was construed as a morbid attribute to insanity, with certain nervous and physical complications, in which syphilis and tuberculosis played an important role as etiological factors.

As far back as 1898, the records of the pathological department of the Georgia State Hospital will show that such colloquial and incorrect nomenclature as foot and mouth disease, marasmus, malignant stomatitis, cochin china diarrhoœa, tubercular enteritis, and solar dermatitis were given to the disease, and these phrases are suggestive of such symptoms as may have predominated in the clinical picture from time to time. Finally, all of this doubt and confusion as regards the malady was concluded, to a degree, in the happy relief afforded by Tropical Aptha or Sprue, which became prominently recognized in Georgia through the splendid reports of this disease by Dr. H. F. Harris, of Atlanta. With the advent of sprue, however, there arose a narrow but perplexing chasm in the differential diagnosis of the two diseases; the differential points are still treacherous, and I fear their significance lies more in the hypothesis of theory than the practical arts of medicinal diagnosis.

I shall only record my personal observations as to the pathogenesis, as the conclusions of the very best men in this country and abroad are practically in accord as to the morbid changes. As to whether the disease possesses an incubation period, the author is unable to say. In all probability it does, yet, the writer has never seen such statement from reliable authority. The peculiar stomatitis, with or without aphthous deposits; pain in the back; a red-brownish pigmentation, sometimes of a fungoid character,—about the face, neck, chest, inner surfaces of thighs, vulva and upper folds of buttocks, are, no doubt, familiar to you all. The characteristic epidermal changes about the elbows and the dorsal surfaces of the hands and feet, varying from a mere discoloration of the skin to fissures and superficial necroses are too common to dwell on here. I wish to say, however, that oftentimes the epidermal changes involve the palms of the hands, soles of feet and, indeed, quite a number of instances have come under my observation in which the flexor regions of the wrists have also shared

in the necrosis. This is a rather infrequent condition and has not been observed by some writers on the subject. The enlargement of the glands; the low hæmoglobin percentage; in some cases nucleated red cells and an occasional leukocytosis. Ulcers in the cæcum, often tubercular in character, are frequently associated with pellagra. So far as I have been able to learn, there has been nothing of clinical significance in the spinal fluid. Brown and Low observed no increase of cells in the case examined by them. The re-action of the moist epidermal changes are as a general thing slightly alkaline. When there is diarrhœa, the stools may be from three to fifteen a day, either slimy pea-soup or light brown in color; sometimes frothy, and of alkaline or acid re-action. Dysentery may precede any definite pellagrous symptoms for weeks or months. Coarse tremor, with disturbances of co-ordination, is often present, depending apparently upon the degree of infection.

The cycle of the skin lesions holds no definite place, as to development and recession, in the morbid picture. The rash, while present more often in summer, does appear in winter, and I have seen bullæ develop on the hands, fingers, and thighs, of white females, from twenty-four hours to two days before death. Their occurrence is indicative of profound infection, and is usually associated with moribund states. The temperature curve is void of typical features, more often it is normal, or may be sub-normal or go as high as 105 degrees. The pulse appears to bear some relation to the degree of intoxication and extent of mental involvement. In my experience, there has been a striking absence of vaso-motor influence in relation to the skin and its eliminative function; the surface, even in the delirious forms, being dry and scaly. I have observed multiple abscess in two cases. The appetite is usually poor, in some cases ravenous. Vomiting is infrequent. The urine has shown nothing of clinical value. The musculature in mild cases presents the picture of fatigue toxæmia. The reflexes may be exaggerated, diminished or absent. Sensation among the insane is quite variable,—it is hard to determine whether there is any true alteration or not. In cases where the spinal-cord seemed to be involved, I have seen marked intention tremor simulating pseudo-convulsive attacks.

In considering the neuro-psychic phase of pellagra, it will be expedient to study, first, the two conditions together, without attention to their strict division into separate clinical entities. Indeed, I might say, seldom do we have in Georgia an organic disease of the nervous system, of pellagrous origin, without invasion of the psychic

realm. On the contrary, so intimately associated are the two maladies in many of their clinical aspects, that we are often perplexed to know which, if either, merits the place of priority in occurrence. An illustration will more adequately explain the confusion which has arisen in the author's mind as to whether a primary neurosis, of a non-pellagrous etiology, might exhibit a secondary symptom complex, simulating the pellagrous syndrome. Take, for example, amyotrophic lateral sclerosis complicated with insanity—the two diseases may progress with the preponderance of symptoms favoring a spinal cord lesion as the primary site of invasion. The mental states may vary from mild confusion to complete delirium; the reflexes may be exaggerated with a variable Babinski and Gordon paradox; the patient gradually develops a sore mouth, with alternating diarrhœa and constipation; later the skin lesions appear with variable intensity. Have we pellagra with amyothrophic lateral sclerosis and insanity as complications, or have we insanity and organic cord disease with a pellagrous complex? The cord lesions are usually confined to the lateral columns; the posterior or sensory may also be invaded, especially in the tabetic forms. The cortical cell changes, in the insane, are probably dependent to some degree upon the type of psychosis and its duration.

In our American nomenclature, we have no distinctive classification for pellagrous insanity; the different psychic alterations falling under such category as the symptom complex may indicate;—scientifically speaking, all psychoses of pellagrous etiology—excluding organic diseases of the brain and nervous system, should fall principally under the intoxication or infective exhaustive group. However, for the sake of clearness, I have divided the classification, generally speaking, into four headings. The conclusions, as regard the separate mental reaction states, having been obtained by carefully isolating such symptom groups as are more or less peculiar and constant in the different insanity phases.

First.—

The cases showing a profound intoxication, with early delirium, high temperature range, with symptoms pointing to acute organic changes in the cord or brain.

Controlling phase: Complete or incomplete psycho-motor suspension.

Classification: Acute Intoxication Psychosis.

Second,—

Those cases of an apparent mild infection with some mental anxiety, apprehensive hallucinosis, gradually increasing mental confusion,—finally delirium,—temperature subnormal or slightly elevated,—this type usually covering six weeks to two months or more, ending in a slow but progressive exhaustion.

Controlling phase: Psycho-motor Retardation—Excitation (active, passive).

Classification: Infective Exhaustive Psychosis.

Third.—

Those cases showing symptoms of mild melancholia, chronic in character, with remissions and exacerbations, impending fear, suicidal tendency, due more to apprehension than self-reproach; temporary recovery.

Controlling phase: Psycho-motor Retardation (inconstant, passive).

Classification: Symptomatic Melancholia.

Fourth.—

Those cases of mixed type showing at times symptoms of depression, exaltation, confusion, impulsive acts, apprehensive hallucinosis, exhaustion, slow mental reduction.—including the Dementia Præcox Class.

Controlling phase: Psycho-motor Retardation—Excitation. (Active, passive, negative).

Classification: Manic-depressive—allied states.

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DISCUSSION ON THE PAPER OF DR. MOBLEY

Dr. W. H. Dial, Laurens, South Carolina: I would like to ask those gentlemen familiar with pellagra whether or not it is necessary to have a dermititis in order to make a diagnosis of this disease. Last evening a gentleman from North Carolina, who mentioned a case in which there was stomatitis and other symptoms of the disease, said he had made a diagnosis of pellagra without the skin manifestations. What we want to know now is whether dermatitis is necessary as a symptom in pellagra. I would like to ask Dr. Babcock or any other member of the conference to answer this question.

Dr. J. W. Babcock, Columbia, South Carolina: With your permission, Mr. Chairman, I will state what little I know about the subject, and repeat what was brought up in the conference last year, namely, that students in the London School of Tropical Medicine are taught that they must make a diagnosis of pellagra regardless of the dermatitis; that they must make a diagnosis of pellagra without waiting for the skin manifestations to develop. Then, there are those cases which the Italians emphasize as of the greatest importance, namely, those which have an obstinate diarrhœa, with marked mental symptoms, and exaggerated knee jerks. In other words, to give a picture of pellagra without any skin manifestations at all, so that recognizing that we are all beginners in the study of this great disease, I think it is up to the general practitioner to formulate for those who only see this condition in the advanced stage to emphasize for us the particular symptoms which will enable us to make an early diagnosis regardless of the skin manifestations, because it is in that stage when, if we are to help the patient, we must make a diagnosis. When the disease reaches the stage of mental involvement, when it reaches the asylum condition, we have all the evidences of an overwhelming lethal tendency. I do not know whether I have made my remarks clear or not.

Dr. W. B. Young, Rock Hill, South Carolina: I have a case I would like to report. The patient is about 55 years of age, a farmer by occupation, who has a water mill and grinds most of the meal that he uses, and he has certain nervous symptoms which I do not understand. He did not have a dermatitis when I first saw him. He had increased knee jerks, stomatitis, but has never had diarrhœa. On the contrary, he was the subject of obstinate con-

stipation instead of diarrhœa. In working about the little store near his mill he would pick up an article and be unable to put it down, so that it became necessary for somebody to take it from his hand. Again, he would start to walk in one direction and go in the opposite direction. In attempting to sit in one chair in the room he would get into another.

Mental symptoms came on after the dermatitis manifested itself, and then I made a diagnosis of pellagra. Since the dermatitis and constipation have become more obstinate, the patient has great difficulty in moving his bowels. This is particularly the case when the dermatitis is most severe. In the spring the dermatitis appeared, and this fall it appeared again at both times when constipation had been very obstinate.

Dr. B. R. TUCKER, Richmond, Virginia: In regard to those cases of pellagra that have occurred and are occurring outside of institutions, in some of them the mental changes have come on early, while in others they have appeared late. Is it true that the cases of dementia which have developed pellagra have exacerbations of their mental symptoms at the onset of the disease? I have the histories of nine cases and in eight of them excessive crying was noted as a symptom, showing an emotional state. I would like to ask whether any other members of the conference have observed this symptom?

COMPLEMENT FIXATION WITH LECITHIN AS ANTIGEN IN PELLAGRA—FURTHER OBSERVATIONS

C. C. BASS, M. D.

NEW ORLEANS.

In a preliminary note published in the Journal of the American Medical Association, October 9, 1909, I reported a positive complement fixation reaction with lecithin as antigen in six consecutive cases of pellagra. One of these has since been found to have had syphilis and would probably have given a positive reaction from this cause. Another one of the tests was made on blood taken at autopsy twenty-four hours after death, and is therefore not to be credited fully. There remained, however, four cases that gave a positive reaction without any apparent cause except the presence of pellagra. Since that publication I have tested the blood of ten other cases of pellagra for this reaction, and wish to report them here. In these ten cases, six were positive and four were negative. Of the six positive cases one was known to have had syphilis and would probably have given a positive reaction without the presence of pellagra. In the tabulation below it will be convenient to include the former six cases.

Case No.	Type of Disease.	Reaction.
4	Chronic case, 4 years' duration; severe acute attack; insanity and death	Positive
5	Mild acute case; first year, improved	Positive
6	Severe acute case; death, had had syphilis	Positive
7	Severe acute case; death, blood taken 24 hours after death	Positive
8	Mild chronic case, severe skin lesions; improved	Positive
11	Moderately severe case; also had T. B., which contributed to her death	Positive
12	Severe first attack; death in one month	Negative
13	Severe case; diarrhœa 1 1-2 years; erythema 10 days	Negative
14	Severe case; diarrhœa and vaginitis two years; erythema two months; had had syphilis	Positive
18	Mild chronic case; improving	Positive
20	Very severe case; death	Positive
21	Mild case; first (?) year	Positive
22	Moderately severe, acute attack; three or four summers erythema; three months' emaciation; diarrhœa and indigestion 27 years	Positive
23	Moderately severe case first summer; estivo-autumnal plasmodia in blood; great anemia	Positive
24	Three years' diarrhœa; severe mental symptoms now; erythema pretty well cleared up	Negative
25	Severe case, diagnosed by Dr. Lavinder	Negative

Technic.—It will not be necessary to describe the technic in detail, as it is the Wasserman serum reaction for syphilis, with slight modifications to suit my own convenience, and substituting as antigen lecithin for syphilitic liver extract. The hemolytic system used was

sheep blood corpuscles, guinea pig complement and sensitive rabbit serum amboceptor.

The hemolytic unit used in all tests was 1/20 c. c. of sheep corpuscles. The lecithin solution used for antigen is a 0.3 per cent. solution in equal parts absolute alcohol and salt solution. One-tenth c. c. of this per hemolytic unit was quantity used. One-tenth c. c. patient's serum per hemolytic unit must bind the unit of complement or the test is considered negative. All except five of the tests here tabulated were made with inactivated serum. All the tests were controlled by running through at the same time a normal negative serum and also a reacting syphilis blood exactly as is usually done in making Wasserman's reaction for syphilis.

An analysis of the sixteen cases shows that two have had syphilis, one was done on old autopsy blood, and another had estivo-autumnal plasmodia in the blood when the test was made. Excluding these possible sources of error, we still have eight out of twelve cases giving a positive reaction. Of these eight positive cases seven were of the mild or chronic type and only one was of the severe acute type. Of the four negative cases all had severe acute attacks and two had their first attacks. Two of these are alive but do not promise to recover from the present attack. The reaction seems more likely to be present in chronic mild cases and those showing some resistance to the disease which is in keeping with the fact that the complement fixation reaction is due to the presence of antibodies for lipoid substances.

The observations here reported are on far too few cases to permit final conclusion. They should be confirmed by study of a much larger number of cases by competent observers.

The complement fixation reaction with lipoid substances as antigen has been found in syphilis especially, but also in trypanosomiasis, sleeping sickness, kala-azar, certain cases of malaria, a few cases of scarlet fever, and probably other diseases. All of these, except possibly scarlet fever, are protozoan diseases. The reaction has not been found in bacterial diseases except in rare instances.

At the suggestion of Dr. Dock the strength of the reaction was determined in three positive cases. One-tenth c. c. serum in one case fixed two units of complement; another fixed four units; and one, a case of two years' duration, fixed twenty units of complement.

I am indebted to the Charity Hospital staff and to many physicians of New Orleans and vicinity for courtesies shown.

741 Carondelet Street.

THE WASSERMANN REACTION (NOGUCHI MODIFICATION) IN PELLAGRA—REPORT OF THIRTY CASES

Report of Thirty Cases.

DR. HOWARD FOX

NEW YORK CITY.

In a recent communication (Jour. A. M. A., 1909, p. 1187), Dr. C. C. Bass, of Tulane University, reports that he has obtained six positive Wasserman reactions in six cases of pellagra, using lecithin as antigen. He suggests that these results if confirmed may "tend to strengthen the idea that the disease is of protozoan origin." He further states that it would add another disease to be considered in interpreting a positive Wassermann reaction. It is with the purpose of continuing these researches that the writer has come to the State where so many cases of pellagra have been recognized. Owing to the kindness of Dr. J. W. Babcock and Surgeon-General Wyman, free access to a most unusual material has been obtained.

Thirty cases of pellagra have been tested by the Noguchi modication of the Wessermann reaction. The writer would have preferred, as has been his custom, to have performed both the regular Wassermann and Noguchi tests simultaneously. Owing to the limited amount of time available, it was only possible to employ the more convenient modification of Noguchi. The writer feels convinced, however, that the Noguchi test is fully as accurate as the original method of Wassermann.

The cases examined included eight white and twenty colored women, one white man and one colored boy. All of the patients were from South Carolina, and with the exception of Case 1, were inmates of the State Hospital for the Insane. All of the cases, with perhaps one exception, have shown unmistakable symptoms of pellagra, though at the time of examination some did not present very active symptoms of the disease. The cases which were chosen for examination were those which apparently showed no evidence of syphilis. To have excluded syphilis from the patient's history would have been difficult or impossible from the nature of the cases.

The technique was that described by Noguchi and by the writer in previous communications (N. Y. Med. Record, March 13, '09, and Jour. Cutaneous Diseases, Aug., '09). The materials used

included 0.04 c. c. of fresh guinea pig serum, a weak suspension of human corpuscles (preferably washed) in the proportion of one drop to 4 c. c. of physiological salt solution, one capillary drop of patient's serum (active) and the antigen and amboceptor in paper form. The tubes were incubated for one-half hour for the first and two hours for the second period, after which the results were read. Two different antigens were used in testing every case. One of these consisted of an extract of syphilitic liver; the other (especially prepared by Dr. Noguchi for the present investigation) was a composite extract of syphilitic liver and normal hearts and kidneys. Both had previously been tested by Dr. Noguchi and found to be entirely satisfactory.

In performing the reaction, a known negative serum and one or more known positive sera were always used for comparison. The positive sera included five cases of syphilis and two of leprosy which the writer had previously tested and found to be strongly positive. The entire series of thirty cases was tested four times. With the exception of one case, no strongly marked positive reactions were obtained. In this case it was later found that a previous syphilitic infection was quite probable. In two other cases there was a positive reaction of moderate intensity and in five cases the reaction was only weakly positive. Even in the cases giving a moderate positive reaction the inhibition of hæmolysis was far from being complete and was very easy to distinguish from the marked reaction given by the syphilitic and leprous sera.

While positive reactions are at times given in apparently non-syphilitic cases there appears to be only one disease, namely, leprosy, in which a strong positive reaction is a frequent occurrence. In an examination of fifteen cases of leprosy during the past few months in New York the writer found twelve positive reactions, many of them being very intense. Somewhat similar results have previously been obtained by other observers. The writer feels confident that pellagra will not prove to be a disease in which a positive Wassermann reaction will be frequently found. If such a sensitive test as that of Noguchi (and the objection is sometimes made that it is too sensitive) fails to show many positive reactions, it does not seem probable that they will be obtained by the regular Wassermann method.

CASES.

Case 1.—G. S., boy, colored, 12 years old—symptoms of pellagra first noticed five years ago. At present there is an extensive erup-

tion of face, neck and back of hands, wrists, elbows and legs. There is constant salivation, distressing thirst, red tongue, severe uncontrollable diarrhœa, spastic gait, greatly increased reflexes, unequal pupils. There are frequent tonic, muscular spasms drawing the body to the left side. Patient is greatly emaciated. Mental condition is not affected. Reaction: Weakly positive.

Case 2.—H. M., woman, colored, about 50 years old—manic depressive insanity. First attack of pellagra four years ago. Fairly well till one month ago, when symptoms recurred. At present eruption on face and hands. Diarrhœa. Knee jerk absent. Reaction: Negative.

Case 3.—F. W., woman, colored, about 28 years old. Manic depressive insanity with pellagra. Symptoms of pellagra first noticed three weeks ago. At present red tongue, diarrhœa, erythema of backs of hands. Knee jerk increased. Reaction: Negative.

Case 4.—H. J., woman, colored, about 28 years old—manic depressive insanity with acute pellagra. First symptoms of pellagra noticed ten months ago. At present characteristic eruption of hands, feet and neck—salivation. Reaction: Negative.

Case 5.—M. H., woman, about 45 years old—epilepsy. First symptoms of pellagra developed six weeks ago. At present marked characteristic eruption of hands and neck, diarrhœa, exaggerated knee jerk. Reaction: Moderately positive.

Case 6.—M. L., woman, colored, about 50 years old—manic depressive insanity with pellagra. First symptoms of pellagra noticed six weeks ago. Characteristic eruption of hands and neck, diarrhœa. Knee jerk abolished. Reaction: Negative.

Case 7.—M. H., woman, colored, about 35 years old—manic depressive insanity with pellagra. First symptoms of pellagra noticed three weeks ago. At present characteristic eruption of hands and neck, diarrhœa, red tongue. Knee jerk abolished. Reaction: Moderately positive.

Case 8.—L. L., woman, colored, 18 years old—pellagrous insanity. Admitted with symptoms of pellagra three months ago. At present eruption of hands, elbows and legs. Has attacks of rigidity of dorsal muscles—tongue rough, with elevated papillæ—diarrhœa. Knee jerk much exaggerated. Reaction: Negative.

Case 9.—E. P., woman, colored, about 25 years old—paresis with pellagra. First symptoms of pellagra six weeks ago—tongue slightly checkerboard. Knee jerk abolished. Reaction: Weakly positive.

Case 10.—R. A., woman, colored, 43 years old—pellagrous insanity suggesting alliance with paresis. Patient admitted one month ago with characteristic eruption on hands and feet—diarrhœa. Tongue fissured (checkerboard). Knee jerk abolished. Reaction: Negative.

Case 11.—J. B., woman, colored, about 60 years old—pellagrous insanity. First symptoms of pellagra one year ago. At present nearly well. Exaggerated knee jerk. Bronzing of exposed surfaces. Reaction: Negative.

Case 12.—S. K., woman, colored, about 25 years old—pellagrous insanity. Admitted to hospital eight months ago. Hands bronzed—marked salivation. Knee jerk abolished. Reaction: Negative.

Case 13.—M. W., woman, colored, about 30 years old—manic depressive insanity with pellagra. First symptoms of pellagra noticed four months ago. Characteristic eruption of hands and neck. Knee jerk controlled. Reaction: Negative.

Case 14.—C. M. B., woman, colored, about 40 years old—manic depressive insanity with pellagra. First symptoms of pellagra noticed one month ago. Characteristic eruption on hands and neck. Reflexes exaggerated. Reaction: negative.

Case 15.—H. C., woman, colored, 35 years old—pellagrous insanity. Admitted to hospital two months ago with symptoms of insanity. Tongue fissured and slimy. Nails slightly clubbed—elbows slightly pigmented. Knee jerks normal. Reaction: Negative.

Case 16.—J. C., woman, colored, about 45 years old—pellagrous insanity—melancholia one year ago. Eruption first noticed during past summer. At present typical eruption on hands and neck—pigmented tongue. Knee jerk abolished. Reaction: Negative.

Case 17.—J. T., woman, colored, about 35 years old—pellagrous insanity. First symptoms noticed one year ago. At present eruption on hands. Lips exfoliated. Knee jerk brisk. Reaction: Negative.

Case 18.—L. M., woman, colored, 28 years old—pellagrous insanity. History of attack one year ago. Present attack began one month ago. Eruption on face, hands and feet. Severe diarrhœa—red tongue. Knee jerk slightly exaggerated. Confined to bed. Reaction: Weakly positive.

Case 19.—M. P., woman, colored, about 50 years old—clinical diagnosis of paresis on admission nine months ago. Symptoms of pellagra first noticed two months ago. At present eruption of

hands and feet—broad slimy tongue. Some ptyalism. Diarrhœa. Confined to bed recently. Reaction: Negative.

Case 20.—T. W., woman, colored, about 30 years old—manic depressive insanity with pellagra. First symptoms of pellagra noticed one year ago. Depression, diarrhœa, pigmented tongue. Knee jerk abolished. Reaction: Negative.

Case 21.—D. H., woman, colored, 30 years old, married—pellagrous insanity. History of attack one year ago. At present few symptoms. Reflexes exaggerated. Reaction: Negative.

Case 22.—L. B., woman, white, 32 years old, married—pellagrous insanity of manic depressive type. Has run a typical course with eruption, stomatitis and diarrhœa. Duration of disease two years. At present mental instability—erythema of back of hands. Reaction: Weakly positive.

Case 23.—M. H., woman, white, 28 years old, married—pellagrous insanity with suicidal tendency. Has run a typical course of pellagra with predominance of mental symptoms. Disease associated with pregnancy. Duration less than one year. At present no eruption. Reaction: Negative.

Case 24.—M. O., woman, white, 36 years old, single—pellagra engrafted on hallucinatory insanity. Has run typical course with marked anemia. Repeated attacks of pellagra several times a year. Duration of pellagra two years. At present diarrhœa, anæmia, erythema of backs of hands. Reaction: Weakly positive.

Case 25.—M. W., woman, white, about 50 years old, married—manic depressive insanity of long duration. Pellagra has developed within the last year and has run typical course. At present marked diarrhœa, eruption, stomatitis, depression. Reaction: Negative.

Case 26.—B. G., woman, white, 35 years old, married—pellagrous insanity. Duration of disease four months. At present marked, very slight eruption, anæmia, diarrhœa. Reaction: Negative.

Case 27.—F. R., woman, white, 36 years old, single—pellagrous insanity. Duration one year. At present anæmia, depression, diarrhœa, eruption. Patient emaciated. Reaction: Negative.

Case 28.—L. B., woman, white, 45 years old, single—Moral imbecility with pellagra. Duration of pellagra two years. At present tongue swollen and indented by teeth. Stomatitis, diarrhœa, marked emaciation—eruption on back of hands. Reaction: Negative.

Case 29.—M. B., woman, white, about 40 years old, married—pellagrous insanity. Duration of disease six months. At present

anæmia, diarrhœa, marked emaciation—very slight eruption on back of hands. Reaction: Moderately positive.

Case 30.—S. C. L., man, white, about 35 years old—pellagrous insanity. Duration of disease two years. At present delusions (suicidal), stomatitis, uncontrollable diarrhœa. Reaction: Negative.

The writer desires to express his heartfelt thanks to Dr. J. W. Babcock, Superintendent of the State Hospital for the Insane, and to Dr. Walter Wyman, Surgeon-General, U. S. Public Health and Marine-Hospital Service, for permission to examine the cases. He is also indebted for laboratory facilities to Dr. C. F. Williams, Secretary of the State Board of Health, and Dr. F. A. Coward, Director of the Laboratory. For Case No. 1 the writer wishes to thank Dr. J. J. Watson.

CONCLUSIONS.

1. Cases of pellagra do not often give a positive Wassermann reaction.

2. A positive reaction, when obtained, is generally weak and is easily distinguished from the strong reactions found in syphilis and in many cases of leprosy.

3. The value of the Wassermann test is not affected by the findings in pellagra.

TRANSFUSION IN PELLAGRA

H. P. COLE, M. D., AND GILMAN J. WINTHROP, M. D.

MOBILE, ALA.

The existing uncertainty as to the etiology and as to the toxin or toxins producing the symptoms of pellagra, prohibit, at present, the formulating of a scientific or rational therapy. Any remedial or surgical measures employed must necessarily be based on empiricism and experimentation.

Study on the etiology of the disease, chiefly by Italian and German workers and more recently in this country, has led to three chief theories as to the cause of pellagra. Firstly, the maize or "zeist" theory, which holds that decomposition of the oils or "zein" of corn produces the poisons causing pellagra. Secondly, the "verdet" theory of Lombroso, which claims that the growth of certain fungi, penicilli and aspergilli on maize produces a toxin. The ingestion of this toxin containing corn produces pellagra. Thirdly, the microbic or bacterial theory, suggested by Tizzoni, Panichi and others, which supposes that pellagra is due to a specific bacterium which grows on maize and elaborates characteristic toxins.

That maize, especially when exposed to moisture and heat, bears a close causative relation to pellagra is rather generally conceded. Whether the toxic principles are developed in the corn itself or whether maize is pathogenic only in so far that poisons can develop in it by the action of micro-organisms is still a matter of speculation.

A discussion as to the theories of the etiology of pellagra is beyond the scope of this paper. We shall confine ourselves to a consideration of the theories, experiments and facts bearing on the nature and action of the toxic substances of pellagra, no matter what be their origin, and suggest the possibility of combatting these toxins in vivo by antagonistic substances formed in the body by natural or acquired immunity.

Granting, as seems justifiable, that pellagra is an intoxication, let us consider the work done as to the biologic actions of the toxins.

Tizzoni (1) has shown that by injecting the blood of a pellagrous patient into animals typical symptoms of the disease are produced. This may be explained by the passage of toxins from the affected to

the non-affected animal, or, as Tizzoni suggests, to the transmission of living (toxin-bearing?) bacteria specific of the disease.

Lombroso (2) claims to have produced typical pellagrous manifestations by the administration of an alcoholic extract of impure maize ("pellagrozein").

These experiments seemingly prove the existence of a toxic principle capable of producing pellagrous symptoms, laying aside any consideration as to their chemical, microbic or fungus origin.

Admitting, then, the existence of specific toxins, may we not rationally consider the probability that the body tissues form anti-bodies or anti-toxins to combat their action? Much of the late work on sera and toxins tends to answer this supposition in the affirmative.

Babes (3) and other workers have found in the serum of pellagrins a substance antagonistic to the extracts of damaged maize that is a specific anti-toxic body.

Giovanni and Gatti (4), from their work on pellagrous serum, conclude that the serum of pellagrins has a hemolytic action much above normal, demonstrating the presence of an anti-body. They further claim to have demonstrated precipitative properties in pellagrous blood.

D'Ormea (5), working independently, likewise concludes that there is a definite specific anti-body developed in pellagrins.

Antonini and Marianni (6) claim that a definite immunity is developed in cured cases of pellagra and that the serum exhibits a definite anti-toxic action against maize poisons. They have developed an artificial immunity in rats, rabbits and goats against pellagrous toxins. Finally, they conclude that a serum therapy can be instituted in grave pellagra cases.

Accepting the statements of these workers, we are led to the following conclusions:

(1.) Pellagra is an intoxication.

(2.) The toxic principles of pellagra exist in the blood of pellagrins and will produce pellagrous symptoms when transferred to other animals.

(3.) Pellagrous serum exhibits definite precipitative, hemolytic and anti-toxic properties.

(4.) An artificial immunity can be produced in animals and exists in cured pellagrins.

Our work in pellagra was undertaken independently of any knowledge of the researches of others as to the existence of anti-bodies in pellagra. Being interested in the subject of transfusion and working

on its effect in shock and hemorrhagic anemia in dogs, we were led to try transfusion in pellagra, at first, simply as a relief for the existing anemia in these cases. The possibility of the existence of a specific curative agent of the nature of an anti-toxin, performed in cured cases and which might be transfused with the blood, very naturally suggested itself.

Through the kindness of Drs. McCafferty and Tisdale, of the Mt. Vernon Hospital, we were enabled to perform a transfusion in a severe case of pellagra, using a cured case of pellagra as the donor. As far as we are able to ascertain, this is the first transfusion performed in pellagra. (7.) The results of this first transfusion were quite suggestive and led to further experimentation. The results and conclusions of some nine cases of pellagra so far transfused are noted in the second part of this paper.

The difficulty of securing cured cases as donors has forced us to employ normal, healthy individuals in a number of our transfusions. We have chosen these non-pellagrous donors from among persons living in the same suroundings, eating the same food and subject to the same chances of infection as the patients themselves. It is possible that these donors have developed a certain amount of immunity and contain anti-toxic bodies in their blood. Whether these antibodies be present or not, the amount of blood furnished on transfusion must, at least, combat the existing anemia and stimulate the protective and recuperative activities of the recipient.

TRANSFUSION IN PELLAGRA.

The number of cases of pellagra so far treated by serum injection and direct transfusion of blood is too small to permit of final deductions as to their therapeutic value in the disease.

We attempt to give below a resumé of the cases in which serum therapy and blood transfusion have been employed. The data for this report has been obtained from monographs and personal communication with the various workers.

The curative value of pellagrous serum has been chiefly tried by the Italians. Antonini and Marianni (8), using the serum of recently recovered typhoid types of pellagra, note cures in several severe cases. Their work was most carefully performed and the potency of the serum was tested on animals before being administered to patients. Lombroso (11), in a personal communication to the authors of this paper, comments on the work of Antonini and Mari-

anni, and states that the use of serum is of undoubted value in the treatment of pellagra.

Dr. Lavinder, of the Marine Hospital and Public Health Service, has employed serum in the treatment of two cases of pellagra. The first case received but one injection of serum and died within three days of an intercurrent pneumonia. The second case received four injections, and at first seemed to improve, but this was not continuous, and at the last report the patient was "steadily losing ground."

The belief of the Italian workers in the efficiency of pellagrous serum will, we hope, lead to a more extensive trial of this most simple therapeutic measure and so furnish information as to its true value.

Besides our nine cases of direct blood transfusion in pellagra, which are given in detail below, we wish to note two other cases. Drs. Wood and Green (9) of Wilmington, N. C., transfused a case of three years' duration. This case, which they state was in a "hopeless condition," received about six to eight ounces of blood on transfusion. No apparent improvement was noted and the patient died.

The second transfusion to be noted was performed by Drs. McCafferty and Tisdale, of the Mount Vernon Hospital, Mt. Vernon, Ala. The patient was transfused from a cured case of pellagra and received approximately a pint of blood. Five days after transfusion the skin lesions began to disappear, and in fourteen days the patient was up and walking about.

TECHNIC.

The following cases were transfused by the canula method of Crile or by the suture method of Carrel:

REPORT OF CASES.

Case 1.—Alice F., age 25; colored. Referred by Dr. McCafferty and Dr. Tisdale, Mt. Vernon, Ala.

This patient has had pellagra for three weeks and presents, on examination, the characteristic skin lesions over the hands, arms, face and legs. There is a stomatitis so severe as to seriously interfere with nourishment, incontinence of the bowels, marked emaciation and asthenia. Hemoglobin, 70 per cent.

The patient is in a moribund condition.

Transfusion—August 3, 1908. The donor is a well nourished negro woman who recovered from a severe attack of pellagra one

year ago. There was a good transfer of blood for about twenty minutes.

August 4, 1908. Twenty-four hours after the operation the recipient shows marked signs of improvement, both in her mental and general condition.

August 7, 1908. Four days after the transfusion the patient has improved markedly and is walking about the ward.

This patient went on to rapid recovery. She has presented no symptoms of the disease since the operation, performed fourteen months ago. The recovery in this case was so immediate and complete as to be extremely suggestive.

Case 2.—Bessie B., age 22; white. Referred by D. D. Armstead, Campbell, Ala.

For the past four years this patient has had attacks of sore mouth, diarrhœa and indigestion, beginning in the spring and lasting about six months each. During these attacks the patient was confined to her bed. About fourteen months ago a red eruption appeared over the backs of both hands and wrists; this was never painful and disappeared in a few weeks. The patient has had sore mouth ever since the eruption appeared, and there have been four or five soft bowel movements a day for the past four months. For several weeks there have been marked mental and nervous symptoms; these are increasing in severity. Sensory symptoms have been present in the feet and head. In the past eight days there has appeared an erythema over the dorsum of each hand, this has extended well up on the forearms, has become pigmented and has been associated with burning and pain. Coincident with this there has been an exacerbation of the mental and nervous symptoms. There has developed a pulse rate out of proportion to the temperature.

On examination the patient presents marked emaciation and asthenia. Weight, 60 pounds; average weight, 100 pounds. Hemoglobin, 75 per cent.

There is a dark, dry, pigmented eruption on the backs of both hands extending well up on the backs of both forearms. There are marked mental and nervous symptoms. Muscular reflexes are increased.

Transfusion—July 3, 1909. The donor is a strong, healthy male adult, a brother of the patient. The donor has never had pellagra. There was a good transfer of blood for thirty minutes. The donor had an attack of syncope upon leaving the operating room.

July 4th, twenty-four hours after the operation, there are marked signs of improvement.

July 7th, four days after the operation, the skin lesions began to desquamate.

The marked nervous and mental symptoms began to rapidly disappear and were almost entirely absent within a week from the day of transfusion. The sore mouth disappeared within four days, the bowel movements were less frequent after the fourth day, and entirely disappeared within three weeks.

July 13th, ten days after the operation, the patient weighs 68 1-2 pounds, a gain of 8 1-2 pounds.

The patient returned home and has steadily improved. Three months after the operation she states that she is stronger and has gained about 15 pounds; that there are neither eruption, diarrhœa nor mental symptoms.

This patient has made a gradual improvement from the day of operation, and will eventually recover entirely.

Case 3.—Annie H., age 42; white. Referred by Dr. P. A. Trice, Morvin, Ala.

One year ago this patient had an attack of diarrhœa of six months' duration; there is also an indefinite history of an erythema over the backs of both hands at this time. There was marked asthenia, anemia and emaciation during the attack. There is a history of nervous excitability but no mental disturbances. There is no history of sore mouth.

On examination the patient shows indefinite signs of an old desquamative skin lesion over the backs of both hands, increased reflexes, asthenia, nervous excitability and a marked grade of anemia. There is considerable emaciation, the patient stating that she has lost thirty or forty pounds in weight.

Transfusion—July 18, 1909. The donor is a healthy adult, male (patient's husband).

There was a good transfer of blood for twenty minutes.

July 21st, three days after the operation, the patient shows definite signs of improvement.

Twelve weeks after the operation her physician writes that her physical condition is better; that she has gained fifteen pounds in weight; that her anemia is much improved. The patient herself says that she is stronger than she has been for the past three years.

Case 4.—Mrs. T., age 27; white. Referred by Dr. Bailey, Demopolis, Ala.

This patient developed an erythema over the backs of both hands six weeks ago; this extended to the forearms, elbows, sides of the neck and upon the face. This has been associated with a marked stomatitis and nausea in the past three days. The stomatitis has been so severe in the past few days as to seriously interfere with feeding. There has been a profuse diarrhœa and an increasing asthenia, anemia and emaciation. The nervous symptoms have been pronounced in the last few days.

On examination the patient is found to be in a practically moribund condition; an ulcerating and deeply pigmented eruption covers the dorsal surfaces of both hands, forearms and the sides of the neck. There is a deeply pigmented mask over the entire face. There is ulceration of the mouth, a swollen, blackened tongue protrudes from the parched and ulcerated lips, from which there constantly drools forth a fetid, slimy discharge. This pigmentation extends over the legs and feet as well.

The patient is markedly emaciated and presents a terminal stage of anemia. The reflexes are almost absent; there is incontinence, marked anemia and frequent emesis. Pulse 150-160.

Transfusion—July 11, 1909. Two donors. The first donor is a white adult female who recovered a few weeks ago from a severe attack of pellagra. A good transfer of blood was obtained for ten minutes, when the transfusion was discontinued on account of the weakened condition of the donor.

A second transfusion was performed from a healthy adult male (the patient's husband), who has never had pellagra. A good transfer of blood was obtained for fifteen minutes. The transfusion was then discontinued on account of the weakened condition of the recipient.

This patient was in a hopeless condition at the time of operation and showed no signs of improvement after the operation. The patient died three hours after the operation.

Case 5.—Mrs. B., age 58; white. Referred by Dr. Bondurant, Mobile, Ala.

This patient was treated by her family physician, Dr. O. G. Bruner, of Fort Deposit, Ala., for a persistent case of diarrhœa extending over a period of two years. One year ago there appeared an eruption over the backs of both hands, this was followed by desquamation. This eruption reappeared in the spring of 1909, about six months before transfusion, and a few weeks before transfusion the patient developed marked mental and nervous symptoms.

On examination there are remains of a desquamating skin lesion over both hands and forearms. There are constant involuntary muscle tremors, increased reflexes and the mental condition is that of constant delusions, and at times a muttering delirium. There is asthenia and marked emaciation; weight, 70 pounds; average weight, 100 pounds.

Transfusion—July 17, 1909. The donor is a healthy adult male (the patient's son), who has never had pellagra. There was a good transfer of blood for twenty-five minutes.

July 18. Twenty-four hours after the operation the nervous and mental condition is improved. This patient gained eight and one-half pounds in the first week and went on to a rapid recovery. Eleven weeks after the operation the son writes that the mental condition seems perfect; there are no symptoms of pellagra and there has been a gain of thirty-four pounds in weight.

Case 6.—Mrs. H., age 50; white. Referred by Dr. Sarah A. Castle, Meridian, Miss.

This patient has had attacks of diarrhœa, associated with sore mouth, for the past two years. Four months ago the patient had an attack of diarrhœa of two weeks' duration, and this was followed by a stomatitis that has grown very severe and persists. An eruption appeared on the hands about four weeks ago and extended over the forearm, face and legs. There have been marked nervous and mental symptoms for the past six weeks. This condition has been that of acute delirium at times. There have been severe sensory and motor symptoms, and incontinence of the bowels, a condition present for about a week.

On examination there is found an almost hopeless anemia and emaciation. A desquamating skin lesion extends over the hands, arms, legs and face. There is a drooling, fetid discharge from the mouth, a marked stomatitis, incontinence of the bowels, and at times a low muttering delirium.

Transfusion—July 22, 1909. The donor is an arteriosclerotic, plethoric, male (the patient's husband), who has never had pellagra; age, about 60. The anastamosis was accomplished with a great deal of difficulty because of the extreme degree of atheroma present in the donor's radial artery. There was only a very small amount of blood transferred.

Transfusion—August 17, 1909. This patient made no definite signs of improvement and was again transfused twenty-six days after

the first attempt. At this time she was in the same physical condition, except for a much more severe anemia.

The donor is a female, age 18 (the patient's daughter). This donor recovered from a definite attack of pellagra two years ago. There was a good transfer of blood for twenty-five minutes.

August 18, 1909, twenty-four hours after the operation, there is an aggravation of the mental symptoms.

August 23, 1909. One week after the operation the patient has shown no definite signs of improvement, except that the stomatitis has cleared up to some extent. Death occurred four weeks after transfusion.

Case 7.—Mr. G., age 30; white. Referred by Dr. J. L. Bryan, Greenville, Ala.

Six months ago this patient developed an erythema, followed by a pigmentation over the backs of both hands and forearms. During this period there has developed a severe diarrhœa, mental deterioration and an extreme degree of emaciation.

On examination this patient presents marked asthenia and anemia. There are pronounced motor and sensory disturbances, a desquamating skin lesion still presents over the backs of both hands and forearms. The sore mouth and diarrhœa, while present, are not severe at this time.

Transfusion—July 25, 1909. The donor is a 14-year-old boy (the patient's nephew), *who has never had pellagra.* Because of the extreme restlessness of the youth only a small amount of blood was transfused in the course of twenty minutes.

July 26th, twenty-four hours after the transfusion, the patient shows no signs of improvement.

This patient became steadily worse and died August 20, 1909, twenty-six days after the transfusion.

This case was not transfused with any appreciable amount of blood.

Case 8.—Mrs. S. H., age 30; white. Referred by Dr. Armstead, Nanafalia, Ala.

This patient had a severe attack of diarrhœa about one year ago, associated with sore mouth and marked nervous and mental symptoms—duration, two months. There were no skin lesions at this time.

The present attack began three weeks ago with an erythema on the backs of the hands and wrists. This went on to the induration, pigmentation and desquamation characteristic of the disease. This attack is associated with a severe stomatitis. During this attack the

patient has had marked nervous and mental symptoms and has become emaciated and anemic.

On examination there are the remains of a desquamating skin lesion over the dorsum of the hands, wrists, forearms and elbows. There is stomatitis and a mild grade of diarrhœa. The patient has lost about forty pounds in weight.

Transfusion—July 25, 1909. The donor is a healthy adult male (the patient's husband), who has never had pellagra. There was an excellent transfer of blood for fifteen minutes, when the operation was discontinued, as the recipient showed signs of cocainism.

July 26th, twenty-four hours after the operation, the patient shows an improvement in her mental and nervous condition.

Ten weeks after the operation her physician writes that the patient has improved wonderfully since the operation. There is no skin lesion, no sore mouth, no diarrhœa. The mental and nervous symptoms are much improved. The patient says that she feels better than she has in five years. She has gained about twenty pounds in weight and "feels 100 per cent. better than before the operation," according to her physician's statement.

Case 9.—Mrs. M., age 36; white. Referred by Dr. McMillan.

This patient has had attacks of severe diarrhœa every spring for six or seven years. These attacks were of about one month's duration and cleared up without medical treatment.

Eighteen months ago the patient developed, for the first time, an eruption over the backs of the hands. This eruption lasted about a month and went on to desquamation without pigmentation. At this time the patient developed a sore mouth and salivation, which was present about one month. The patient had another attack of diarrhœa about four months ago; at this time she was anæmic, emaciated and showed marked nervous symptoms.

Four weeks ago sore mouth again appeared, associated with diarrhœa, anæmia, emaciation, asthenia and an erythema, which extended over the backs of both hands well up on the forearms. This eruption became pigmented and desquamated. At this time the nervous symptoms were aggravated and there developed a mild grade of dementia.

An interesting point in this case is that there was a suspicion of diabetes for several years, and the patient was placed on a diet consisting largely of corn bread.

On examination she presents the remains of a desquamating skin lesion over the dorsal surfaces of both arms and hands. There is asthenia, emaciation and anemia. Hemoglobin, 70 per cent. Weight,

74 pounds; average weight, 100 pounds. There is a profuse diarrhœa. The mental condition at times is that of a mild dementia.

Transfusion—October 18, 1909. The donor is a healthy adult, male (the patient's husband), who has never had pellagra. There was a good transfer of blood for thirty minutes.

October 19th, twenty-four hours after the operation, there is a distinct improvement in the mental condition.

October 25th, one week after the operation, the patient has improved markedly in general condition; the mental symptoms have disappeared, the appetite is much improved, and the patient has gained seven and one-half pounds in weight.

TRANSFUSION IN PELLAGRA.

It must be noted that the only medicinal agents used in the recovered cases were tonics of strychnine, forced feeding, and in some cases carbonate of iron.

In every case benefited by transfusion the improvement was immediate, as shown by marked gain in weight within the first week—in one instance, as much as eight and one-half pounds. All the patients, two months after transfusion, are either apparently cured or markedly improved, and have gained from five to thiry-five pounds in weight.

Of the fatal cases, case No. (4) was moribund at the time of operation, and died three hours after transfusion; case No. (7) reecived no appreciable amount of blood on transfusion; case No. (6) received practically no blood at the first operation, and at the second transfusion, when a pellagrous donor was used, while she received a good flow of blood, her condition, we felt, was hopeless.

We suggest the following conclusions:

(1.) Transfusion offers a means of combatting the anemia, stimulating the recuperative functions and perhaps of furnishing antitoxic substances to pellagrins.

(2.) The lessened mortality and marked improvement in transfused pellagrins leads us to anticipate the establishment of a serum therapy in the disease.

(3.) Transfusion may be offered as a surgical therapeutic procedure in pellagrous cases pending the perfection of a successful serum therapy.

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DISCUSSIONS ON PAPERS OF DRS. BASS, FOX AND COLE.

DR. J. H. TAYLOR, Columbia, South Carolina: Regarding the use of serum in the treatment of pellagra, I wish to say that while actual transfusion according to Crile's method was first done by Dr. Cole, so far as I have read, yet two Italians, Antonini and Mariani, some years ago noted in their experiments on animals that: (1) Those poisoned by the toxines of corn acquired a certain degree of immunity against the toxic action of serum from a pellagrous patient, and also against new injections of toxic extracts of corn. (2) The toxic action of the serum from a pellagrin is neutralized by the simultaneous or successive injection of the serum of a cured pellagrin. (3) The addition of serum from the blood of a cured pellagrin in certain proportions to that from an active case of pellagra exercises a specific relieving antitoxic action. (4) The serum from the blood of a cured pellagrin exerts an antitoxic action when injected subcutaneously into cases of typhoid pellagra. Moreover, this action is in direct proportion to the degree of natural immunity acquired by the cured pellagrin, since the best results have been obtained by using the serum from a convalescing typhoid pellagrin, They demonstrated this last observation on four cases of typhoid pellagra, three of whom showed a prompt, marked and rapid improvement with ultimate recovery, and the fourth case began improvement, but owing to the donors passing from under their control no more serum could be obtained and the case died.

In view of these facts it would seem that actual transfusion of blood should hold out to us splendid possibilities.

DR. C. C. BASS, New Orleans, Louisiana: I wish to speak with reference to the paper of Dr. Fox and to further emphasize the fact that his experiments with complement fixation in pellagra differ materially from mine in that he used as antigen extract of syphilitic

liver and normal hearts and kidneys. These do not yield a strong solution of lecithin. My paper referred only to results with lecithin as antigen. When using the same sort of antigens as he did I got about the same results as he did. Complement fixation experiments I have done using many different antigens, including various extracts from different tissues and organs from pellagrins, as well as cultures isolated from patients and also from corn meal, have all been reserved for future reports and not considered worth while to include in the paper here read. The only explanation so far suggested to me for the fact that lecithin serves better for antigen in pellagra than organ extract is that because of the destructive process in the nervous system, a large amount of lecithin is set free and there may be formed a response to this increased lecithin content of the blood much anti-lecithin amboceptor.

Dr. W. B. Young, Rock Hill, S. C.: With reference to the paper of Dr. Cole I would like to know what preparation of iron was used and how it was given. Dr. Babcock used arsenite of iron hypodermically and did not obtain any results. We have been using it and it has seemingly improved the general condition. I have used it in six or seven cases and the condition of the patients have improved so far.

Dr. Fox (closing the discussion): I am very pleased to have heard from Dr. Bass, who has only obtained positive reactions when he used lecithin as antigen. I feel that our results are somewhat discordant because he has six positive reactions in six cases, and I only obtained seven reactions, most of them weak, in twenty-nine cases. As he states this reaction only occurs when he uses lecithin as antigen and it does not occur when he uses other extracts, including extracts of syphilitic organs, so that I feel that our results are not really as antagonistic as they might appear to be.

REPORT OF PELLAGRA AT NASHVILLE, TENN.

J. M. KING, B. S., M. D.

Professor of Dermatology, Vanderbilt University.

In presenting this paper I do not propose to discuss pellagra in a general way, but shall deal with the subject as observed at Nashville, Tenn.

There were two groups of patients, one occurring in the Baptist Orphanage, consisting of seventeen cases, the other, consisting of two cases, which were found in the country fourteen miles from the city, and one single case. In dealing with the orphanage, I wish to present as near as possible the order in time of the occurrence of the disease among the children. In the fall of 1905 a family of four children, cases Nos. 1, 2, 3 and 4, were admitted from Newport, Tenn., with the ages of 6, 8, 10 and 12 years. They came from most destitute circumstances. Very little of their past history could be obtained, either from their parents or their physician, who failed to answer any letter of inquiry. Upon admission the youngest child, Addie M., case No. 1, presented manifestations of some skin disease to which very little attention was paid. The children remained at the orphanage during the winter and the next spring this youngest child was allowed to play out in the yard in a low-necked dress and short sleeves. After a few days an erythematous eruption was observed on the exposed surface, the face, neck, shoulders, backs of the hands and feet. It was regarded as sunburn by those in charge. The eruption became more aggravated during the summer, but improved in the fall and winter. During the winter the child was away from the orphanage for a while, but was returned sick. In the spring the eruption on the skin reappeared and was associated from time to time with an uncontrollable diarrhœa, stomatitis and much emaciation. At this stage of the disease in this child, measles broke out in the institution, and it is reported that she contracted the disease and did not recover. The diagnosis of pellagra had not been made up to this time.

The next youngest child, in this family, Roy M., case No. 2, developed a skin eruption during the summer of 1907, rather mild in type at first. He passed through the winter and during the spring and summer months of 1908 the most aggravated pellagrous erup-

tion developed, erythematous, bullous; later, with thickened skin, cracked and very rough on the hands and feet. The eruption extended over the face, neck and shoulders, over the hands and forearms to the elbows, over the tops of the feet and up the legs to the knees. This case progressed and died in September, 1909. In June, 1908, is the time when I first examined the children, and the disease had further progressed in this boy than in any of the group then living, his younger sister being dead. His sister, case No. 3, Annie M., next older, and his brother, case No. 4, Jesse M., 14 years of age, had presented mild skin manifestations during the summer of 1908, the girl having an erythematous eruption on the face, hands and feet, while the boy had a mild erythematous eruption only on the feet. At this time, in June, 1908, when I first saw the cases and made a diagnosis of pellagra, twelve children were affected, showing the eruption on the hands, feet, and some on the face. As a group they had not suffered from any perceptible gastro-intestinal disturbances, except case No. 2, who had had a few spells of diarrhœa, and there were no nervous manifestations.

Case No. 5, Charley T., had the eruption, rather mild in form, over the face and neck and shoulders, on the backs of the hands, a little above the wrists, and on the tops of the feet to his knees, and in this respect he was as far advanced in the disease as case No. 2. He was a stronger boy, both mentally and physically, than case No. 2. At this time I outlined a general course of treatment which was carried out at the orphanage and at St. Thomas Hospital, the authorities of the orphanage allowing cases 2 and 5 to be taken there for treatment, rather as test cases. These two boys were given daily a warm bath with green soap, and anointed with vaseline and were not allowed to be exposed to the sun. They were given full diet and the medicinal treatment consisted of Fowler's Solution, one drop, and elixir of iron, quinine and strychnine—one-half a dram after meals. The eruption on both boys readily cleared up. They improved in appearance and after ten days they were apparently well, with the exception of very slight redness over some of the parts affected. Case No. 2 was troubled with diarrhœa while in the hospital. The two boys were returned to the orphanage, where they remained for several weeks during which time the eruption reappeared, but in a more mild form than at first. The children who had been treated at the home had also improved on the same line of treatment.

In the latter part of the fall of 1908 the management of the orphanage secured a separate building, the annex, and placed the affected children there for the winter and spring, in charge of a special attendant and under my medical care. The object of this segregation was to place the children in such surroundings that they could have more careful attention in the way of medical and hygienic treatment, and because the disease was thought at this time to be infectious or to be transmitted in some unknown way. While the food at the orphanage had at all times been excellent—really as good as that taken by the average individual—still we thought by giving a more nutritious diet they would be benefited. The medical treatment consisted of arsenic in small doses with iron, quinine and stychnine and lactic acid bacillus tablets, given after meals. Each child received a daily warm bath with green soap and the erythematous parts were kept covered night and day with an ointment of boric acid, lanolin and vaseline. During the winter months the eruption disappeared from all the children except case No. 2, who constantly showed some skin signs of the disease. None of the children developed any nervous symptoms or gastro-intestinal manifestations, except case No. 2, who gradually grew worse, his mental state becoming more impaired, and his gait more ataxic.

In the spring of 1909 two more children of the orphanage, eight and nine years of age, showed a very slight erythematous eruption and were sent to the annex. In the early spring case No. 5 ran away from the annex and returned to his home in Knoxville, Tenn. In the spring of 1909, as the warmer weather came on, case No. 2 grew much worse in every respect of the disease, and case No. 6, Charley M., developed a severe erythematous eruption over the lower part of the face, neck, the backs of the hands, and tops of the feet, with some fever. Case No. 2 went into such a hopeless condition that we thought he would not live longer than a few days. However, with liquid diet and no medicine, he slowly improved until he was able to walk, and lived about two months.

Case No. 3, Anna M., developed a mild erythema on the feet and legs. Jesse M., case No. 4, has shown no signs of the disease since the summer of 1908.

At this stage of the situation, about the middle of the summer of 1909, the authorities of the orphanage petitioned the county to take charge of the cases, and they were transferred to a home five miles from the city under the control of the county and the county health officer, Dr. B. G. Tucker, with whom, in consultation, I have seen

the children several times. Following the suggestion of Dr. Lavinder we put the entire group on daily salt rubs and gave salt internally.

At the county home case No. 2, Roy M., was placed in separate quarters from the other children, and with an adult case of pellagra in the last stages. The boy's condition at this time was extremely pitiable. He was pale, emaciated, extremely weak mentally, and his mouth so sore that he could scarcely take any kind of food, and was suffering with an uncontrollable diarrhœa. At times he grew delirious and would cry out for hours at the time. During his lucid moments he would say that he was suffering excrutiating pain from head to foot. During the last two months he had a marked coarse tremor in both hands, and a very slight ataxic gait. Death followed in a few days.

An autopsy was held by Prof. Wm. Litterer with the following report:

"Extreme emaciation and absence of adipose tissue. The external lesions had markedly improved from several months ago.

"The lungs (hypostatic), heart flabby and small, valves normal. Liver (enlarged slightly), spleen three times larger than normal and very dark and friable. Kidneys were slightly congested, normal in size and capsule stripped easily. The stomach was enlarged and very thin. In places it appeared as if it had no mucous membrane. No blood found in stomach. In the jejunm, ilcum, colon there was a marked thinning of the mucous membrane. Blood was found in the entire intestines. In a few areas the wall of the intestine was so thin that it gave the appearance of an erosion.

"In opening the spinal cord I was struck with the fragility of the bones. The spinal cord showed an inflammation and thickening of the dura mater.

MICROSCOPICAL.

"(1) Lungs are hypostatic, congested.

"(2) Heart muscle undergoing fatty degeneration in areas while in others an atrophic change has taken place.

"(3) Liver shows it to be cloudy swelling, fatty; also there is an excessive pigmentation of the cells of the liver.

"(4) Spleen shows a marked hyperplasia and cloudy swelling with some pigmentation of cells.

"(5) Stomach and intestines show a marked thinning of the mucous membrane with congestion in areas. (Further study will be given this.)

"(6) In the spinal cord is found a degeneration of the posterior column differing from that of tabes in showing no involvement of the posterior roots. There is also found in this case a degeneration of the cells in Clark's column; also changes in the large Betz cells of the ant. horn.

"Cultures were made from heart blood, liver and spleen with negative results.

"The Wasserman reaction was positive."

The next worse case, Charles M., No. 6, at this time in August, 1909, showed some eruption, had a slow fever, was stupid and would stay away from the other children. He was put on Donovan's Solution and fed with care. After a few weeks he was very much improved in appearance, the fever and eruption had disappeared, his appetite was good, and he was taking on weight. He grew more playful and would take part in the games with the children and was apparently well. I saw him on October 28th and observed that he had failed to a certain extent, he had lost weight, and expressed his feeling as if he was drunk or dizzy. Outside of this he was apparently well.

Case No. 3, Anna M., on October 28th, showed a very mild erythema on the sides of the neck and backs of the hands, which appeared upon slight exposure to the sun. With these two exceptions, the children as a group were apparently in good health.

I shall next discuss the second group consisting of a man and a woman who lived within a short distance of each other, in the country, fourteen miles from Nashville. The man, case No. 7, H. as he reported, had some eruption during the summer of 1908. He retired to his home in the winter, and there had no one to assist him in any way. He got his food the best he could, it being sent in usually by his neighbors. Having no one to do his laundry, the woman, case No. 8, attended to it for him, and in the spring of 1909 she developed the disease. These two cases were first seen by Dr. Lavinder in company with Drs. Tucker and Core, but both called to see me at different times, when I had full opportunity to investigate the situation. The eruption on the man was that of the usual type. He was very much broken in health, was very weak, emaciated, pale, with loss of appetite, sore mouth and an inveterate diarrhœa, conjunctivitis, and later corneal ulcers; there were no distinct nervous symptoms, except mental dullness. He was finally

placed in the county home, where he rapidly grew worse and died. He had had syphilis as he reported.

Case No. 8, Mrs. W., when first seen by me had failed very much in health; she was weak, had lost weight, and complained of a diarrhœa. There were no distinct nervous symptoms. She was not alert and active mentally. The eruption was on her face, neck, hands and feet. She was put on practically the same treatment that had been given the children. She improved and, as was reported to me by Dr. Tucker, the county health officer, on October 28th, was doing very well at that time.

Case No. 9, Mrs. H. H., a widow, was examined by me on October 22nd, with the opinion that the case was pellagra. The eruption started about the middle of March, 1909, previous to which she had been feeling very bad, as she expressed it. She said she had no ambition to do any work, and tired very easily. She was dizzy and described the feeling as if she needed calomel, and insisted upon her doctor giving it to her, although her bowels were constipated. After the administration of calomel, she had a terrific diarrhœa for four weeks, as she expressed it. The mucous membrane of her mouth became very sore, red and swollen, and finally blebs and ulcers developed. At the end of eight weeks she went to bed. The eruption appeared on the hands three weeks after the sore mouth began. The hands were red and swollen with the formation of blebs. During the summer her condition gradually grew worse. At present she is still in the active stage of the disease, with its accompanying symptoms and signs.

The laboratory work consisted of several blood examinations; examination of the serus exudate from blebs on Case No. 2, examination of feces of case No. 2 for uncinariasis by Dr. Oughterson, and at my suggestion, Wasserman's test was made in cases Nos. 2, 3, 6 and 7 by Dr. Wm. Litterer. These examinations were negative except the Wasserman in case No. 2, which was positive. In case No. 7 it was positive, but the patient had syphilis. There was a slight positive reaction in No. 6. It was negative in case No. 3, Anna M. These cases are the first, as far as we know, that have been subjected to the Wasserman reaction.

"In June, 1909, the blood of Anna, Charles and Roy was obtained and cultures were attempted from the lesions. The result follows:

In Anna—Case 3:

Red blood cells.. 4,200,000
Hemoglobins.. .. .. 80%
Whites.. 8,500
Cultures from the lesions were negative.

In Charles M.—Case No. 6:

Red blood cells.. 4,080,000
Hemoglobin.. .. .. 72%
Whites.. 6,900
Cultures from the lesions were negative. The Wasserman reaction was negative.

In Roy—Case No. 2:

Red blood cells.. 4,000,000
Hemoglobin.. .. .. 70%
Whites.. 13,000
Cultures from lesions proved to be staphylococous pyogenes aureus.

Blood cultures were obtained, taking 10 c. c. of blood giving negative results on all of the media used. The Wasserman reaction was distinctly positive at this time. (June.)

September, 1909.

Anna shows negative Wasserman Charles shows doubtful Wasserman Roy shows positive Wasserman	No history of syph.
Hampton shows positive Wasserman	History of syph.

Blood cultures obtained two days before death in Hampton Case, No. 7, showed the staphylococcus pyogenes albus."

In commenting upon the two groups above reported, the first consideration is, has there been any transmission of the disease from one case to another through food or any other way? Is the disease infectious? We know well enough the opinions of the authorities pro and con. There seems to be absolutely no doubt as to the diagnosis of these cases. In the orphanage the disease was first introduced by a family of children from destitute parents; later on, up to 1909, other cases developed, in the same institution, and in children from all sections of the State. It does not appear that the children outside of this immediate family of four entered the institution with the disease. Every proof shows that they contracted it

there. Every proof shows, as far as it can be obtained, that the disease was introduced by the youngest member of this family of four, the other three members being also later affected, with the intensity of the disease *less,* in the older children. The food at the orphanage was wholesome, well prepared and well served. The inmates showed all evidence of being well fed. Their food, in fact, was equal to that of the average home. The corn meal from which their corn bread was made was procured from one of the most reliable millers of the city of Nashville, and all of the children ate the same quality of bread. The same grade of corn meal is used by the Central Tennessee Insane Asylum. A most careful inspection was made of that institution by Dr. Lavinder, Dr. Tucker, Dr. Olin West and myself and not a single case of pellagra was found even among the colored female inmates.

In group No. 2 it appears as if there was a direct transmission of the disease to the woman from washing the clothes of Case No. 7. The man had a well marked case of pellagra. The woman did his washing during the winter and in the spring developed the disease. There seems to be no other way by which she could contract it. He came into the community with the disease. There was nothing in common between the two, as far as we could learn, except this washing. On the face of the report of these two groups, this disease has every appearance among us of being infectious, or in some unknown way communicable.

DISCUSSION ON PAPER OF DR. KING.

Dr. C. H. Lavinder, Washington, D. C.: I do not like to let this paper pass without saying a word or two in reference to it. I visited Nashville and saw the situation of affairs there, and while I admit that Dr. King has some reason for the conclusion he has drawn, I do not think, with regard to transmissibility, that we should draw a general conclusion from a specific instance in this way. While I do not propose to discuss this matter, I want in a friendly way to offer my protest against such a deduction, so opposed to the conclusions and experiences of all other students of the disease.

Dr. W. E. Hibbett, Nashville, Tennessee: I do not rise to discuss the paper of Dr. King, but simply to emphasize some facts. We have three State institutions and one county institution besides

the Baptist Hospital in the immediate vicinity of Nashville, all of which have the same food supply. In all the other institutions the number of inmates is relatively large, running from four hundred to one thousand. In the Baptist Orphanage we have only seventy-five inmates. The disease was brought in, as the paper states, by a child brought from Newport, Tennessee, four members in the family having had the disease, and one case after another developing it until there were seventeen cases in that institution. After a most careful study, with the exception of the group alluded to, no other case was found in the vicinity of Nashville or in any of the institutions. Now, with those facts before us and while we have never gone on record to the world stating that pellagra is transmissible, yet what else can we think other than there must be some way by which the disease is communicable from one patient to another? Of course, we are not trying to establish the infectious nature of pellagra, but we are here to learn and are open to conviction.

We have been criticised for what was supposed to be quarantining these cases. We do not quarantine them, but we have carried out what we term advisory isolation which has been made necessary because of the peculiar surroundings of the cases in the Baptist Hospital. These children were considered a menace to the health of the community, and if they had been allowed to remain there, undoubtedly some violence would have been done, and so at the earnest solicitation of the members of the Board of Trustees of the Baptist Orphanage permission was obtained for these patients to be put on an isolation farm where they are doing very well.

We do not consider that our water supply has any bearing on the development of these cases. At the Baptist Orphanage water obtained from a reservoir is used. The water supply in the city comes from the Cumberland River.

Dr. Louis Leroy, Memphis, Tennessee: I wish to take the liberty of saying a few words on this paper. So far as amœba and uncinaria are concerned, I would say that in none of our Tennessee cases has amœba or hookworm been present, and they have been sought for in each case. Hookworm is not at all prevalent with us. Most of the cases in Tennessee come from the outside. Amœbæ, on the other hand, are very prevalent. In the neighborhood of Memphis we have an area in which amœbiasis is endemic. There is any amount of it. And still I have never seen a case of amœbaisis give evidence of pellagra, nor have any amœbœ been found in any of our

pellagrous cases, and they have been sought for diligently in each case. As to the corn meal which we get in Memphis, we get it from those infected districts which have been criticised, namely, Kansas City, St. Louis, Chicago and the North. Almost all of our corn meal comes from those places.

As to the technic of making blood examinations which was discusssed last night and the manner of hunting for leucocytosis or an increase in the neutrophiles, I have used a method which seems to avoid piling up and gives a better fringe, and that is, instead of using one slide over the other I use a piece of cigarette paper. This would seem to give a more uniform count and with that method I have found a slight increase of twelve per cent. in the large mononuclears.

With regard to the mental symptoms of our cases of pellagra, the patients have not shown any particular variety. Mostly the mental trouble has assumed the form of a true melancholia, but there is a mental attitude you might say of inanition in that these patients get weaker and weaker, lose interest in everything and there is a less desire to make an effort to do anything. They have not been morose or reproaching themselves with anything. They have not been unhappy, but there has been greater weakness developing with a lack of mental activity.

As to the transmission theory of the disease, I do not know whether Dr. King mentioned the fact in his paper or not, but in these Nashville cases we had one other remarkable thing develop, namely, a nurse who is taking care of these cases in the Baptist Hospital has developed a pellagrous eruption on her hands. There were two other cases that came under my notice and these were school teachers who came from Arkansas having developed the disease in a mild form.

Dr. Lavinder: Where did the nurse come from?

Dr. Leroy: She came from some part of Mississippi. She had not had the disease before she left there.

I would like to make another suggestion with regard to the treatment of these cases, namely, most of the remedies which have been recommended and given will act beneficially in any condition of dyscrasia or of malnutrition. We would look for improvement in these cases from the use of mercury and we would likewise look for good from the use of arsenic, etc.

THE QUESTION OF THE ETIOLOGY OF PELLAGRA

J. H. TAYLOR, M. D.

COLUMBIA, S. C.

It is not my purpose, nor would I presume for a moment to belittle the remarkable contributions of Lombroso and his school to the study of this disease, but, throughout it all, there is not, to my mind, a satisfying and convincing proof, either that corn is the sole factor in its production, nor has it yet been shown that a protozoan element does not play a part.

So dominated has the Italian thought been by the zeitoxic theory of Lombroso that it comes as a surprise to hear even scientists of that country question the truth of the sole influence of spoiled corn as an etiological factor. Yet such is the case, and we note that at the Third Pellagrological Congress, held at Milan in September, 1906, "Palidino pertinently suggests that in Mantua, though good maize is used, yet pellagra does not diminish. Mangieri asserts to have seen in twelve years but two pellagrins at Constantinople, where much maize is used. Terni holds that the etiology is doubtful and the real cause unknown. Moreover, he asserts that in Egypt pellagra is not related to the use of molded corn, nor does the acme of the epidemic correspond to the Nile flood, which might cause the humidity needed by mold. Ceni described pellagra among people who eat no maize and emphasized its disappearance upon improving sanitary conditions, the food remaining the same. Meschinelli remarked that, in spite of all efforts, pellagra seems to diminish little. Here and there is some respite in the advance of the disease, but in general we cannot say that the disease is giving way, or that the number of the infected regularly diminishes, so long as in some places it increases. At best, the struggle against pellagra but holds it in check, if indeed that. The results and the measures taken are disappointing."

On the other hand, in France, England and Spain, where the Lombrosian influence is not so strong, we find a widely spread dissatisfaction with the zei-toxic claim and a casting about for more convincing proof, or else the seeking of another cause. Notably is this the case with Samson and Manson in England, and Nicolay and Jambon in France, though Sandwith, in the former country, ascribes

a failure to accept the corn theory *in toto* as evidence of insufficient study of the subject.

Following the incrimination of corn by Marzari in 1810, every possible shortcoming has been laid at the door of this splendid cereal, and every bacterium and fungus found growing upon it has come in for its share of the odium. First, it was a lack of an animalizing substance; then the "Sporosorium Maidis," and one thing after another, each, strange to say, being stated by its particular champion capable of producing the symptoms in animals. Finally, in 1870, Lombroso put forward the zei-toxic theory, based upon experimental evidence, by no means convincing, which deems the cause toxic products analogous to alkaloids, developed during the growth of certain molds upon spoiled corn. Neither of itself is capable of producing the disease; in fact, the mold is harmless when grown on barley, rye, grapes, jam, etc.; yet let it but grow on spoiled corn, and there is developed an intense poison, capable of producing, it is asserted, the symptoms of a distinct pathological entity, differing in every detail from all other known fungoid diseases. This very uniqueness, as compared with diseases of a like origin, is calculated to make us sceptical, for in the past our greatest assistance in forecasting the nature of individual diseases has been by analogy. A moment's thought brings to mind examples of this.

The lights of the past can but reflect on the present, and it would be well to bear in mind that from "Sporosorium Maidis" Ballardini produced in man gastritis and diarrhœa, while with chickens it caused loss of weight, droopiness and a falling of the feathers. This he called pellagra. Neusser and De Glaxa accused the colon bacillus of decomposing corn, even sound corn, after ingestion, with the production of a poison which causes pellagra. The latter scientist reports obtaining the characteristic anatomical lesions in dogs fed on sound corn porridge and, moreover, the proper symptoms and lesions in animals inoculated with a toxin produced by the colon bacillus cultivated on a maize medium.

In 1894, Pelezzi and Tirelli injected dogs and rabbits and fed by mouth toxic substances from cultures of the bacteria of maize and obtained spastic paresis of the posterial limbs and other symptoms, which they likewise deemed characteristic of pellagra.

Similarly, Carrarioli, Di Pietro, Ceni and Besta each finds a different specific cause for pellagra and obtains in animals the desired symptoms.

Can we have any clearer demonstration of the powerful influence

mental attitude exerts over our interpretations of physical phenomena, notoriously inaccurate at best, and does it not tend to make us very cautious about accepting as final the statements of any one man or set of men about so fundamental a thing as the cause of a disease?

In order to prove beyond a reasonable doubt the causative relationship existing between pellagra and spoiled corn, experimental evidence has been sought for, through animals, and continues its role of deceit, made possible by false interpretations, through misdirected enthusiasm. Lombroso, especially, has invaded this field and presents to us certain results that he considers final. Indeed, I am told that he has asserted to have said the last word on the subject twenty years ago.

Experiments[1]: First, ten dogs and a large number of chickens were fed for six or eight weeks up to the natural end of life on corn spoiled with aspergillus and the following symptoms developed in the dogs:

Weight: All the dogs lost weight except one.

Temperature: There was nearly always an elevation of temperature coincident with the diminution of weight.

Muscular Spasm: A symptom frequent, but not constant, was a tonic muscular spasm and the increase of tendonous reflexes.

In seven cases out of the ten, a complete torpor of the muscles was produced. In six cases, cerebral torpor; in three, loss of sensibility; and in four cases out of the ten, muscular tremors.

Diarrhœa: The most frequent complication was diarrhœa preceded by a refusal of food and dysphagia. In seven cases, the latter symptom was most often due to muscular spasm.

Skin: The erythema of the skin was manifested once only completely and certainly, and then in a dog which remained free from anaemia, torpor and spasmodic phenomena, and in which the sitophobia and paresis disappeared with the appearance of the erythema. The microscopic examination of the skin in this case did not, however, exclude all idea of a parasitic cause.

In twelve chickens fed on spoiled corn convulsive phenomena were exceptionally noted and there were produced only atrophy of the feather follicles with changes of the skin and horny appendages.

Finally, the experiment that in the opinion of the Lombrosians goes farthest towards definitely proving their contention was that made in the case of human beings. The tincture of corn spoiled by

penicillium glaucum was given for a length of time to twelve sound and healthy individuals, soldiers and laborers at work in the city, and, at the time of the experiment, well-fed. The following results were obtained:

Symptoms.	Number of Cases.
Bulimia	9
Pruritus of back and face	8
Loss of weight	8
Urine diminished and of high specific gravity	8
Diarrhœa	6
Diurnal Somnolence	5
Eructations	5
Persistent muscular weakness	5
Desquamations	5
Headaches	4
Anorexia	3
Burning of the eyelids	3
Repugnance for water	3
Hyperidrosis	3
Ephelides on the arms and hands	3
Palpitation of the heart	3
Syncope	3
Variation of the pulse	3
Increase in weight	2
Intense thirst	2
Increase of muscular force	2
Sensation of heat in the head	2
Sensation of warm water on the back	2
Irritability and emotionalism	2
Restlessness at night	2
Erythemas	2
Tinnitus aurium	2
Redness and burning of the skin	2
Burning of scrotum	1
Mydriasis	1
Ptosis	1
Prickling sensation	1
Oppression and Precordial pains	1
Vertigo	1
Furunculosis	1

Enteralgia	1
Sensation of foreign body in the head	1
Salty taste	1
Burning in the throat	1
No symptoms	2

In these experiments we note at once an utter lack of uniformity in the symptoms presented by the different individuals, and, indeed, in some there were no symptoms at all. Now, can one accept these phenomena of acute intoxication as definite proof that corn spoiled by penicillium and asperigillus produces pellagra, a disease characteristic only in its entire symptom complex? One of the most striking elements of the disease, a symmetrical skin lesion, is entirely wanting, and, moreover, would it not be desirable, if we are to accept as positive evidence, that the eruptions really occurring should tend to show some slight periodicity, a glaring characteristic of the true disease?

Pellagrosine, the essential poison of Lombroso, will produce in dogs and chickens but droopiness and diarrhœa and in man torpor, anorexia, nausea and diarrhœa. Would it not be reasonable to expect something more classic than this?

We would not pretend to deny that these experiments show a development of a powerful toxine, that affects most unhappily the victims subjected to its influence, but, on the other hand, animals can be made to develop diarrhœa and succumb by being fed on any kind of mold and the symptoms produced in Lombroso's experiments can be reproduced with extracts from spoiled and moldy wheat and barley. Ciotto has even succeeded in demonstrating the presence of alkaloid substances both in unaltered corn and in other cereals. Furthermore, Babes and Sion[2] state that amygdalin causes appearances similar to pellagra in plant-eating animals, yet is inert in carnivora, and, moreover, they state that the following symptoms have been produced in mice, rabbits and dogs by other toxines than that of spoiled maize, viz.: loss of appetite, diarrhœa, inflammation of the bowels with hemmorrhage and general weakness; paralysis, beginning at the hind extremities, tetanus and clomus-like cramps; opisthotonus; skin affections and particularly, also, desquamations and falling of the hair.

We admit it is not possible to deny the overwhelming evidence of the frequent and very suggestive association of pellagra with the ingestion of corn, and especially of spoiled corn. Indeed, these may

contain very essential elements in the production of the disease, but this is far from naming them as the ultimate and essential element in its production.

Aside from the bare experimental phase of the question, certain discrepancies in the maize theory are worthy of our close scrutiny. The cultivation and extensive use of maize antedates by about 200 years the appearance of pellagra in Italy, and zeists explain the long years of freedom from symptoms in the individual by asserting that the poison acts very slowly, taking these years to produce the disease in man, yet, when a guinea pig or other animal is killed within a few short days by the identical poison, so stated, it is still accused of dying of pellagra.

There are extensive regions today where pellagra is widespread, but where corn is neither grown nor eaten. This is notably the case as cited by Pons Sanj[3] in the province of Vodjoz, Spain, where pellagra is endemic, though the inhabitants eat no maize; and, again, the province of Ovido was once ravaged by pellagra, but in 1900 it suffered least, though no changes had taken place in the culture, storage, preparation or quantity eaten of maize, while the province of Madrid, where maize is only seldom used as food, suffered severely.

Now, what are we to say about those cases of pseudo-pellagra, so-called, and sporadic pellagra, reported as far back as 1852 and more recently in France as developing in persons who had never eaten the products of corn? Most of the French cases are admittedly alcoholic, but we cannot ascribe their origin to alcohol alone, and we find ourselves still face to face with an unknown causive agent, just as we are, in the ultimate analysis, in cases of true pellagra.

In 1903, Garbini identified several cases of undoubted pellagra at the Messina asylum, in natives of Cicily, where maize is not cultivated, and, what is more to the point, they had been inmates of the institution for a long time and had certainly eaten no maize for several years prior to the appearance of their erythema.

In Jamaica,[4] it is reported that insane patients who had been shut off absolutely from a corn ration for the period of a year had developed pellagra. Can these instances be satisfactorily accounted for by the Lombrosians?

Again, is there adequate explanation for only 20,000 cases of pellagra among five million Roumanian peasants in 1898, as reported by Babes and Sion, and for the few hundreds at present among our own millions? Certainly, in Roumania the status of corn culture and

preparation has not changed in a great many years as is held up against our country, or, if change has taken place in Roumania, it has been for the better, in the light of the Zeist propaganda. The Zeist explains this by predisposition in the individual, yet it seems hardly probable that neighboring peoples would show such marked variations in susceptibility. Of course, we still have that indefinite and uncertain factor, hereditary pellagra—atavistic pellagra, as Lombroso would have it—found developing in grandchildren of recognized pellagrins, but here it seems to me we are again facing our unknown etiological factor. Is this, indeed, transmitted susceptibility to the poison of spoiled corn? Why should certain individuals of the same inheritances, the same household and living under identical food conditions, be attacked and the others remain free of symptoms? And, again, why should persons of the rural districts, and rarely those in towns, be attacked, though both use maize as a staple article of diet? The Zeists explain this last contention by claiming that the best corn is sent to the city and the poorer quality eaten by the country peasants; but this seems not to explain the phenomena enirely to our satisfaction, for surely much corn must spoil in storing. And, moreover, statistical data of Lombroso himself and of Babes and Sion show that scarcely 25 per cent. of pellagrins can be shown to be in the habit of eating spoiled corn. Again we face an unknown element.

In considering the glaring incongruities of the maize theory of pellagra, the striking and distinct periodical appearance of the skin symptoms, a phenomena incompatible with toxine poison of the maize type, and its striking similarity to certain diseases of a protozoan origin, Sambon[5] has mentioned as possible a similar etiology for it. This suggestiveness runs throughout the entire symptomatology, pathology and treatment of both syphilis and sleeping sickness, or trypanosomiasis, both protozoan diseases, and the Wasserman reaction justifying us in considering general paresis and tabes dorsilis as syphilitic manifestations, we include these under this head:

Type: They are all essentially slow progressive toxæmias, 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 microbe nature, and are readily explained by the theory 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 a symmetrical, while in sleeping sickness there is no definite arrangement. In pellagra it appears usually 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 a 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 illdefined 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 spasms, 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 delirium of exaltation rarely being observed, while, on the contrary, despondence 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. Œdema of the central nervous system and a chronic leptomeningitis 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, has reported his experiments with syphilis on apes in Java, where he demonstrated the extraordinary influence of an arsenical preparation, atoxyl, on affected animals. 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. From this drug alone has benefit been received in the case of sleeping sickness. While this particular form of arsenic has proven a disappointment with us in pellagra, yet, Fowler's solution of arsenic seems to hold out the greatest amount of relief in cases taken in the early stages. On the other hand, Lombroso himself has spoken of splendid results from atoxyl, while Babes and others report brilliant results from a com-

bination of it with arsenic trioxide.[6] Thus we see arsenic alone giving relief in two of the three diseases and acting as a specific in the third. In this connection, while pointing out the similarities between pellagra and other protozoan diseases, there is food for thought in the striking influences of light on the skin lesions of pellagra and smallpox—another protozoan disease—as also is the fact that there has been noted in the past and Lavinder finds it in the cases examined here, a constant relative increase in the monocular cells of the blood, a phenomenon likewise characteristic of syphilis, sleeping sickness, smallpox and protozoan diseases in general.

Finally, regarding the pathogenic protozoa in man, we have but little more than lifted the edge of the veil, but even that opens to us a boundless horizon. With the exception of the malarial organism, the complete life history of not one protozoa causing disease in man is known. Of the others, we know only disconnected portions of life cycles that may, and probably have, many varying forms. We do know, however, that the majority of identified forms are restricted to a particular organ or tissue, as the malarial parasite and the trypanosome and others to the blood; the organisms of scarlet fever and smallpox to the skin; some to epithelial cells; some to cell nuclei and others to muscle cells. Because we find nothing suggestive of protozoan life in the blood and other organs, so far examined, of pellagrins, it does not argue that we shall not eventually find, with fuller knowledge of this form of life, the real organism tucked away in some obscure recess of the body. However, it is an interesting fact that in some forms of protozoa there are formed under certain conditions protective cysts within which the living cells lie quiescent for varying periods, until environmental conditions are favorable to liberation and renewed activity. We note especially this quiescence in the malarial organisms, which may remain latent for many years in the blood and by changes in the density of the surrounding medium or through some minute change in the composition of the host's blood, be stimulated to renewed activity. We are all familiar with the latent periods which are characteristic of syphilis. In the case of pellagra there is irresistibly borne in upon us some as yet intangible relationship between corn, especially spoiled corn, and the development of the disease. Now, knowing what we do of the quiescent phases of certain protozoa and the stimuli necessary to their renewed activity, is it not possible that, either one cycle of an organism causing pellagra, if there be such, finds its habitat in corn, or else may it not lie in the tissues somewhere, possibly the intestinal tract, and the ingestion

of corn, producing the chemical stimulus necessary to its development, there results an invasion of the host with a development of the usual symptoms? It seems to me this would account for the recrudescences following the eating of corn products, and also we might reasonably stretch the analogy to include those cases developing sporadically or pseudo-pellagra, accounting for these by the ingestion of some other product than corn, producing a like stimulus, but much more seldom eaten than corn.

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DISCUSSION ON THE PAPER OF DR. TAYLOR

Dr. William Allen, Charlotte, North Carolina: Dr. Taylor, in his paper, drew an analogy between the treatment of syphilis and pellagra. I have had several cases of pellagra that gave a past history of syphilis. They were first put on the treatment recommended by Babes last winter, which failed absolutely to bring about any improvement. Later they were put on hypodermic injections of salicylate of mercury and recovered. It is rather suggestive that the cases which gave a history of syphilis should recover under the use of mercury. Now, I am trying hypodermic injections of a combination of mercury with arsenic, the cacodylate of mercury. I would like to ask Dr. Taylor whether he has ever tried mercury in any of his cases either with or without a history of syphilis?

Dr. Walter H. Buhlig, Chicago, Illinois: Along the line of the etiology of pellagra, I wish to mention the results of a few experiments we have made at the Peoria State Hospital for the Insane, under the auspices of the Illinois State Board of Health.

First, with reference to amœbæ. I should have covered this phase of the subject yesterday, but we were so pushed for time that I did not mention what we had done. Dr. Nichols and Dr. Siler told you in their joint paper yesterday of finding amœbæ. As we were on

the ground we knew they found them. The water was examined by us and amœbæ were found in it. A little later, in the middle of October, I sent an assistant for more water and got the following samples: The city water in Peoria; the city water of Peoria at the institution which corresponded to the water examined in the first experiments; the well water of the institution; and some water which came from the refrigerating plant and which flowed into the large tank which furnished water for the institution. We found no amœbæ in the city water of Peoria. We found none in the city water taken at the institution, contrasting this with our previous experiments in which we did find them. In the well water no amœbæ were found, but in the water which came from the refrigerating plant we found amœbæ in large numbers. We could not understand the disparity in the findings in the city water in the earliest and this last examination. So I wrote to Dr. Zeller and asked him whether this tank had been cleaned out, and he told me that about the 1st of October they cleaned and steamed it. That probably accounted for the absence in the last experiment of the amœbæ in the city water at institution; but the pipes of the refrigerating plant had not been cleaned out and the water coming from them still contained amœbæ.

With this as a clue, I examined the water at our school, the Northwestern University Medical School, where somewhat similar conditions obtain. We get our water from Lake Michigan, and it is supplied to us in two ways. The water comes direct from the mains on some of the floors, but on the upper floors the water is supplied by means of a large tank on the roof, to which water is pumped from the basement. In the water from the mains direct there are no amœbæ, but a great many flagellates, and in the water from the tank I found large quantities of amœbæ as well as flagellates. These experiments have a distinct bearing on the question of the pathogenicity of the amœbæ found in the water and the stools at the Peoria State Hospital.

We have also gone into the question of the bacteriology of the stomatitis of pellagra. In direct smears we found a large number of spindle shaped bacilli and spirochetes of the Vincent's angina type. In addition we saw a good many bacilli of the bacillus ærogens capsulatus type and some large cocci that are much like the large diplococci found in the stools of normal individuals. Culturally we found practically always gram negative bacilli of colon morphology, and after plating what we thought confidently were colon bacilli proved

to be two organisms, one that stimulated the bacillus lactis ærogenes and the other one that had all of the characteristics of the bacillus cloacæ.

CAPT. H. J. NICHOLS, U. S. Army: I have been very much interested in listening to Dr. Taylor's paper, and, as I said yesterday, the parasitic theory of any disease is the most attractive one, but I do not feel that we are safe in following in Dr. Taylor's lead on this proposition.

We have had the same experience with beri-beri. This disease in the tropics has been a great problem for many years. At first, it was thought that rice was the cause of the disease; then opinion swung over in favor of a protozoan or bacterium, but finally it has come back to rice again, and unquestionably, there is some definite connection between white rice and beri-beri, although the exact relation is still unknown. We are now going through the same phases in this disease.

First, every one was pretty well satisfied that it had something to do with the diet; now theories are being urged in favor of a protozoal or bacterial origin of the disease. It seems to me that this is largely a psychological reaction, rather than a reaction based on reliable data. Dr. Sambon has made quite a stir in medicine on account of his free lance methods of theorizing about different diseases. For instance, black water fever, which men in India feel sure is connected definitely with malaria, is attributed by Sambon to some special organism. It is the same way in regard to pellagra. These theories have a psychological basis rather than one founded on actual data. So far as our position is concerned, it should be a conservative one, and we should take the ground that as far as we can tell a corn diet has some definite connection with pellagra, and that in our prophylaxis we must cut out this article of diet among predisposed persons. Until further studies are made we are not justified in going into the air for any hypothetical protozoal or bacterial origin of this disease.

DR. J. J. WATSON, Columbia, South Carolina: I wish to express some sentiment in regard to this subject. I do not think we should let the ideas of any one man or any set of men disturb us or carry us away from the real truth. Many arguments are advanced concerning the probable protozoic origin of pellagra, and that the drugs that are used in the treatment of protozoic diseases are beneficial or

curative in cases of pellagra. But I want to say that there is no drug which cures pellagra. I have had extensive experience with this disease, and I have seen no drug that would cure it. Arsenic does not do it. I have had patients recover when I gave them morphine. Yet, on the other hand, I have had others recover when I gave them nothing, and when I got out of the way they got well. So the argument that such and such a drug which is used in protozoal diseases beneficially does not hold here. I am familiar with Dr. Sambon's theory as to the protozoal nature of pellagra; but I want to say in this connection that ordinary good corn does not produce pellagra. It is when corn is damaged that we get pellagra. That is the experience of those men who are dealing with this disease and who have studied it closely for generations, and we cannot afford to pass the observations of those men of vast experience over lightly. The trouble is not with corn. God Almighty in his infinite wisdom has given us a climate where we can mature and cure corn, but in Italy it is entirely different. There they have an artificial way of drying corn. They cannot leave it in the fields until it is matured, and until the excessive moisture is evaporated by the glorious orb of day. The trouble is in the way we handle corn, and not in the corn itself.

Dr. Hiram Byrd, Jacksonville, Florida: Those of us who live in the South in the yellow fever territory, and who have had occasion to look over the literature on yellow fever, will recall very vividly that twenty years ago, yes, ten years ago, it was thought that yellow fever was transmitted by fomites. It is true, here and there was a dissenter, some eccentric somebody who did not accept the theory, but we nevertheless believed that yellow fever was transmitted by fomites. We had to apologize for a number of things in order to live up to that theory. But we apologized and accepted the theory and went upon the assumption that yellow fever was transmitted in that way. Today, when we come to make assertions, our friends say to us, what about yellow fever? We have had to take back water. We all know that. The one thing that impresses itself upon us with regard to pellagra is that we do not know its cause. Let us not put ourselves on record prematurely. Now, if there is any one thing that this conference has forced upon me, it is the belief that we do not know the etiology of pellagra; that we are today in the dark. This conference has taken upon itself the character of an international body. Every part of the world, where pellagra is an

important problem, is represented here either in person or in sentiment. The proceedings of this conference will be read by the entire world, and is it a safe thing, in the present state of our knowledge, for us to put ourselves on record that corn is the cause of pellagra? I contend that it is not. We had better say, we do not know. This is a scientific body. It ought not to accept anything short of absolute proof which the most exact science can furnish. So far as corn is concerned, the least we can say is, it is unproven that it is the cause of this disease, and let us not put ourselves in the humiliating position at some future time of having to take back what we may say in this conference. (Applause.)

Dr. W. H. Dial, Laurens, South Carolina: While I am a physician, I was raised on a farm and am still interested in farming. We all know that sound, properly cured corn is one of the greatest and best of our food products. But the way of harvesting in these later years has had something, no doubt, to do with damaged meal. We now cut the stalks from the ground before the corn is ripe, tie them in bundles, and stack them in great shocks, to thus mature in the fields, claiming that the stalk holds or retains enough nourishment to properly mature the grain.

This is different from the old way of stripping the blades from the stalk and allowing the corn to mature on the stalk still growing in the ground before gathering or harvesting. May this difference in the maturing of the corn not have something to do with damaged corn and meal? And damaged meal, we are told, produces pellagra. This late way of maturing corn should be thoroughly investigated.

Dr. J. D. Jones, Sweet Water, Alabama: I hail from the great old State of Alabama, near the City of Mobile, and have had considerable experience during the last three years in treating pellagra. I have now under my observation twenty cases of pellagra, mostly in the country district. This disease has been under my observation for three years. For a year and a half I did not know what it was, but by the aid of Dr. Searcy of our State Insane Hospital, I was led into the light to know what it was. I have had during that time three deaths. I have now under my treatment three patients who are insane. When I left home one of them was nearly dead, and may be dead before I get home. I am also engaged in the mercantile business. Three years ago, if you remember, in October of 1906, about the 10th, we had a killing frost which reached from the North

to the South, even to Alabama, and by reading the papers you found it was plainly stated that the corn fields of Kansas and of the middle West and North were blackened by that frost; that corn was not matured when the frost came. And the next spring we had considerable wet weather, so that it was impossible for the merchants of my section to procure sound corn for their patrons. The corn meal at that time was cheaper on the market than pure corn, and that is always the case in the mercantile world. You will find a bushel of damaged meal can be bought cheaper in such a time than a bushel of pure corn. I could not secure more than enough corn to supply the demand of my customers for more than ten days at the time because the corn would become damaged, and I could not sell it. I would have to give it away. In that very fall the first case of pellagra came under my observation, and from that time on the disease has been spreading. The first year I had three cases of it. I did not know what it was; but I treated these patients symptomatically. The disease was not severe in its type. The next year I had six or seven cases, and this year it is spreading over my land like another Niagara. Our State Officer, Dr. Sanders, said a month ago it was on the wane. In my section, in the last six weeks, I have had six new cases of pellagra to develop, mostly amongst children. I am treating today a child, two years of age, with pellagra. It is getting along nicely. The idea has been advanced here that corn is doing the damage. Corn is one of the chief merchantable commodities of the United States, but I tell you, gentlemen, it is not corn according to my idea. It may be damaged corn, but pure, fully developed, hardened corn—hardened by the rays of the noonday sun—will never produce pellagra. That is my idea about it, and we should be very careful, in giving our ideas here, not to let it go out to the outside world that corn is doing this mischief, is producing this disease. People are crazy all over the country over the idea that corn produces pellagra.

Dr. Louis Leroy, Memphis, Tennessee: The subject of the etiology of pellagra is the all important one of this conference. It strikes me that if we look back on the etiology of any of these diseases it is only a year or two ago, comparatively speaking, when reports as to climatic conditions, rain-falls, etc., were tabulated and brought forward to explain the etiology of typhoid fever. We have had all kinds of statistics to show that typhoid fever was caused by certain conditions of climate and certain conditions of rain-fall. I

believe that we are not far from a similar condition here, when we seek to ascribe to moldy corn and improper harvesting of it the etiological factors in the development of pellagra. That there is a relationship existing between pellagra and corn, I do not believe we can doubt, any more than we can doubt the existence of a relationship between impure water or impure milk and typhoid fever; but that does not mean that the last word has been said by any means as to the etiology of pellagra. What is it that makes corn moldy? It is not the climate that made it go bad. The corn crop has been blighted, but that is not what made it go bad. It is some other living form of organism which is gaining a livelihood on that corn upon which it can grow after it is once blighted, or from climatic conditions, until the protective coat of the corn has not had an opportunity to form fully, or for some reason through the improper formation of the husk it has gained entrance to the kernel, and while we perfectly admit that corn plays an etiological role, we cannot stand up and say that corn is the cause, and the only cause, of pellagra. There is something else, and we want to find out what that something else is in the corn. Corn is only a half step forward. We cannot be satisfied by letting the case rest at that point. Whether this disease be protozoal or bacterial in origin we do not know. Our attitude must be this, that up to the present point corn has acted as a carrier of the infection; that in certain cases corn may carry the disease, and in saying that it will not bring us into conflict with commercial interests. While we admit the likelihood of damaged corn as a cause of the disease, at the same time it does not leave out of consideration the fact that a protozoal infection, or infection by some micro-organism, is the cause of the disease. Personally, our cases that have been reported show unquestionable evidence of transmissibility of the disease, and for that reason, in addition to others, it is my personal opinion that some day we shall discover a living cause; but let us not go upon record as saying it is corn, and only corn, but let us say it is corn and something in the corn that may cause the disease. (Applause.)

Dr. J. H. Taylor, Columbia, South Carolina: With reference to the use of mercury in the treatment of pellagra, I will say that Dr. Wright, of Colorado, noted the happy effect of mercurial treatment in tuberculosis cases and has been using the succinamide of mercury in their treatment. He has published several articles on this subject, reporting splendid results.

With Wright's success in mind, Dr. Lavinder, while stationed here at Columbia, in ten cases of pellagra used the succinamide of mercury in one-eighth grain doses given hypodermically. One case showed decided improvement. This, however, was a patient badly infected with syphilis. The other cases showed no improvement.

With reference to the remarks of Dr. Nichols, who said we must go cautiously and gingerly, so far as corn is concerned, I agree with him that we must continue our investigations and efforts in the corn direction. There is no question as to some relationship between corn and the production of pellagra; therefore, we must cut out the bad corn, although we do not know as yet what is behind it. Corn is a medium in some way, and we shall find out some day how.

We do not know the cause of beri-beri, but it is said to be associated with the eating of rice, and in the same way the eating of corn may be associated with pellagra. I have seen but one case of beri-beri, and that was shown me by Dr. Sandwith in London this summer at the London School of Trophical Medicine. I know very little about the subject.

As to the remarks of Dr. Watson, I expected more from him and was a little disappointed. I am glad to see, however, that he only criticises the arsenic treatment in cases of pellagra. The rest of my paper he seems to pass over without objection. I referred to Dr. Babcock my statement about improvement in the early stages of pellagra from the use of Fowler solution and he sanctioned it. I have never said that arsenic cured pellagra, but it is the only drug with which we can note any improvement traceable to its use.

Dr. Whaley, I think, is talking about blind staggers from the toxins of bad corn, in all of which I agree with him perfectly. He has not been discussing pellagra.

PERSONAL EXPERIENCE WITH DAMAGED CORN

J. SWINTON WHALEY

EDISTO ISLAND, S. C.

Gentlemen, when I survey this august assemblage and view the many M. D.s and men of scientific thought and investigation coming from every part of our country, the question comes to me, What has a plain planter and common farmer to do in their midst?

In an unguarded moment I mentioned to Dr. Babcock an experience I had had with damaged corn, and he, always looking for facts and seeking to know the truth underlying all conditions and diseases, thought this experience of mine might be of value to the conference, so at his request I am here to tell you about it, and what I know of damaged corn.

Damaged corn! Why should it be considered here? Because some have thought this to be the first cause of the dread disease you gentlemen are conferring about.

If, as has been asserted, pellagra in man and what we farmers know as blind staggers in horses are analogous diseases and produced by the same cause, then, this being true, it is well for you to consider the matter, look into this subject, and find out how the corn becomes damaged.

I had heard of my neighbors losing their animals with blind staggers, and that it comes from feeding bad corn, but I did not realize it until in the fall of 1903 I lost two mules and one horse, and as I could not determine the cause and not knowing how far the disease might go, I sent for a veterinary surgeon to examine my stock. To my surprise he found several more mules and horses affected. He immediately told me I was feeding on damaged corn. I did not agree with him, as to all appearance the corn I was feeding was as good as I usually made. He insisted that we make an examination. To do this we had to shell some of the corn, as I was feeding on ear corn. Upon doing so we found the grain to be black about the eye. He advised me to stop feeding on it at once or I would lose all of my stock. I told him I had nothing else to feed on and I could not get oats, which he advised me to order, for several days. "Better to feed on hay if you want to save this stock." I did so and lost no more with blind staggers, and from then on I have used oats and only corn that I know has been properly cured.

Now, how was it that this corn of mine became damaged? Because, realizing the truth of the statement that there is as much

feeding value in the stalks or stover as in the grain, I attempted to cure my crop after the shocking method instead of the old-time method.

Now, as most of this assemblage are probably not familiar with the old-time method of harvesting the corn crop in the South, I think I am justified in taking up your time for a few moments in describing it, for I am sure I can convince you, as I have been to my cost, that the trouble is not in the corn itself, but in the way it has been harvested.

The custom in the South used to be to strip the blades as soon as the shucks started to turn yellow and turn the stalk down below the ear, then the corn was not broken in until last of October or first of November. This permitted it to cure thoroughly and the turning down of the stalk prevented any rain getting in the ear, especially where the shuck may have been broken by birds or from other causes, and so long as I continued to use this method I did not know what blind staggers was. But in my desire, as stated above, to utilize the whole plant, I cut the stalks down at the time I usually stripped the blades and shocked them up, leaving those shocks in the field exposed to all kinds of weather with most of the ears turned up for a month or so, then shucked the corn and stacked stalks to be fed as roughage during the winter. The roughage was fine for the cattle, and I congratulated myself that I had hit upon a way to carry my cattle through the winter in fine condition, but this gain was nothing to the loss, as I found to my cost, in damaged corn. So I abandoned that method and have had no more blind staggers.

Many of my neighbors on the truck farms have been obliged to give up the shocking method, as some of them have lost in one season their entire stock of work animals, and while some have to continue to practice that method on account of having to have the land on which a crop of corn has been raised after one of early truck, such as cabbage or potatoes, to prepare for the same the next spring, they have to be very careful in feeding, one trucker telling me that he not only sorted out all the rotten ears when breaking, but had all dotted ends cut off before feeding to stock.

For the past few years I have had to stop buying Western corn, and have bought oats entirely, as the corn has not been fit to feed on, being in the same condition my damaged corn was. Why is it that you cannot get the good corn from the West now that one could in the past? I believe for the same reason I damaged mine. The corn is cut and shocked, exposed to rain and possibly snow, both

soaking into the ear, then it is taken to the shredder, the stalk fed to the machine and the corn comes out at one end and the stover at the other, but what does the man who is feeding the machine know of the condition of the corn he is shelling? The fans may take out those grains that are so rotten as to become light, but what of the heavy grains with the black eye, the blind staggers and pellagra producing grains—they go to market. And if we have damaged corn we have damaged grist and meal. It seems almost out of place in a gathering like this to have a planter talk of the different ways of curing corn. But if blind staggers in horses and pellagra in man are one and the same disease, and to my lay mind the first time I saw a case of pellagra the movements of the patient resembled that of a horse in the last stages of blind staggers, then this matter of damaged corn becomes of first importance, for that it is a sure cause of blind staggers is an accepted fact. Especially when the corn is raised after truck. One and all realize this and are seeing to it that their corn is properly cured. We have no blind staggers in horses or mules without damaged food. No impure water produces it, for in every case stop the bad corn and you stop the disease no matter what the oher conditions are.

I realize, gentlemen, that this is a large and important subject, for we are attributing to one of our greatest and most valuable products the cause of a most malignant and far-reaching disease. Is this to jeopardize the usefulness of the product? Not at all, for it is only when it is bad that the results are evil, so we must see that no damaged corn or its resultant products are put on the market. For the people amongst us that are showing the largest percentage of pellagra are those of our population whose chief food is corn and its resultant products manufactured and shipped to us from the corn-growing States of the West, for the advent of pearl grist and meal has taken the place of the home-made product and we are not getting the good article now that we did when it was first introduced, and we must publish its ill effects universally and we will have it remedied, for just as soon as the grower and the handler—the man through whose hands it passes—are made to realize that damaged corn and its resultant products are unsalable they will mend their methods.

Let the Government bring its pure food law to bear and see that the manufacturer does not put a bad product on the market and I think you will see a great change in the number of negroes, especially, having the disease.

Hoping my effort may have added some little to the solving of the problems, I must thank you for your patient hearing.

SOME FACTS AND THEORIES OF PELLAGRA

H. E M'CONNELL, M. D.

CHESTER, S. C.

While material is being collected as to the nature of pellagra, it is the duty of every practicing physician to contribute his mite, and consequently I will say that the facts herein set forth are drawn from my personal experience with twenty-four cases in my home county of Chester, dating back to 1903.

After the Conference of last year, which was so largely attended and in which so much interest was shown, I had hoped before another twelve months had rolled around, we would not still be groping in the dark, but that we could lay our finger on the etiological factor and say this fungus, or this bacillus, this protozoa causes pellagra. And even that the name itself be found a misnomer. Then, too, a specific cure had been hoped for, either medicinal or a serum, but still we must wait and listen to the theory of the Zeist and Anti-Zeist.

In each of my cases there has been a definite history of corn products of questionable character being used, and this puts me in the class with those still holding to the maize theory. Then, too, I cannot pass lightly over the history, experience and teaching of the past 175 years. As we were wont long to associate malaria with damp lowland and stagnant water, the sleeping sickness, to living near rivers and lakes with wooded shores, so must we associate pellagra with spoiled corn. But why pellagra in America at this late date when corn had been the staple food even before civilization came to our shores? Indeed, it seems strange that after the lapse of centuries a disease due to corn should come back to us from the old world when the seed was first obtained in America. I believe this fact due entirely to the changed methods of cultivating and harvesting corn, whereby un-matured corn is placed in the most favorable condition for fermentation and fungus growth.

At present, as a rule, it is always handled in large quantities and by machinery, with no means by which the damaged or rotten corn is separated from that which is sound and suitable for food. It is meal from such corn as this that most of us use if we eat corn products at all. In my town, with a population of eight to ten thousand, if you had good sound corn that you wished ground into meal, you

would have to send it five miles to the nearest mill, and then with the possibility that your meal be infected with the fungus of spoiled corn being ground just before yours. Often the doctors are asked: Why don't all of us have pellagra because nearly all Carolinians eat corn in some form? My reply to such inquiry is by explaining the different degrees of resisting power peculiar to each individual. They had as well ask why we don't all have tuberculosis or typhoid fever. Often the laymen ask me if it is contagious. My reply is in the negative, for if it were with the association I have had with my cases I would have contracted it. But it is always well to say that the only way to avoid it is to avoid spoiled corn. The fact, too, that so seldom more than one case occurs in a family is against the idea of contagion. In my experience, in only two instances have more than one case been observed in the same family.

I have here a photograph of a healthy baby three years old who nursed a pellagrous mother. (Exhibit photo).

From the similarity of the disease in all cases I am led to believe that it is due to a fungus or a bacillus with their toxines associated with spoiled corn. It's port of entry is through the stomach and intestines and when once engrafted it is hard to dislodge. All these cases had in the beginning a stomatitis, salivation, burning in the stomach and diarrhœa, more marked in some than in others. The slower developing cases usually present these symptoms twelve months before the rash appears, the acute cases after a few weeks.

By this rash we make the diagnosis of pellagra, the blood showing no specific organism; varying from the normal only by a slight lymphocytosis and the urine and stools containing no amœbæ nor bacillus as yet of recognized pathogenicity. I further believe that this fungus is unaffected by heat, or, which is more probable, enters the stomach with poorly cooked food. Apparently cured cases seem to relapse easily after corn products are used even though they be of the best quality.

While the stomach and intestines are the primal foci, I think most of the symptoms are produced by the action of the toxines on the nervous system as the history of the following case will show:

Mrs. C. F. S., age fifty-eight. Good family history, eight children, all healthy. Lived on farm with good hygienic surroundings. Was called to see her on December 31st; with slight cerebral hemorrhage affecting the right hand and with slight aphasia. No heart or kidney lesion. Arteries somewhat sclerosed. Advised light diet with plenty of milk and mush. May 24th she had a second attack of

cerebral hemorrhage, was completely unconscious for several days; right hand and foot paralyzed. Consciousness slowly returned, and gradually some use of the foot, but not much of the hand returned. Was in bed three or four weeks, then up in a roller chair. September 14th was again called to see her and found her with a sore mouth, salivation, burning in the stomach, diarrhœa and a slight rash on left hand which in a week's time was typical, but *no rash on the paralyzed hand.*

This to me was a point of interest and I reasoned, whether correctly or not, that the motor area in the brain being damaged and the trophic nerves along with the motor being degenerated, could not transmit the stimulation from the toxines to the skin and produce the erythema. This patient did not improve under treatment and died suddenly October 2nd, 1909, from cerebral hemorrhage.

Of the ten cases observed in death the minds of all were affected except the case which died suddenly already referred to. In all, the last few days of life were distressing. Of the ten cases seen up until the 1st of January, 1909, seven were women and three men, and singularly all the women are dead and all the men living and apparently well. One of whom I present today.

Mr. W. W. Mc., age sixty-nine. During the summe. of 1906 was treated by Dr. J. M. Brice, was so reduced by the disease and mind so badly affected that if he got a short distance from his house he was unable to find his way back. Was seen and treated by me for pellagra during the spring and summer of 1907. Had all the typical symptoms, profuse erythema over both hands, the scars of the ulceration you can yet see. Treatment: Fluid extract Hydrastis fifteen drops before meals and ten drops of nitro-muriatic acid after meals. Plenty of milk, eggs, orange juice and vegetable broths. Strong leucodescent lamp applied to his hands and over the region of his stomach until the skin was reddened. This light treatment was applied by Dr. J. G. Johnston. He has been out of doors during the summer of 1908-1909, during which time he has taken no treatment, yet has not had the return of the slighest symptom. Has never eaten any more corn bread.

Of the fourteen cases seen this year the proportion of males has increased to the same as that of females, seven of each. Of these two females (white) and one male (colored) have died. Of the 24 cases four only were negroes. Three of these I prescribed for during the summer of 1908, as suspected cases of pellagra, only to

have my suspicions verified the following spring. The rash is not so distinct or as easily recognized as in the whites.

From these cases it seems that no age or sex or race is exempt. In my earlier experience women seemed to be more affected than men, and not until this year did I see a negro with the disease. It may be that the negroes have a greater immunity, as we all know they eat more corn bread than the whites.

The course of the disease has varied from three years to a few weeks. The most acute case being Mrs. B., with this history:

Mrs. B., age, 20. Nursing baby 16 months. Good family history. Developed diarrhœa and sore mouth in August, and was sick about three weeks when a rash appeared on her hands. Rash was severe and extended high up arm. After paying her a few calls, thinking it a simple diarrhœa, was surprised to find it a pellagrous rash when I was called in again. Her baby was weaned and is now in perfect health. This is the most acute case I have seen as the prodromal symptoms were not longer than three weeks. Treatment: Fowler's solution and Hydrogen Peroxide. In two weeks the sore mouth and rash was gone and she seemed well as you will see by photo. (Exhibit photo). She does not eat any more corn bread.

In regard to treatment will say that atoxyl was given widely this year, but not hypodermically, as these patients were not directly under an attendant capable of giving these injections. The results have not been superior in my hand to Fowler's Solution and Hydrogen Peroxide. The leucodescent light had the most beneficial effect on the erythema of any remedy tried. The two little boys, whose histories are here attached, were treated with Fowler's Solution, the older one receiving Atoxyl in the beginning and later changing to Fowler's Solution and Hydrogen Peroxide, with equal or more benefit. The younger was treated entirely with Fowler's Solution and Hydrogen Peroxide.

L. P., age nine. Father and mother both healthy. Bowel trouble began in March, 1909, also sore mouth. History of some bowel trouble in summer of 1908. Skin eruption first seen in June. Rash on hands, feet, legs, and a ring around his neck like a collar. Treatment Atoxyl and Hydrogen Peroxide, later Fowler's Solution. Much improved.

J. P., age five. Bowel trouble began about last of July. Skin eruption came on the last of August; very distinct on hands, feet

and legs. Both boys ate toll corn last year, and shipped meal this year. Treatment: Fowler's Solution. Very much improved.

The only case treated with Soamin was Mr. McD., who was treated with it here in the hospital, and it is being continued once a week in my office.

W. S. McD., age 40. Farmer. Had rash on hands and sore mouth, but not much diarrhœa during summer of 1906-1907. Was diagnosed as pellagra by me in spring of 1908. Had some treatment, but not continuous. During spring of 1909 the sore mouth and rash on hand was much worse. Was treated with Atoxyl and Hydrogen Peroxide without benefit. His mental symptoms becoming so bad that on April 10 he was committed to State Hospital. Here he received Soamin and now you see him today apparently well. During the spring of 1906 he ate meal from toll corn. Does not eat any more corn bread, but says he did eat some roasting ears while here in the hospital and it did him no harm.

Realizing that conclusions drawn from a few cases are apt to be fallacious, these opinions here expressed are in no way meant to be final, but are meant to be changed when the weight of evidence tips the balance in another direction.

DISCUSSION ON THE PAPER OF DR. McCONNELL.

DR. W. B. YOUNG, Rock Hill, South Carolina: I would like to ask Dr. McConnell what he used hydrogen peroxide for. What was his purpose?

DR. MCCONNELL: I used it at the suggestion of another physician as a mouthwash as well as internally, and I must say that it relieves the sore mouth in these cases better than anything I have tried.

SYMPTOMATOLOGY OF PELLAGRA AND REPORT OF CASES

J. J. WATSON, M. D.

COLUMBIA, S. C.

Definition: Pellagra is an endemic disease attributed to eating Indian corn infected with certain hyphomycetes. It is characterized by digestive disorders; symmetrical dermatitis on parts of the body exposed to the sun or subjected to constant pressure, various mental phenomena, principally depression, and in most cases by symptoms referable to spinal degeneration.

Symptoms: The malady is so insidious in its onset that it is difficult to state absolutely what are the earliest premonitory symptoms. The first thing usually complained of by the patients is some gastro-intestinal disorder; this may be loss of appetite, burning sensation in epigastrium, excessive desire for food or drink, or more often diarrhœa with more or less stomatitis and salivation.

Soon after these digestive disorders manifest themselves or coincident with them the patient detects a disinclination to any exertion, becomes irritable and sleepless. Duties that were formerly dispatched with a feeling of pleasure are now looked upon with a dread; and procrastination takes the place of habits of promptness; so that the personal appearance of the victim, or the aspect of the home and children, if the sufferer is a woman, indicates neglect; all of which is evidence of the psychic depression that forms later a prominent figure in the clinical picture of the disease. After these symptoms have persisted with usually increasing severity, in some cases for weeks, in others for months, the characteristic symptom of the disease almost invariably appears, namely: Erythema on the hands and arms not covered by the clothing.

The Skin Eruption: This may be either *dry* or *wet*.

Dry form: The eruption appears usually in the early spring months, and while it is the most characteristic objective feature of the disease, it is subjectively one of the least important as very few of the patients suffer any physical discomfort from it other than a burning sensation, complaining principally of the unsightlessness. The eruption commences as an erythema suggesting sunburn on the backs of the hands, and extensor surface of forearms, extending up the arms to the point that the sleeves reach, and ends abruptly there, being absolutely *symmetrical* if the sleeves are symmetrical,

which is usually the case. In persons who do not "hold their hands" the eruption extends to the flexor surface in a characteristic shape, commencing on the radial border it extends toward the ulner by an oblique line forming a patch of erythema on the flexor surface somewhat triangular in shape, the base of the triangle being the radius and the apex near the styloid process of ulner. Patients who are well advanced in the disease and unable to work, and who "sit about" and "hold their hands" have the eruption on the flexor surface, to the same extent as on the extensor. The erythema lasts for a few days and then commences to fade, and the skin to desquamate in fine scales, if the dermatitis has been mild. As the redness fades the site of the erythema assumes a somewhat cyanotic hue, this is gradually replaced by a characteristic light liver or chocolate color, which, if once seen, cannot be mistaken or confounded with any other skin disease. In negroes it is readily recognized by an increase of pigmentation, the site of the eruption appearing as though soot had been smeared on that part of the hand and arm.

The patients often make vain attempts to remove the *dirty* appearance of the hands with soap and water. After a time this eruption scales off and leaves the hands soft, velvety and *glistening,* quite a contrast to the dirty hands that they were the sorrowful possessors of only a few weeks before, but like Banquo's ghost, it has come to stay for a term; the erythema re-appears and the eruption follows the same course as previously described; this may happen several times during the summer. As a result of these repeated inflammations the skin of the hands become thickened, hard, wrinkled and inelastic, and frequently deep fissures form on the fingers, notably the index finger, at or near the first joint. Portions of the body covered by the clothing subjected to pressure also shows the eruption in some cases. The skin over the olecranon, trochanter, sacrum and knees being especially liable to become affected. The forehead, face and neck are often the seats of the eruption. In those who go barefooted the feet and legs do not escape. The eruption commences in the spring months and persists until July, August or September, and then disappears to make its appearance in the following spring. In some cases there is a mild relapse in October. *The crowning characteristics of the pellagra eruption are its symmetry and color.*

After the discolored skin has been exfoliated there usually remains a fringe of dirty appearing epithelium, a relic of the line of demar-

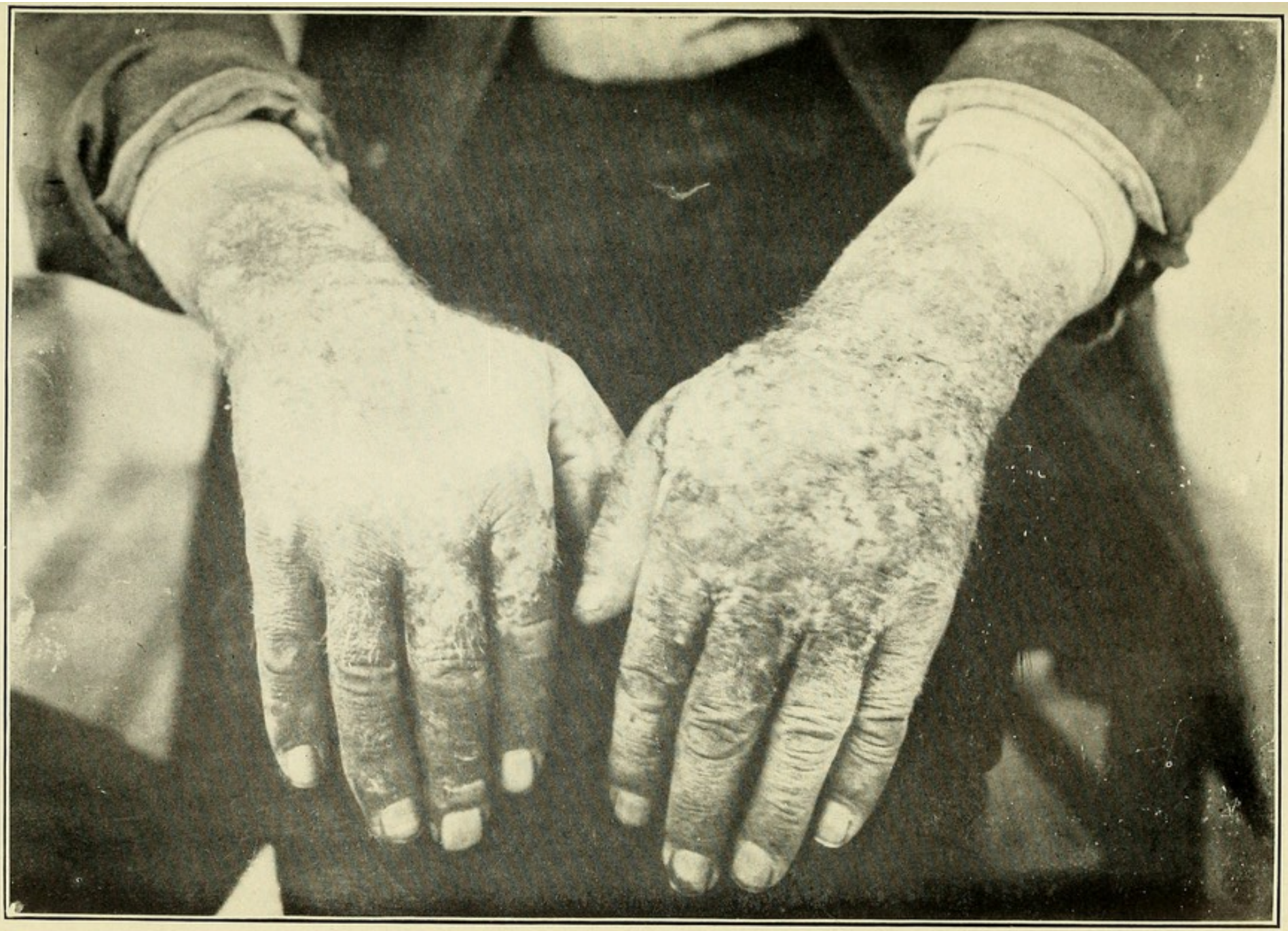

ILLUSTRATION NO. I.
Showing symmetry and color of pellagrous eruption. Desquamation on right hand.

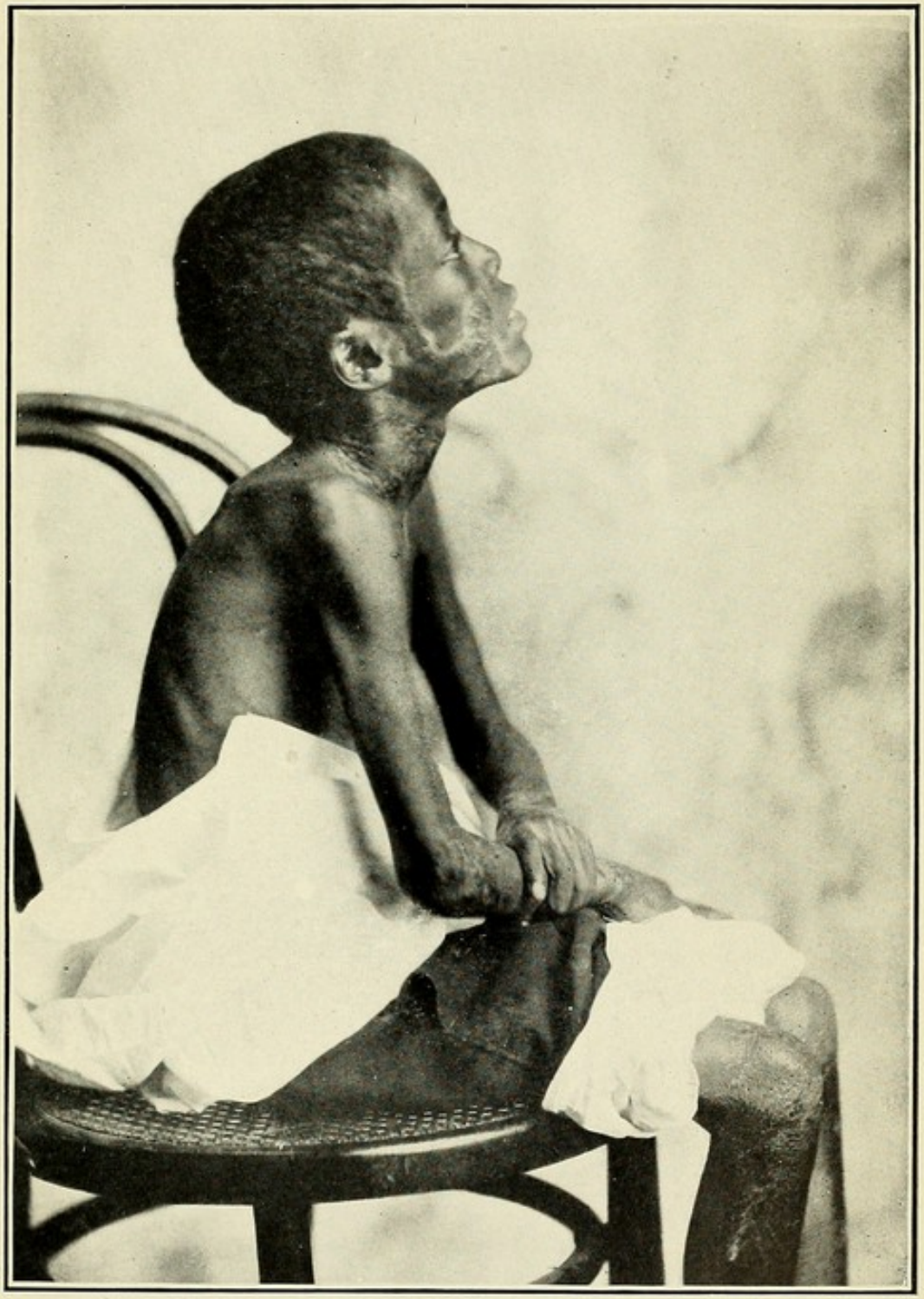

ILLUSTRATION NO. II.

Pellagrous eruption on face, neck, elbows, hands and knees. This case did not have the usual winter intermission of pellagrous syndromes for last three years of life. Died 10 days after this photograph was taken.

cation of the dermatitis. In some cases the sebaceous glands on and around the nose are hyperactive, there being a seborrhea. Purpuric spots are sometimes seen on the hands and face, a favorite site being under the eyes, here the spots have a crescentic shape and are symmetrical. On the hands these spots are sometimes on the palmer surface.

Wet form: This differs only in degree. Where the dermatitis is severe bullæ form, sometimes containing serous, serosanguineous or seropurulent fluid, when the bullæ break large ulcers are left to mark their site, or large flakes of skin desquamates, leaving a raw surface, slight cicatrices remain as an evidence of former ulceration. "The whole clinical puncture being analogous to a burn of the first degree." (Dr. Babcock). The wet form involves a greater area than the dry, such as the axillæ, groins, etc. This form has been frequently diagnosed as dermatitis exfoliativa. The eruption is an index of the severity of the disease; an extensive, severe eruption usually bespeaks a severe infection.

Digestive disorders: The buccal mucosa becomes very red, the tongue and mucosa of lips showing particularly this characteristic symptom. If the throat is examined this redness will be observed as far as one can see into the pharynx. Flakes of exfoliated epithelium will be seen adhering to the gums, and the tongue being denuded of its epithelium is smooth and glistening. Now its color being a cardinal red and it being a cardinal symptom of the disease, I have denominated it the *cardinal tongue.* Sandwith calls it the "bald tongue." This stomatitis is accompanied by a very profuse flow of thick saliva, in some cases so profuse that the saliva dribbles out the corners of the patient's mouth. Ulcers often form on the tongue. Intelligent patients suspect they have been salivated by mercury. When the mouth is opened strings of saliva will extend from the upper to the lower teeth. My attention was directed by Dr. Babcock over a year ago to small black or bluish black spots on the tongue, and since then I have observed these papilae in a number of cases, all negroes. The name of "stipple tongue" has been given this condition by Dr. Lavinder. The tongue may be either pointed and tremulous, or large, flabby, swollen and indented.

The salivary glands may be swollen and tender, this with the salivation and condition of the swollen gums has been mistaken for mercurial salivation, but just remember that in mercurial salivation there is always quite a disagreeable odor to the breath, and while there is an odor to the salivation in pellagra, it is not the same dis-

gusting foetid odor that is characteristic of the mercurial ptyalization.

The acme of the stomatitis corresponds to the acme of the eruption on hands. Other mucous membranes are inflamed, proctoscopic examination reveals a bright red mucous as far up the gut as can be seen.

Hemorrhoids are sometimes complained of. One of my patients thus complaining showed on examination only an intense redness of the mucosa, still she insisted that she suffered acutely from piles. This patient was then in a state of mild delirium, and probably there was some irritation that caused her to refer to trouble in that locality. She was far advanced in the disease, and this was the only symptom complained of. It is not unusual, however, for patients to complain of discomfort in this locality. The mucosa of the vagina is also a seat of inflammation and vulvovaginitis is not at all infrequent.

Stomach: Burning sensation in the œsophagus and stomach is quite frequently present. Pyrosis is sometimes a prominent feature with or without belching. "Pyrosis is never absent." (Lombroso.) Vomiting occasionally occurs, but is not a constant feature of the disease. When the disease is advanced dysphagia is complained of by some patients, and this may be accompanied by strangling when fluids are taken.

Marked gastric symptoms are in evidence in some cases. I have known a case of pellagra diagnosed as gastric cancer. The only abnormality detected by abdominal section was an excessive redness of the peritoneal coat of stomach. After a few days the patient was rolled out into the sun and soon there appeared on her forehead and hands an intense erythema. This aroused suspicion, and two competent consultants were called in. From the history of repeated attacks of eruption and the picture presented by patient, the diagnosis of pellagra was made and the subsequent course of eruption (color, etc.), tongue, diarrhœa and depression, put the diagnosis beyond doubt. Hematemesis is sometimes seen.

Diarrhoea: This is a feature of the disease at some time in its course. It varies from a few soft stools a day to twenty or more, sometimes they contain blood and mucous; they are frequently involuntary when the patients are bedridden. In a number of cases I have noticed that the stools are as frequent at night as in the day. The diarrhœa is *obstinate* and not affected by the ordinary treatment or diet. I have seen it persist in spite of large doses of bismuth and

opium, and a rigid diet, and improve when drugs were discontinued and diet not restricted. It is not dependent upon errors in diet, but is a neurophathic manifestation due to disease of the spinal cord and the sympathetic system.

The fact that the normal reflex in the intestines is greatly increased by the pathological changes in these structures may explain the diarrhœa; since it produces hyper peristalsis, in the same manner that irritants applied to the skin produce an exaggerated vasomotor dilatation with the erythema as a result.

In rare cases there may be constipation. These cases are very mild and show very slight mental depression or none at all. In the terminal stage when temperature sets in the diarrhoea sometimes stops. Hemorrhages from the bowels may occur. Meteroism is present with the diarrhoea and sometimes persists after the bowels have lost their frequent action. The diarrhoea follows the same course as the erythema and stomatitis, i. e., it has exacerbations and remissions and persist through the spring and summer months, with a slight recrudescence in October, then disappears during winter to re-appear the following spring.

Pupils: Pupillary abnormalities are quite striking in some localities and in some seasons, varying in the same locality from year to year. In the year 1908 in South Carolina, pupillary dilatation was the rule, the mydriasis being extreme in some cases. It may be either bilateral or unilateral, if unilateral the right pupil is most apt to be dilated. During this year, 1909, I have seen few cases with dilated pupils, contraction of pupils is sometimes met with. The pupils react sluggishly to both light and accommodation and "resist the action of homatropin considerably longer than the normal." (Dr. Whaley). Diplopia and photophobia are not unusual. These pupillary phenomena must be due to a disturbance in the cilio spinal center. This center is situated in the spinal cord between the first cervical and second dorsal nerves, the portion of the cord that is nearly always affected in pellagra.

Pain in the back: This was a striking feature in some of the Italian cases that I saw, the pain being so severe that the sufferers walked stooped over, this attitude furnishing one of the seven varieties of the disease described in the Italian proverb. I have had only a few patients to complain of pain in back among the number studied in America. Pains in various portions of the body are often complained of. *Tenderness* at some point along the spinal column is almost constant. It is usually in the mid dorsal region and is easily

elicited by pressure with the fingers along the spine. The tenderness varies on the two sides, in some cases being more acute on the right. The tenderness is not over the spinous processes, but over the point where the nerves emerge from the canal.

Temperature and Pulse: The mild cases are practically afebrile, i. e., temperature less than 100. The morning temperature is often sub-normal 96 to 97 1-2. Temperatures of 102 to 108, or over, are not unusual in cases progressing to a fatal termination from so-called typhoid pellagra.

The pulse is accelerated 80 to 100 in ordinary cases, but increases with the toxaemia or temperature, and counts of 160 are not unusual in fatal cases. Very rapid respiration is occasionally encountered without any appreciable cause for it.

There is nothing characteristic about the urine, except that alkaline urine is a bad prognostic sign. (Lombroso).

The reflexes are usually exaggerated, though they may be normal, diminished or lost. Like other signs, they vary with locality and season. The patella reflex being especially more lively, the slightest touch eliciting a lively jerk, the knee jerk varies on the two sides, in some cases, being more lively on the right, the side that has the most exquisite spinal tenderness. In very severe cases, or rather those in which there are tetanic contractions, ankle clonus may be found. In those cases that are paretic the reflexes are abolished.

There is usually analgesia or anesthesia at the site of the eruption.

Vertigo is complained of by nearly all of the sufferers, and should always be asked about, if not mentioned by them. Its presence should always excite suspicion, and other pellagrous stigmata sought for.

Psychic Phenomena: Briefly. Mental depression is as constant as the erythema and diarrhoea, and varies from a mild case of the blues to severe melancholia. The patients seem to have "forgotten how to laugh." The poor sufferers imagine they have not a friend on earth, that even their own children and parents dislike them, and have some irreconcilable grievance against them. They are easily provoked to anger, and in many ways indicate lack of mental force. *Hallucinations* and *delusions* are sure to occur at some time in the disease, and no two patients will have the same delusions. In Italy 10 per cent. become insane. As yet we cannot form any opinion as to what portions of our patients will become insane, but

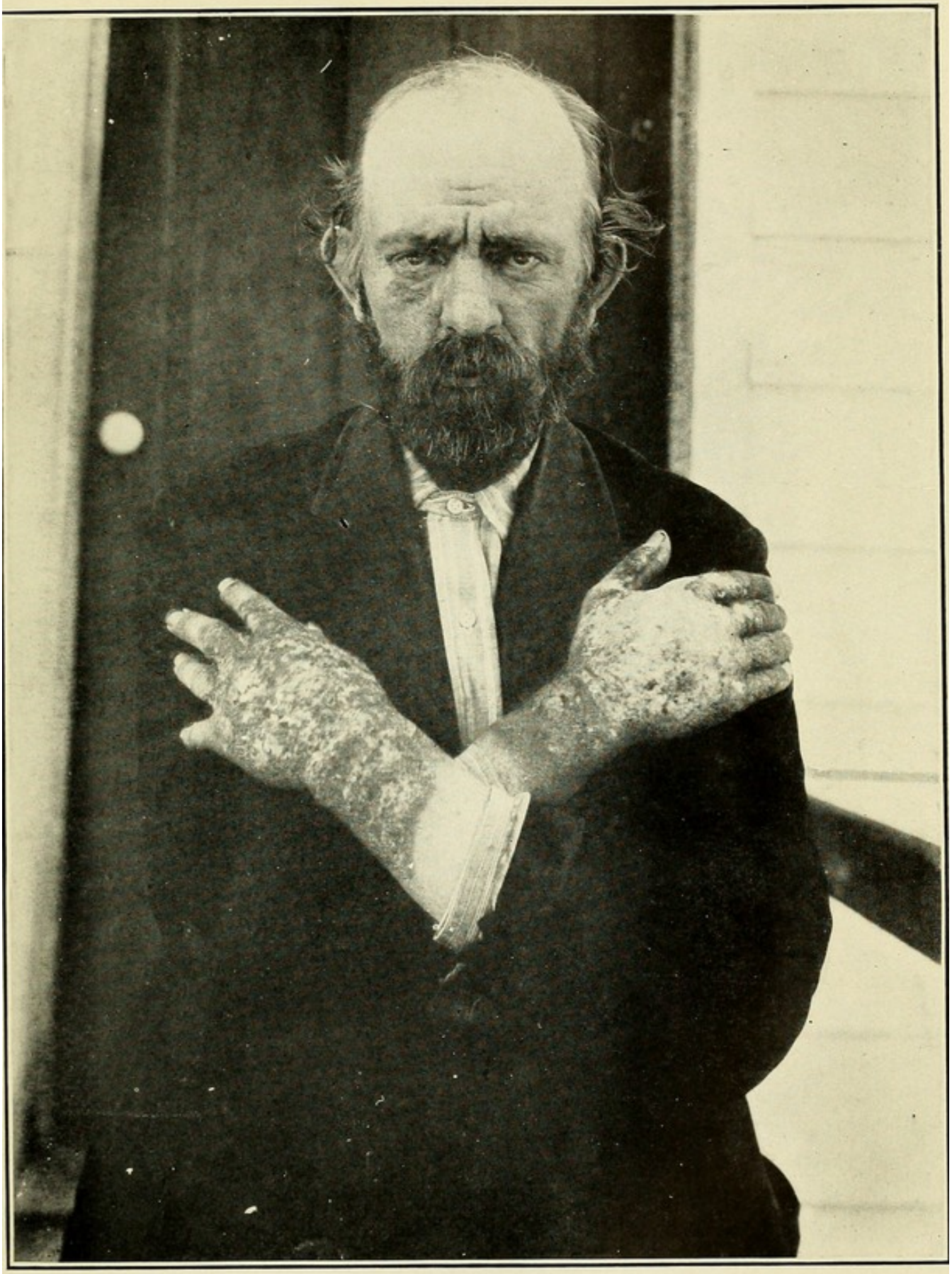

ILLUSTRATION NO. III.

Showing characteristic facial expression.

if statistics are properly kept it will be a very easy matter to ascertain what proportion is demented.

While pellagrins are never loquacious, at times they are complaining of real or imaginary ills. As the disease advances they talk less, often not answering questions, and finally pass into a state of absolute *mutism*. This portion of the pellagrous syndrome is very important, and pellagrous insanity properly calls for separate consideration.

Gait: The gait is either simple paralytic or paralytic spastic. The patients walk with their legs far apart, and as paresis sets in the stride is very much decreased, and the patient assumes a peculiar shuffling gait.

Case 1: Mrs. T., white, widow, no children, age 62, family history good. Always in good health until present illness, which commenced December 1st, 1907, with diarrhea and lack of energy. The diarrhea gradually became more severe and the motions were as frequent at night as they were during the day. About January 1st, 1908, mouth was quite sore and salivation extreme, mental depression and weakness gradually increased until she was forced to take her bed about May 1st. The eruption appeared on hands about March 15th, and was so severe that her physician thought she had gangrene; temperature ran for three weeks from 100 to 101. May 23rd admitted to Columbia Hospital, weight 89 pounds. Visceral examination negative. Utine normal, pulse 68, temperature 96 1-2 degrees. There existed on hands and fore-arms patches of pellagrous eruption, the dirty fringe being very noticeable. The palms of the hands were exfoliating and had been the seat of an intense dermatitis, bullæ having formed on them and contained sero pus; she endeavored to keep her hands concealed, as they were unsightly, and did not care for any one to touch them, fearing that she might contaminate them; there was also a patch of dermatitis on each elbow about the size of half-dollar. Tongue and buccal mucosa very red, saliva profuse and tenacious. Patella reflexes exaggerated, right most lively, pupils contracted, tenderness in mid dorsal region, right side more tender than left, vertigo on standing. She remained in hospital until November 30th, 1908. During that time she had frequent remissions and exacerbations of diarrhea, they being most severe from time of admission until about the first of August, when she gradually commenced to improve in her physical condition. The greatest number of stools was in July (16th), when fourteen were recorded in twenty-four hours. The stools were principally

involuntary from May 30th to August 3rd. Bed sore appeared June 12th. Nausea and vomiting June 4th, and continued until July 15th. All medicine, including bismuth and opium, was discontinued July 13th, and the 15th there was no nausea or vomiting. Herpes zoster developed September 16th. As her physical condition improved her mental condition became worse, she imagined she was covered with microbes and every day would have great numbers of fibers of lint and wool in a pus basin to show me "the things that were tormenting her to death." The latter part of November she became excited and it was thought best to transfer her where she could be restrained. She was sent to a private institution at the North and remained there until June of this year. There has been no recurrence of the diarrhoea or eruption; she is the picture of perfect health today, weighs 130 pounds.

She admits being very fond of corn bread, that she ate it every day; she also states that for months before she was taken sick that neither her corn bread nor hominy were properly cooked, the servant she had at that time being very careless.

Case 2: Mrs. S., age 31, white, married, three children living, four dead, no miscarriages, native of North Carolina, husband country merchant, distributing considerable quantities of shipped goods, including corn, by car loads. Health always good until four years ago, when she presented the pellagrous syndrome, diarrhoea, eruption on hands, stomatitis, salivation and vertigo. These lasted all spring and summer, disappearing during winter; they have returned each spring with increased severity. The first year the symptoms were noticed quite a number of young chickens in the yard lost their feathers, were very red, did not grow, and finally became ataxic and died. She was in a state of valetudinarianism practically for four years; being frequently accused by her husband of being hysterical, as she often cried, was apprehensive that something dreadful would happen to her. In February, 1909, complained of burning in stomach, later diarrhoea, eruption and salivation appeared, symptoms being more severe than any previous attack.

Condition June 13th, 1909, fairly well nourished woman. Facial expression that of melancholy, absolutely mute, patella reflexes absent. Pupils dilated, cardinal tongue, strings of saliva extends from upper to lower teeth when mouth is opened. Takes no interest in surroundings or conversation, thus being without insight. Remnants of pellagrous eruption on hands and elbows; has always eaten bought meal.

Hospital record: Upon admission was restless, deluded, probably subject to hallucinations, required constant watching. Full tub baths had a soothing effect for a time, following the baths, on the nervous manifestations.

June 14th was nervous, restless, noisy and deluded.

June 15th, same condition except stools frequent and involuntary.

June 16th, involuntary stools, but quiet.

June 17th, refused food, quiet.

June 18th, rested quietly. Bed sore.

June 19th, fairly quiet day, temperature and pulse both elevated and limbs slightly *rigid* and *tremulous,* low muttering delirium, continuous rectal irrigation was commenced. The temperature gradually rose and the *neuro muscular manifestations became more accentuated until the condition reminded one of strychnine poisoning.* Evidence of intense toxaemia were present, low muttering delirium, carphology, subsultus and dry tongue. All of these symptoms continued with increasing severity until the morning of the 24th, when death closed the scene.

Case 3: Before taking up the anamnesis of this case it will be necessary to review the family history; although the patients were not seen by me.

L. C., male, age 11, parents living, father being a miller. One sister died three years ago of pellagra at 14 years, and brother of same disease at 11 years, four years ago. Both died in August. The two fatal cases having developed the disease in their fifth and fourth years respectively. The disease manifested itself in the spring, and was supposed to be *ivy poisoning*. Remissions and exacerbations occurred during the spring and summer, and in winter the dermatitis and diarrhoea were entirely absent. The disease increased in severity for six years in the case of the boy till finally, after being unable to walk for two months, he was confined to bed four months, and following one month of excessive vomiting and diarrhoea, died of exhaustion. On several occasions he vomited dark clotted blood. The girl had the disease mildly at first, sore mouth, diarrhoea, and eruption on hands and feet. With her the disease lasted nine years. She had vertigo, burning in stomach, pain in back of neck and between shoulders, head drawn back, high fever, was treated for "spinal trouble" and "hysteria." There was a tendency to stagger and fall backwards after any exertion. This was a striking feature of the case for the last two years of her life. There was no vomiting till shortly before death, nor was the diarrhoea so

severe as in her brother's case. In April, 1906, she complained of intense burning in stomach "like she was burning up." The eruption, stomatitis and diarrhea then appeared, and she soon became very weak and was confined to bed till August, when she died, evidently of tphoid pellagra. For about two weeks before death she had convulsions with increasing frequency. Hemorrhages from bowels. There was opisthotonos arms and legs rigid, mouth quite tremulous "like she was chewing," sublustus marked, and for the last week of life, temperature was "very high."

These histories are obtained from the father and mother. Furthermore, the father stated, that it was the habit of these children as soon as they were old enough to accompany him to his work, when playing about the mill-house, frequently to eat raw meal hot from the rock. The first year that either of the children was affected, he remembers that the local corn crop was practically a failure the previous year, and that he ground a great deal of shipped corn that he knew was damaged, spoiled or rotten.

So far as can be learned neither the father nor the mother has at any time had the symptoms of a disease that suggests pellagra. It should also be recorded that the disease was not recognized as pellagra during the lives of these two children. Furthermore, the father states that so far as possible he selected the best corn only for his family use. This may explain why the parents escaped, while the children ate raw meal of any sort and contracted pellagra.

L. C., male, age 11, brother of the above described cases. He developed the disease at two years of age. The pellagrous syndrome, dermatitis, stomatitis and diarrhoea appearing every year in the spring. The diarrhoea has never been severe, three or four actions a day in the early spring months. He has a tendency to stoop forward, and when tired falls down on his all-fours.

Physical Examination: A fairly well nourished, but small boy, mentality low for his age, pupils moderately dilated but not as much as they were in June. Patella reflexes aggravated. Not mentally depressed. Sometimes plays as other children do, and again is unusually quiet.

The accompanying photograph will show the extent of the eruption.

Case 4: Miss C. C. S., age 25, white woman. Aunt on mother's side had "nervous spells." Family history otherwise negative.

Previous health good. Was taken sick in March, 1904. First

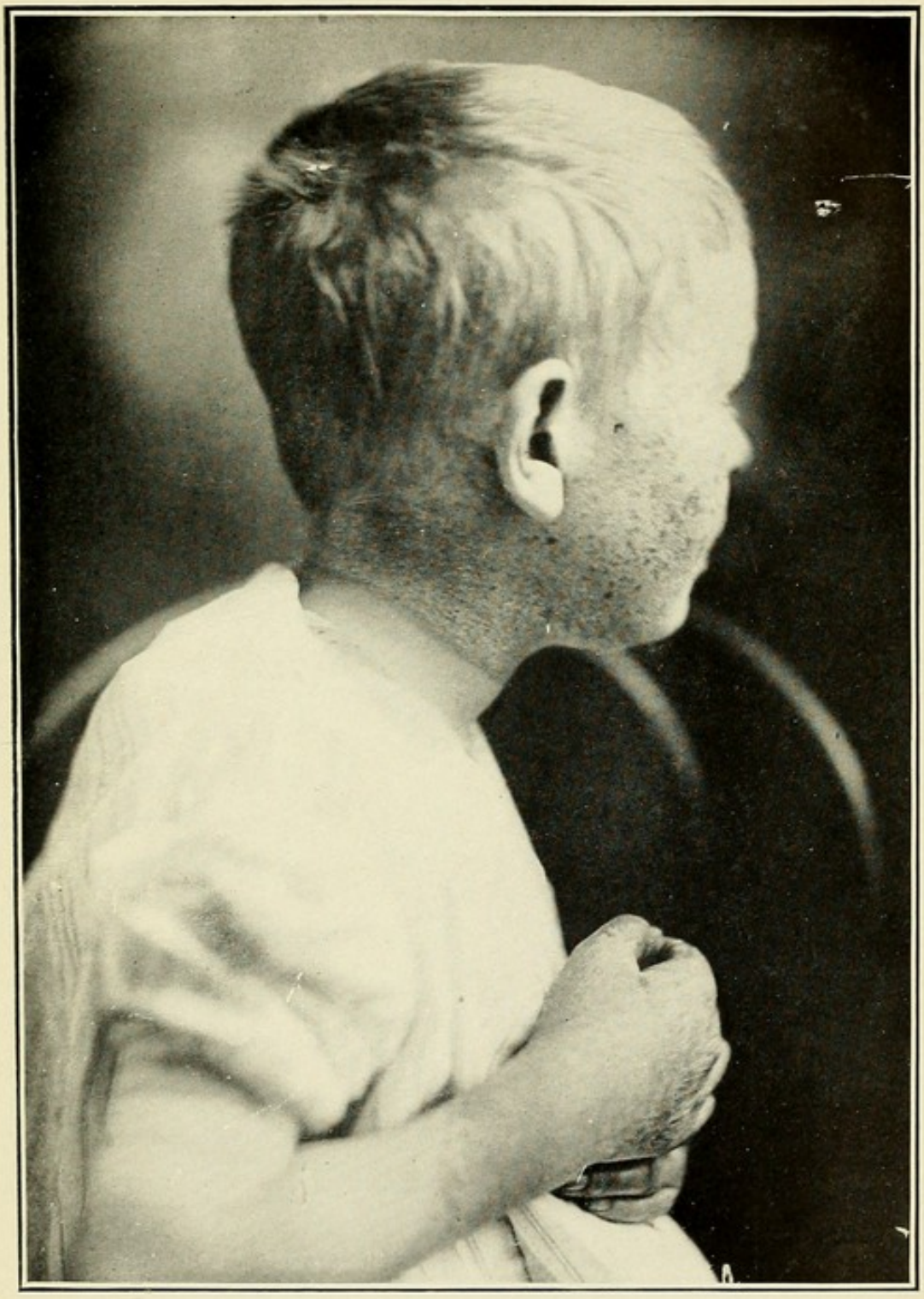

ILLUSTRATION NO. IV.

Case III., S. C.—Showing pellagrous eruption on face. Note line of demarkation on neck.

symptom was indigestion, diarrhoea and burning pain in stomach and "smothering feeling." Could not stand to look at water or hear it splash, as it would make her very nervous, blind and dizzy, and cause her to have shortness of breath. Was *very thirsty,* but was almost prohibited from drinking, by the sensation produced by the sight of water, and only when thirst could be endured no longer would she force herself to swallow a few mouthfuls. Mouth was *very red* and sore, with quantities of thick saliva.

Eruption came on hands and arms about two months after onset of diarrhoea, and one month later came on face. These symptoms continued until September, when improvement set in and she thought she was well.

In spring of 1905 there was a repetition of all the symptoms, but more severe—well during winter.

In spring, 1906, light attack, except mental depression was more pronounced.

These exacerbations and remissions have occurred each year since. In 1907 the diarrhoea was more severe than at any other period of the disease, the bowels acting as often as twenty times a day, for days at a time.

March, 1908, all symptoms returned, eruption being more extensive than any previous attack. Mental depression marked, vertigo severe. Had a desire "to run and go away hundreds of miles." Would be unable to sleep at night, and would get up and walk around house for hours and not stop until forced to do so from exhaustion.

Seen by me July 16th, 1908.

Examination: Emaciated woman, apparently 35 years old. Heart and lungs normal, abdomen negative, except for slight meteorism. Radical arteries palpable. Pellagrous eruption on hands, arms, elbows and below elbows, neck, face and chest. The extensive distribution in this case was due to her wearing a very thin shirtwaist; although extensive the eruption was symmetrical. Tongue a cardinal red and bald, abundance of thick saliva. Pupils widely dilated, reflexes exaggerated, marked tenderness in mid dorsal region. Answers questions intelligently and promptly. She was advised to give up all products of corn in diet. No medicine prescribed.

Was seen in May, 1909, was then the picture of health and had gained 25 pounds. Bland's pills with atoxyl prescribed.

On May 20th a slight diarrhoea set in and two weeks later the eruption appeared.

The following report was received from her September 29th: Entirely free of eruption, disappeared two weeks ago, has been very slight this year. Has kept out of sun, every time she went out the eruption would come back. Bowels have not been loose, rather constipated. Mouth has been very sore, as though "burnt with lye." Troubled with sleeplessness, complains of her stomach and feeling weak; though for the last two weeks that has been relieved to a great extent. Some months ago had a very profuse "flow" lasting three weeks, "almost a hemorrhage."

DISCUSSION ON THE PAPER OF DR. WATSON

Dr. C. L. Minor, Asheville, N. C.: In considering the symptomatology Dr. Watson failed to mention one symptom which I regard as of the greatest value, namely, the peculiar slimy greenish stools with a peculiar, and, I believe characteristic, sickly sweet odor which our nurses have noted in all the cases.

AMEBAE IN THE STOOLS OF PELLAGRINS

WILLIAM ALLEN, M. D.

CHARLOTTE, N. C.

Amebiasis is endemic in Mecklenburg County, North Carolina, and I believe it is very widespread throughout the South. In Charlotte our death rate from amebiasis constantly exceeds that from typhoid fever. Hence in a series of cases of any disease we should expect to find amebiasis as a complication in a certain per cent. of them. That this is true of pellagra is shown by the following case:

Mr. S. O., white, male, age 69, was reported[1] in February, 1909, as a case of amebiasis. Two weeks rest in bed, with liquid diet, large doses of ipecac and colonic irrigations reduced the number of stools from 12 to 6 daily. During this time the occurrence of blood in the stools became rare and amebæ entirely disappeared. But the patient still averaged six stools a day. Some three weeks later skin lesions pathognomonic of pellagra appeared and explained the disappointing results obtained by our previous treatment.

The high percentage (5 out of 7 Pellagrins examined) that show monads in their stools would indicate that these cases are unusually liable to intestinal parasitic infection.

The finding of amebæ in the stools is variously interpreted by different observers. Probably pathogenic amebæ at times inhabit the lumen of the bowel without attacking the mucous membrane; and there are probably species of amebæ inhabiting the lumen of the bowel which are unable to attack the mucous membrane, although Musgrave and Clegg[3] deny this. Quinke and Roos[2] describe three (3) species inhabiting the human intestine, two of which they consider pathogenic. Celli and Fiocca[2] describe five (5) species, all presumably pathogenic. But Calkins[4] very clearly points out that a species of amebæ can be identified only by following out its entire life cycle. This the average practitioner is not qualified to do. However, two species occurring in the human intestine have been thus carefully studied by Shaudinn[5] and named by him entameba coli and entameba histolytica.

In examining stools containing amebæ we should expect to see all shapes and sizes of amebæ from budding youth to encysted old age. Hence it is impossible, particularly in the case of small amebæ, to assign them to any given species or to say whether or not they are

patheogenic, without tedious experimental work. However, I wish to record, merely as a matter of interest, the finding of small ameboid organisms in the stools of four out of five cases of pellagra that have been carefully studied. These organisms measure .010-.015mm in diameter, or a little larger than a red blood corpuscle. Their size necessitated a magnification of 600 diameters or more for satisfactory observation. There were no larger amebæ present; no nucleus, vacuoles, or included particles could be made out. The cytoplasm was semi-transparent, finely granular and lighter than that of a red blood corpuscle. The pseudopodia consisted of ectosarc of clearer material than the cell cytoplasm. Whether or not these organisms belong to the amebæ family, or are monads in the pre-fusion stage,[6] or are ameboid cells from some other source I cannot say.

The danger of mistaking amebiasis for pellagra and vice-versa is considerable. For example, a few weeks ago I studied an epidemic of amebiasis in which all the cases showed sore mouths and most of them diarrhœa. When the first case was seen a tentative diagnosis of pellagra was made. As soon as it became evident that the trouble was epidemic the stools were examined and entameba histolytica demonstrated. Conversely a few days ago a case was brought to me to confirm the diagnosis of pellagra. There was a slimy fissured tongue and rough thickened skin over the ankles, moderate diarrhœa and neurasthenia. On closer examination the skin lesion was found to be non-pigmented and itched, as distinguished from the burning or tingling of pellagra. This was considered a dry eczema. A differential blood count showed eosinophilia 8 per cent., which is almost invariably pathognomonic of parasites in this latitude.

The symptoms of pellagra and amebiasis are at times so much alike that it is sometimes very difficult to tell with which disease we are dealing. I have before called attention to the fact that in both of these conditions the appearance of the mouth and tongue are similar.[1] In both there are all grades of diarrhœa; in amebiasis, depression and neurasthenia often entirely overshadow the other symptoms of intestinal parasitism[7]; in both there may be marked emaciation. As long as the etiology of pellagra is unknown the treatment must necessarily be largely symptomatic. Therefore we are not justified in diagnosing and treating as pellagra cases that show only sore mouth, diarrhœa, emaciation and melancholia until

amebiasis has first been excluded. This is readily accomplished by a differential blood count and an examination of the feces.

SUMMARY.

1st. Pellagra is often complicated by amebiasis.

2d. Many varieties of amebæ may be found in the feces of pellagrins.

3d. Pellagra is liable to be mistaken for amebiasis and vice-versa.

4th. In diagnosing and treating pellagra in the absence of pathognomonic skin lesions or grave mental symptoms it is first necessary to exclude amebiasis.

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DISCUSSION ON THE PAPER OF DR. ALLEN

Capt. H. J. Nichols, U. S. Army: Dr. Allen's paper is very important in that he raises the point of the diagnosis of pellagra without skin symptoms. For scientific purposes in the study of our cases we ought to eliminate those which do not show skin symptoms. It may occasionally be necessary to diagnose pellagra without skin symptoms, but it is not safe at this stage of our knowledge of this disease, because there are so many other diseases, especially sprue, which follow somewhat the same course which Dr. Allen referred to in cases of amœbic dysentery. Cases of sprue can be easily stretched into a diagnosis of pellagra, if we do not insist on skin symptoms. This paper has a bearing also upon the mortality of the disease. There are a great many deaths attributed to pellagra, which, in my opinion, ought to be ascribed to other diseases, especially dysentery. We should always endeavor to determine how much other disease exists in our pellagrous patients.

Dr. Allen (closing the discussion): At present our health authorities in the South do not recognize amœbic dysentery as a reportable disease. In North Carolina, for instance, there are no records of deaths from amœbic dysentery, and while we see a great deal of amœbiosis and it is invariably reported as something else.

RESULTS OF STOMACH ANALYSES IN PELLAGRA

W. O. NISBET, M. D.

CHARLOTTE, N. C.

I desire to report results of the analysis of stomach contents of ten cases of pellagra. All of the cases were given the Ewald-Boas breakfast of one roll and one and a half glasses of water and contents were removed by tube one hour after ingestion. The acidity was estimated by Topfer's method of titration. A detailed account of the cases will not be given; only the stage of the disease and the points bearing on the stomach secretion will be mentioned. My experience is limited to twenty-five cases of pellagra and in only ten of this twenty-five did I succeed in getting test meals.

Case 1: Miss W., native of Alabama, age 29. History of diarrhœa in spring of 1907. I saw patient first in September, 1908, and found present hand and wrist eczema, bald tongue, burning in stomach and diarrhoea, anal excoriations, slow mental action, despondency, but with reflexes about normal; emaciation to an extreme degree. Only one test was made of stomach contents and showed HCl, O; total acidity, 30; ferments not estimated; mucus in excess. Bile was present during the periods of severe vomiting. Motility not tested Patient died October, 1908.

Case 2: Miss C., age 26, native of South Carolina. First seen in September, 1908; gastro-intestinal and mental symptoms marked and eczema beginning on knuckles. Patient gave history sore mouth, nausea and diarrhoea in the spring of the preceding year. Analysis stomach contents HCl, 4; total acidity, 28; mucus, in excess; ferments, coagulation of milk delayed. Motility not tested. History of presence of bile during severe vomiting. Died six weeks later.

Case 3: Mrs. R., age 35; native of South Carolina. Had been very nervous for twelve months, and had raw mouth and tongue in the past spring. Saw first August, 1908, and presented characteristic gastro-intestinal signs with staggering gait, but no hand eczema; analysis stomach contents HCl, 35; total acidity, 80; ferments normal; mucus, no excess; bile present; motility, stomach empty two and half hours after test-breakfast. This patient got better during winter, but had a return of symptoms in following April; still no hand eruption. At this time a second stomach analysis

showed HCl, 10; total acidity, 40; mucus, no excess. The eczema appeared on hands and wrists in the following August. An analysis at this time gave: HCl, 4; total acidity, 28; mucus, no excess; bile absent; motility, normal. Patient still living.

Case 4: J. E., adult male, age 31; native of North Carolina. History of mild alcoholism and of spring diarrhoeal attacks for several years; mouth, stomach, bowel and cord signs present when seen in August, 1908. Analysis: HCl, 8; total acidity, 33; mucus, in excess; ferments, diminished; motility, normal; bile present during periods of vomiting. There was improvement during winter, but in May symptoms returned in increased severity and with the presence of the typical eczema of hands, arms and elbows. Analysis at this time gave following: HCl, 0; total acidity, 24; mucus, in excess; motility, normal. Patient died the following September.

Case 5: Mrs. W., age 36; native of North Carolina. Eczema and gastro-intestinal symptoms, marked; locomotion and reflexes, normal; mental action, sluggish. Result of analysis HCl, 4; T. A., 18; mucus, no excess; motility, normal; ferments, normal. Patent improved under arsenic, but had a mild recurrence the following spring.

Case 6: Mrs. P., age 34; native of North Carolina. Gastro-intestinal symptoms, with insomnia and vertigo appeared in fall of 1908. Analysis at this time gave: HCl, 24; T. A., 56; ferments, normal; motility, normal; mucus, excess. In June, 1908, the above mentioned symptoms returned in acute form along with decided hand eruption. Specimen of contents at this time gave no reaction for HCl, but there was excess of mucus. Patient died six weeks later.

Case 7: Mrs. E., age 38; native of North Carolina. Sore mouth, burning stomach and periodical diarrhoea appeared in September, 1907. Analysis at this time gave: HCl, 39; T. A., 76; mucus, not in excess; motility, normal. Patient improved under arsenic and cold weather, but in April had a return of symptoms, with the appearance of hand eruption in addition. Analysis: HCl, 20; T. A., 42; mucus, no excess; motility, normal. A third analysis in October gave result: HCl, 8; T. A., 22; mucus, in excess; motility, normal.

Case 8: Mrs. M., age 37; native of North Carolina. Sore mouth and gastro-intestinal signs first appeared in April, 1909. Insomnia and despondency at times for two years. In August, in addition to above symptoms, began to walk unsteadily and noted also the appearance of "sunburn" on knuckles and back of hand. Analysis at this

stage showed: HCl, 19; T. A., 40; ferments, normal; mucus, no excess.

Case 9: Mrs. B., age, 29; native of North Carolina. Hereditary specific history. Nursed a sister who died of pellagra. Six months ago developed red tongue, distress in stomach and nervousness, with despondency. At present has still the above symptoms and beginning eruption on knuckles. Analysis: HCl, 20; T. A., 50; mucus, no excess; motility, normal. This case may clear up under specific treatment and prove not pellagra.

Case 10: Mrs. M., age 39; native of North Carolina. Has had diarrhoea and indigestion for two years. In April of this year developed marked diarrhoea, gastric distress and a red mouth and tongue and case was pronounced pellagra by consulting physicians. At present time she is a typical pellagrin. Analysis: No HCl, nor total acidity; excess of mucus; bile present.

SUMMARY.

Cases 1, 2, 4, 6, 7, 10 show a marked diminution in the acid factors of the gastric juice in the late stage of the disease.

Cases 1, 2, 4, 5, 10 show excess of mucus during the pellagrous periods.

Cases 3, 4, 5, 6, 8, 9 show normal motility, while in 1, 2, 10 motility test was not made.

Cases 1, 2, 4, 10 show presence of bile during severe vomiting periods.

This series of cases is too small to allow us to draw any definite conclusions, but the indications are that in pellagra, as in all adynamic and asthenic diseases, the HCl and ferments of gastric juice progressively diminish.

PERSONAL OBSERVATIONS ON PELLAGRA

T. W. L. BAILEY, M. D.

CLINTON, S. C.

Mr. President and Gentlemen of the Convention: It is not my purpose to take up but a small bit of your valuable time in reporting to you the limited experience I have had with pellagra.

I wish only to give you a synopsis of the most striking symptoms as they occurred to me. Beginning with Case 1, which I observed about five years ago, at that time the correct diagnosis was not made, the symptoms so prominent in that case I have seen in almost all the cases since then. The cases usually begin with an acute diarrhœa; that is, the diarrhœa is the first symptom the physician is called upon to treat. This diarrhœa is very persistent and does not yield to ordinary medication that usually is given in a deranged bowels that is caused from errors in diet. The stools are similar to an acute diarrhœa, but persists for a longer period of time and does not usually assume the catarrhal or dysentery type that follows an ordinary spring diarrhœa. The diarrhœa persists for several weeks, having from 12 to 20 stools daily. In a few weeks after the onset of the disease we have an acute stomatitis, a smooth, plain inflamed condition of the mucous membrane of the mouth, lips, ducal membrane, also extending down in the eosophagus, sometimes a gastritis. I observed this inflammation in the vagina and anus in one of my patients. The vaginitis was a source of great annoyance, producing an irritation and caused pain on passing water. There is a peculiar but a typical eruption or erythema develops symmetrically on the back of the hands, this extends up the arms as far as the sleeve reached. This eruption, in all my cases, did not have a tendency to spread around on the flexor side of the arm except in one case I saw recently in consultation.

I may say the eruption has a characteristic different from any eczema that I have seen. It first usually occurs suddenly as a sunburn, on the back of both hands, very red and almost the appearance of a blister. This erythema or eczema does not cause pain or itching. After a few days the hand shows a peeling or exfoliation of the skin, then you have a scaly and irregular appearance of the skin. This scaly condition goes to a mark that would outline the

beginning of the palmar surface of the hands. At this line it presents a stained appearance, as the patient had stained them walnut or persimmon stain. This discoloration cannot be washed off.

I noticed in a case that is two or more years' standing there is a deep fissure, only on each of the index fingers and at the first phalanx. There are many small cracks or fissures on the backs of the hand, but the index finger fissure is very prominent. The eruption is not found under the clothing. You find a symmetrical patch on the sides of the neck, the molar bones or cheek, and sometimes the tip of the nose. The tongue, as the case progresses in all the cases I have seen, is very typical, that is all tongues look so much alike. The tongue is highly red, not a scarlet tongue appearance, not the *prominent papillae,* but a clean, slick, red tongue with a venous or slightly purple hue, and this characteristic does not vary very much for months or perhaps through the entire course of the disease. I have not found a coated or furred tongue with my patients. It is a disease of chronic persistent gastric disturbance; the patient soon learns that he has to be careful about his diet, and, of course, in some cases the appetite is inordinate, and frequently take food that disagrees and provokes an exacerbation of the symptoms. I have one patient who has an insatiable appetite for corn bread, and at times would take small pieces of corn bread, but invariably would follow an aggravated diarrhœa and eczema. Other articles of hard food to digest would also provoke the same symptoms.

Progressive emaciation, though slow in most cases, is always noticed. This is a *feverless* disease *per se.* I have never found the patient with fever unless you had a plain complication with it.

I have been astonished to find the heart's action so nearly perfect in this disease. The rate has persisted in remaining practically normal, even after marked emaciation had developed.

Nervous Symptoms: Insomnia is the most prominent symptom that develops first of the nervous phenomena, and this comes with the most of the patients and early with the disease. Latterly hallucinations on various subjects occur. A case under my care last year, after the typical eruption had subsided, had a severe attack of hallucinations and continued for a week, and I may say I was greatly alarmed over this case as to recovery; is now in very good apparent health, able to attend to domestic duties, with no return of eczema or glossitis, though she lives in dread or apprehension, and is more or less nervous, has poor digestion and has the hyperasthetic knee jerk. I am waiting with a deal of interest to see the outcome of this

case. The case I had five years ago developed locomotor ataxia and acute insanity and died.

Most of my patients develop a careless way and present a dejected or despaired appearance.

All the patients I have observed are past 30 years old. Have had four females and two males—all white except one colored female.

Now, just a word of synopsis:

1st. The acute uncontrollable diarrhœa and its persistence.

2nd. Salivation with stomatitis and involving the eosophagus, the characteristic slick red tongue that does not coat.

3rd. One case of vaginitis and inflamed anus.

4th. The peculiar eruption on back of hands, sides of neck, both cheeks and sometimes tip of the nose.

5th. The feverless disease.

6th. The hyperasthetic knee jerk occurring primarily in all cases, which should always be examined.

7th. Insomnia, hallucinations and acute insanity. One of my cases developed locomotor ataxia and insanity.

FURTHER OBSERVATIONS ON PELLAGRA WITH POINTS ON PROGNOSIS

JAMES H. RANDOLPH, M. D., AND RALPH N. GREEN, M. D.

Florida Hospital for Indigent Insane

CHATTAHOOCHEE, FLA.

Several years ago there came to a young physician a man who sought advice for relief from a skin disease with which he had been afflicted for a number of years. The patient was beyond the middle age of life and was noted for his eccentricities and slothful habits. Among the latter was a recently acquired taste for alcoholic liquors; and, being a patriotic citizen, he adopted the favorite beverage of the community in which he lived, namely, corn whiskey. He rapidly passed from a state of comparative financial security to one of want for the everyday necessities of life; and upon examination presented an extremely poor physique. The skin disease manifested itself in the form of a rather brilliant erythema, involving first the hands and face, and later the extremities. The sun's rays seemed to aggravate the case, and hence the usual agricultural pursuits upon which the man depended for support had to be abandoned early in the farming season. He was given an ointment consisting largely of iodide of starch and zinc oxide, and advised to suppress his bacchanalian desires and limit his epicurean tendencies to the popular Florida diet of white bacon, hominy and corn bread, and, in addition, to spend some weeks in frequent bathing at the Hampton Sulphur Springs, of Taylor County, Florida. This advice was followed religiously, and as the fall season approached the patient returned to his home apparently much improved. During the early winter months, however, the poor fellow became melancholy, and after a bitter quarrel with his brother, which came near being a tragedy, ended his life by committing suicide.

Today the doctor who in his ignorance treated the case as one of vesicular erythematous eczema, realizes the true nature of the malady which he sought to cure; and further knows that his advice as to diet was little better than adding fuel to the flames of pellagra already lighted in his patient. And now, almost under the shadow of Sand Mountain, and its famous beverage derived from maize, is accorded the great honor of listening to a discussion of this interest-

ing disease by a body of most eminent pellagrologists, and in the few remarks to follow, as one of the essayists, I lay no claim to any new or startling discoveries, but merely wish to report the further occurrence of this new disease at the Florida Hospital for Indigent Insane, and present the result of our observations thereupon since the original report from one of us (Dr. Randolph) at the meeting of the Southern Medical Association in Atlanta last year.

Incidence: Since that time, and for a period of only the last seven months, during which another of the essayists (Dr. Green) has been in charge of the women's department of the institution, there have been among a total of eighty-five women patients admitted (black and white) eleven cases of pellagra—five among the white and six among the colored. Among the men during the same period and extending back even further, to the time of the first report one year ago, there have been found but two cases in a total of over one hundred and eighty admissions divided equally between white and colored, and these two cases were among the negroes. The figures would seem to support the interesting observations of others that there exists for women a marked susceptibility over and above that to be found among the men; and to a less degree, also a similar predisposition on the part of the negroes as compared with the whites. Out of the total of thirteen cases here noted, all but four presented the classic symptoms of the disease upon admission; and of the four who subsequently developed symptoms, only two had been here sufficiently long to warrant the assumption that they may have contracted the disease within our portals.

Distribution: The disease does not seem to be confined to any one section or locality in the State; our cases having been received from fourteen different counties, scattered irregularly through Florida from one extreme to the other. Hillsborough, Dade, Duval and Gadsden Counties contributing two each, while Escambia, Washington, Jefferson, Columbia, Alachus, Bradford, St. Johns, Pasco, Manatee and Desoto are represented by one each.

In the social scale, the five cases observed among the white women represented as many different walks of life, from a pauper epileptic idiot to a highly educated elderly woman of refinement now in a stage of terminal dementia, and includes a farmer's wife, a mechanic's wife and a prostitute. Among the colored women the patients were recruited from the turpentine camps of the State, where there is a notable absence of general or personal hygiene; and one of these cases had been employed in packing leaf tobacco, but inquiry revealed the fact that she had long been a simple-minded,

dissipated individual, addicted to the use of snuff and "nigger gin." With the men the same diversity of social conditions exist if the cases formerly reported be taken into consideration, among which were noted a lawyer, mechanic, carpenter, farmer and negro turpentine laborer.

The ages of the patients that have come under our observation range from that of a child thirteen years old to that of an aged woman (also one negro man) over eighty years of age; the majority, however, being around forty.

Etiological Factors: In all our cases a history of maize diet in some form is obtainable, though not as much importance seemed to attach to this in some as in others, and in all there were to be found contributing factors, such as alcoholism, syphilis, tuberculosis and the naturally lowered vitality of old age with or without long continued mental disturbance. In the light of our present experience and familiarity with the quality and nature of the corn products used in this State, we are inclined to believe that not only may the imported Western varieties of corn be responsible for this condition in our people, but likewise the native-grown crops, which have greatly deteriorated in later years with the advent of the thriftless negro farmer to replace the "old-time" methods of the intelligent planter in the days "befo de wah."

Symptomatology: The symptoms observed have not differed in our experience from the usual classic description of the disease. Depressed or melancholic states predominate, with persecutory and somatic delusions, confusion and a marked tendency to burrow under the bed clothes, which has been observed with some of our patients. Those cases which succumbed rapidly developed a state of profound delirium, facial rigidity, mouth tremors, exaggerated reflexes and profuse diarrhœa; but none showed the convulsive tendencies described by Lombroso for his "Typhus Pellagrosus." We continue to note the occurence of the erythema in the fall as well as spring months, and it would seem to be even a more constant feature in the former season than the latter. One of the white women presented at first the skin lesion only on the inner and dorsal surfaces of the thumb and index finger, but more later the eruption appeared on the face, and, together with the typical stomatitis and gastro-enteritis, left but little doubt in our minds as to the correctness of the diagnosis. Dr. J. S. Norman, clinical assistant at this institution, has called attention to the peculiar mole-skin appearance of the

epidermis in the negro, which he suggests may be a precursor of the later inflammatory condition of the hands, etc.

The temperature, pulse and respiration show no great divergence from the normal curve, except in those cases in extremis, or complicated by myocarditis, tuberculosis, etc.

Repeated examination of stools has failed to reveal the presence of intestinal parasitic infection; and, in connection with this, eosinophilia has been notably absent.

Pathology: In our cases, post-mortem findings have been considerably marked by the presence of advanced lesions of tuberculosis; but the gross appearance in the cord and brain is not materially different from that described by other investigators, including the glistening dura, thickened pia and hard small placques in the arachnoid, as mentioned by Lombroso, and which have been found with striking uniformity in two of our cases. Sections from these tracts have been forwarded to the State laboratory, but as yet mounted specimens and microscopic diagnosis have not had time to reach us.

Differential leucocyte count in two of the women (apparently uncomplicated cases) has been instituted, and would seem to show a preponderance of small mono-nuclears—though these investigations have not as yet progressed sufficiently to prove conclusive.

Diagnosis: So much has appeared in the literature of the past year regarding the differential points in the diagnosis of this disease that little difficulty should now be experienced in its recognition; though for the more rare and obscure conditions described by European writers as "False Pellagra" and "Pellagra *sine* Pellagra," there remains much for further investigation and demonstration. In our own cases the diagnosis of pellagra is considered assured only after the appearance of cardinal symptoms—diarrhœa, dermatitis and dementia—as first described in this country by Dr. Babcock. With regard to the so-called pellagra *sine* pellagra (or pellagra without the dermatitis) we confess to a grave doubt whether such a condition has not been mistaken for sprue, which, with the exception of the skin lesions, bears a striking resemblance to pellagra as noted in a recent report (1908) to the State Board of Health of Georgia by Dr. H. F. Harris, of Atlanta, who thus adds to his honors already gained in researches upon pellagra and hookworm disease.

Course: Those cases which were admitted in the advanced stages of the disease succumbed on an average of about ten days, while in those with apparent recovery the symptoms were noted to persist for about thirty days.

In only one case was there complete disappearance of all mental symptoms, and this was in the case of a vigorous white woman; while in those with continuance of mental symptoms, senile changes were uniformly present.

Treatment: Potassium iodide in combination with bichloride of mercury has seemed to prove of benefit in controlling the number of stools in some cases, but would appear to intensify other symptoms.

Stick nitrate of silver has continued to prove the best weapon in our hands for combatting the obstinate stomatitis.

General tonic treatment is still used with slight material benefit. Alcohol in the form of whiskey and combined with beechwood creosote has proved of doubtful efficacy.

In extreme cases strychnine in large doses and combined with digitalin hypodermatically has proven of temporary benefit; and in one case of extreme shock following bath, nitroglycerin was added to a degree of tolerance with must happy results. This latter drug has been regularly continued since, and, in view of the rather prompt and rapid clearing of the skin lesions in this case, (not forgetting the fact that meta arsenic carbamide had been previously administered), it would appear that further investigations with this drug in its effect upon the skin are warranted.

Atoxyl has been used in doses of grains one-third each day, increased to one grain per diem, both hypodermatically and by mouth, but without any apparent benefit. Recently one of us (Dr. Greene) has used the drug in dosage of seven and a half grains hypodermatically, repeating the dose once only upon the second day, in four cases. One of these, which died two days after the first dose, was in advanced stages of tuberculosis. Of the other three all may be said to have been improved, the erythema fading and the number of stools being reduced in all but one. No toxic symptoms were noted in any, such as amblyopia, gastric pain, etc., but, on the other hand, actual relief from previous and persistent nausea has been observed. In the light of our experience with this drug, we are only willing to believe in the futility of small doses, and the uncertainty as yet of the curative effect of the heroic ones—notwithstanding the notable absence of any untoward effect from same.

We believe that frequent bathing and careful attention to diet should receive first consideration in all cases; and, as a routine, are accustomed to interdicting all articles of food derived from maize. It is interesting to note that in one case (negro male) after apparent

recovery an exacerbation of gastro-intestinal symptoms and ultimately death followed upon the surreptitious addition of corn bread to his diet. A stricely milk dietary is still considered the best form of food for our pellagrins; and to this is added toasted bread, with custards, hot eggs, and gradually additions of other food stuffs (excepting corn products) as the symptoms abate.

Prognosis: While the death rate in pellagra is notably large, as has been pointed out by almost all writers upon this subject, yet sight should not be lost of the fact that the vast majority of these cases have come from our asylums and alms houses, where conditions are most unfavorable and body resistance has been previously lowered by intercurrent disease and debility. Consideration of the cases which are seen by physicians in general practice, and a careful search into the histories of those cases received into our hospitals, brings to light much of interest in the length of time (even twenty years or more) over which this condition may endure, with frequent remissions and recovery and without seriously incapacitating the affected individual, even though unrecognized and without proper dietary treatment. It is to be hoped that in the future such danger and oversight may be avoided, and, by early recognition and treatment, reduced to a minimum.

Of the five cases embodied in a former report from this hospital, there was a complete subsidence of symptoms in one case (white man) which has continued to present time; and has been interrupted only once for a short period when a return to general diet, including daily ration of corn bread, was accompanied by slight erythema and slight gastro-intestinal symptoms.

Of the thirteen more recent cases, eleven of which were among women, there has been complete subsidence of symptoms in two, one of which has returned home and two more still under treatment show great improvement at the present time, with prospects reasonably good for recovery. All the cases that have succumbed have given undisputed evidence of serious complicating conditions, such as senile debility, tuberculosis and syphilis; and this applies with equal force to the new cases seen among the men, both of whom have died.

An analysis of these figures gives a ratio of recovery of about one in every four or five cases—which is not excessive when consideration is given to the complications and general character of the patients received. The one case (woman) which made the most rapid and satisfactory recovery (so that she returned home) was a

young patient from the middle walks of life, untainted by acquired or inherited disease.

We would feel it incumbent upon the medical fraternity to seek to allay, as far as possible, that unnecessary and widespread alarm which has developed so rapidly in the past year among the people of the South, irrespective of class or condition, regarding this disease. While its dangers and consequences are not to be lightly considered or underestimated, yet the prosperity and peace of mind of our entire people demand that we should look at the matter from a more subdued and conservative viewpoint. We cannot believe that pellagra is destined to overrun our beloved Southland, as it has encompassed the portions of Southern Europe for the past century or more; and the very fact of its early recognition among us presages a vigorous warfare against its encroachments, and, with the aid of a conservative public press and an enlightened and scientific medical body, profiting by the vast experience and brilliant researches of our European confreres, may quickly accomplish the steps necessary for its complete effacement and extermination. To this end it would seem to the essayists that Governmental aid and intervention should be sought, and the doctors, as guardians of the public health, while allaying the fears of the populace and teaching personal hygiene at home, should, as a great fraternity of no mean political importance, urge upon our representatives in Congress the great necessity of introducing and supporting such measures as will insure a fuller execution of our pure food and drug law and secure the inclusion of our corn products among those food stuffs receiving careful Governmental inspection and supervision.

CLINICAL OBSERVATION OF FOUR CASES OF PELLAGRA.

REA PARKER, M. D., PH. B.

First Assistant Physician Eastern State Hospital

WILLIAMSBURG, VA.

Since 1735 pellagra has baffled the skill of many of the most eminent scientists of the world. First occurring in Spain, then in Italy, where it followed shortly upon the introduction of maize into that country, and thence from first one country to another it has slowly but persistently spread its wings of terror until its shadow threatens practically every nation of the world. The combined efforts of the best thinkers of nearly every nation have accomplished but little, and the malady is now in practically the same chaotic state that surrounded it a hundred years ago. With the exception of the occurrence of a sporadic case in 1902, and one or two others of uncertain diagnosis, America was singularly free from the disease until 1906, when we suddenly found ourselves the victim of this dreadful malady which has proven a veritable scourge to portions of Europe and other countries.

It is earnestly hoped that we, ourselves, may be able to do something to check the progress of this disease or to stimulate others to a speedy solution of the problem.

In presenting a clinical report of a few of the cases of pellagra that have come under my personal observation, it is not with the idea of contributing anything new on the subject, but rather to give the profession at large a general idea of the disease as it exists in Virginia. The following report is fairly representative of the varied clinical manifestations of the malady as it is seen in that State.

In September, 1908, pellagra first appeared in Virginia in the Central State Hospital on the wards of the writer.

Through the courtesy of Dr. Drewry, Superintendent of the Central State Hospital, that case is given below in part as reported by him and as it appeared in a recent edition of the Virginia Medical Semi-monthly:

Augustus J. (6733)—Admitted first time Sept. 7th, 1908, sent to his home Jan. 13th, 1909, much improved, and returned to the hospital a short time ago, physical and mental symptoms herein described having returned in an intensified form.

He was born and raised in Southside, Virginia; age, 55; color, black; farm laborer; no education; habits as to alcohol and other dissipations good (?); general health fair, until about two or three years ago, when, according to best information obtainable, he began to have occasional diarrhœa, feel weak and good-for-nothing and noticed some "skin trouble" on his hands. These symptoms were worse in spring and summer, better during the cold months, when they almost disappeared. Have been unable to get family history.

Patient said—before he became demented—that he always had plenty to eat, lived in a fairly comfortable house, and worked steadily on the farm, that he got his meal from the neighborhood store, and from corn he raised and had ground at the country water mills.

From information given in the commitment papers, the patient was "sick" in the spring of 1908, and in the following September was much weaker, and mental symptoms had progressed; at times he was excited, at other times depressed, and had hallucinations of fear, thinking some one was trying to shoot him.

From examinations on re-admission and frequently since, his clinical history is here given. (We made a tentative diagnosis of pellagra last fall and kept the patient under careful observation. Dr. Rea Parker, then on our staff, first observed the symptoms, and suggested the diagnosis and so entered on the records.)

General Physical.—Pulse 80-100; respiration 12-20; temperature 96 3-5 to 99 F.; arteries atheromatous; blood pressure 133 m. m.; heart irregular; marked accentuated aortic second sound; lungs normal, except slight bronchial breathing over upper left infraclavicula region; general prolonged expiration; liver small; muscles thin and inelastic, producing apparent rigidity or stiffness of joints; progressive emaciation; weakness and loss of weight; general physical debility; anemia; lipoma, 2 1-2 by 3 inches, at right inferior lateral costal margin; has had gonorrhœa; brown discoloration of bulbar conjunctiva; and arcus senilis. There was observed in this and other cases a peculiar odor.

Gastro-Intestinal.—Obstinate and exhaustive diarrhœa, with occasional remission prolonged in fall and winter; stools thin, light yellow or dark brown; appetite varied, usually very poor, at times excessive thirst.

Mouth.—Unpleasant breath, stomatitis, salivation, ulcerated gums, tongue flabby, otherwise usually normal in appearance, except sometimes furred, a few small black spots on the dorsum near the end.

Skin.—Extending from glabella downward, involving the alæ

nasi, the malar region, outer canthi, the upper and lower lips, mental prominence, and on the sides of the neck there was a distinct dark pigmentation. The surface of the face covered was somewhat irregularly butterfly shaped. Over the lower portion of the forehead, the entire nose and partly over the face there was a hyperthrophy of the sebasceous glands, these being plugged with a sebasceous material resembling a typical case of pityiriasis pilaris. The cutaneous affection, the anomalies of pigmentation, and the saborrhœa observed in this and other cases do not correspond with skin conditions frequently seen in cases of insane adolescents. There was a rather "mummy" appearance of the parts affected, involving the thumbs and fingers, and extending from the nails to about two and one-half inches up on the wrists, and all the way around, the line of demarkation being distinct and symmetrical—glove shaped. The skin lesion on the hands and wrists may be described as varying from a dark pigmentation to a dry desquamation and exfoliation, part of the surface being cracked or parchment like, but there was no induration. The skin on the tops of the hands "cleared up" somewhat, in places, leaving a fairly smooth surface. The feet were involved to a limited extent—the external malleolli and superior surface of the dorsum showing more or less defined spots, of a dark discoloration and a slight scaly dessication. About the scrotum, groins and region around the rectum there was a moist pruitus. There were also external hemorrhoids.

Nervous and Mental.—Motor paresis marked; unsteady gait; difficulty and awkwardness—as if afraid to trust himself—in walking, sitting down or getting up; bent forward attitude; when walking legs far apart; pain in back and legs, particularly over mid-dorsal region, and on pressure over abdomen; occasional headache, varying in intensity but usually dull in character. There was slight itching over the surface covered by the cutaneous lesions; patella reflexes were slightly exaggerated, later abolished; insomnia at night, drowsiness in day. With some remission the mental symptoms were progressive and marked, consisting of general apathy, dullness, indifference, depression, retardation, confusion, defective memory, disturbance of attention, clouding of consciousness, incoherence, mutism, melancholia, mental deterioration, etc., and finally pronounced dementia. At no time did the patient laugh or smile. He died Friday afternoon, September 3. Dr. James C. Bardin, our pathologist, assisted by Dr. A. W. Freeman, of the State Health Department, and Dr. M. S. Brent, of our staff, made a complete

post-mortem examination within four hours after death. The brain and spinal cord were sent to Dr. Simon Flexner, of Rockefeller Institute, New York. Dr. Bardin's findings were as follows:

Chronic pericarditis; hypertrophy of left ventricle; very marked chronic fibrous myocarditis; cloudy swelling of heart muscles; chronic endocarditis. Chronic adhesive pleurisy, both lungs; atelectasis of lower lobe, left lung; old, healed calcareous tuberculosis foci both lungs; chronic passive congestion both lungs; chronic passive congestion abdominal viscera. Cloudy swelling of the liver. Atrophy of the stomach. Acute duodenitis. Chronic atrophic enteritis; acute enteritis with erosions. Chronic and subacute colitis. Chronic appendicitis. Arterio-sclerotic nephritis, both kidneys. Arterio-sclerosis and atheroma of all the arteries, extraordinarily marked in the anterior coronary.

Case 1 (History obtained from son and commitment papers.)

Family History—Negative.

Personal History.—J. K.; white, male; Virginian; age 55; farmer; education fair; married, with several children, all of whom are healthy. Patient has lived in the country all of his life; early habits regular; ate largely of corn. In the spring of 1902 his health began to fail; showed indisposition to work; suffered with headache and vague pains throughout the body; intermittent diarrhœa; slight reddening of back of hands, forehead and neck; insomnia developed, followed by train of mental symptoms—refusing to see his best friends, claimed he was lost and failing to recognize family; committed to S. W. S. H., Va., in 1903; diagnosis: melancholia; discharged in a few months as recovered; each succeeding spring brought a return of the symptoms, the physical predomination—diarrhœa and skin lesions with increased severity; moved to Prince George County, Eastern Virginia, in 1906; drank heavily a part of 1907. With the exception of a slight mental depression the symptoms cleared up during the winter. In the early spring of 1909 the trouble reappeared in a greatly exaggerated form. Admitted to the E. S. H., Va., July 26th, 1909. Upon admission examination showed the following:

Nervous and Mental Symptoms.—Speech slow but slurring; unsteady gait; Romberg symptom; Argyll-Robertson pupil; patella reflexes diminished; patient said he slept but little. There was marked depression with some irritability. He was apprehensive, through had but little insight into his condition and showed defective judgment.

Physical Symptoms.—Somewhat emaciated; musculature poor tone; epitrochlear glands enlarged; complained of pain in rectum and at times in breast, bones and joints.

Circulatory System.—Slight mitral lesion, pulse 95, small and irregular, arteries somewhat atheromatous.

Respiratory System.—Negative.

Alimentory System.—Tongue coated; breath offensive; appetite poor; stomach, liver and spleen showed nothing of interest; slight serous diarrhœa.

Skin.—The skin over forehead was very red and rough. The back of the neck and supra-scapular regions were red and indurated and to a certain extent extending from the junction of the first and second joint of the fingers to a point about one inch above the styloid process of the ulnar, showed dry furred scales, beneath which the skin was dark red. The dorsal surface of the forearms presented the appearance that the skin would show about two days after a superficial scald.

Course of the Disease.—For the first week his condition remained about the same as upon admission. The diarrhœa became worse and not amenable to treatment; salivation also began about this time; the tongue became red and slightly swollen. These conditions gradually grew worse and at the beginning of the third week after admission nausea and vomiting set in, at the same time the reddened tongue became denuded. The skin condition on the back of the neck and supra-scapular regions gradually cleared up during this and the preceding week and the skin over the chest and elbow began to show involvement. The perineal region became red and œdematous; conditions remained practically the same as third week with added loss of appetite, the patient refusing food but craved acids, which he refused when offered him. At the beginning of the 5th week the involvement of the skin over the breast, which began about the middle of the 4th week became more prominent and by the end of this week it involved the supra-clavicular regions having a triangular shape, the base of the triangle being formed by the clavicle with apex extending to the epigastric notch. While this was the area involved it did not show a well defined line of demarcation until the latter part of the week and the beginning of the 6th week. The condition on the forearm during this time had become dry and scaly. Physical decline and general weakness was more rapid during the last few days of this week. At the beginning of the 6th week the skin condition over chest showed a line of well marked

demarcation, the skin being thickened, pigmented and somewhat roughened. The diarrhœa condition remained unchanged. He became so weak that he was unable to raise himself in bed. Tongue remained red and denuded; about this time the patient began to show marked dementia, which became more continuously pronounced. The perineal condition improved, while the other skin conditions remained as last stated. At this time he was taking nourishment somewhat better. On the evening of the 38th day he had a mild convulsion, *pupils becoming irregular, dilating and contracting alternately without any external stimuli, one pupil reacting independently of the other;* during that afternoon the patient was at times excited; the next day he began showing hallucinations, both auditory and visual, also expressing some few delusions. Patient continued in a delirious condition through this and the next day, dying at 3:45 a. m. the following morning. Repeated urinary examinations showed acid reaction; sp. gravity varying from 1.017 to 1.020, indican, few epithelial cells and an occasional hyaline cast. Examination of feces showed no evidence of hookworm. Repeated examination of the blood showed the following:

Polynuclear neutrophiles 85; lymphocytes 11.5; large mononuclear 3.5; transitional 3.5; polynuclear eosinophiles 0; mast cells .3.

Case 1 (Dr. Mary Roche's case) History obtained from commitment papers.

Mrs. M. (1157), admitted to Eastern State Hospital September 21, 1909; white; female; age 33; married, with three children, youngest twenty months old. There was no history of insanity in the parents or near relatives of the patient; cause of insanity not given. Mental symptoms were first noticed in June, 1905, four years previous to admission, with marked depression and delusions fearing some one was going to kill her.

Upon admission the patient was very weak and emaciated and suffering with diarrhœa. Temperature subnormal; pulse 120-130, low tension. There was complete anorexia, nausea and vomiting.

Physical Examination.—Patient a small, emaciated woman, expression anxious, eyes sunken, pupils widely dilated, reacted sluggishly to light. Across the forehead beginning about three cm. below the hair line is an irregularly outlined pigmented area extending down on right cheek. On dorsal surface of hands, particularly over the articulations, wrist and feet the skin was slightly thickened and covered with dry, harsh, brownish scales. The little finger of one hand showed some contractions of flexors. There was slight

thickening of radial arteries; muscles atrophied and slightly rigid. Patient lying on her side with knees drawn up; pulse weak and small, 100-120; heart, second sound clear, first sound soft and blowing; lungs negative; tongue red and fissured; flow of saliva almost constant; teeth poor; breath fetid; appetite poor; liver and spleen not palpable; abdomen retracted. Patella reflexes irregular, increased on right side, diminished or absent on left.

Vaginal examination showed vulva very red and inflamed, vaginal mucosa very red; there was a deep lateral tear of cervix; uterus retroflexd and slightly enlarged.

Mental.—Patient was markedly depressed, almost constant mutism, occasionally answered a question intelligently, muttering sometimes. After admission the patient grew steadily weaker; temperature ranging from 97-100 F. Diarrhœa with very offensive odor was at first marked, but eventually controlled by an acid diarrhœa mixture containing morphine. Intestinal antiseptics were given, but with no material effect upon the odor of the stools. At first nausea, vomiting and salivation were marked, nothing being retained for the first few days but albumen, later peptonized milk and selected diet was given. Hiccough was very troublesome at times. When first admitted there was a purulent vaginal discharge which improved later under treatment by douches. No hookworm was found in this case.

From such history as could be obtained, together with the clinical manifestations of the case, I made a tentative diagnosis of pellagra—my diagnosis being later confirmed by one of my associates at the hospital who had seen and studied a number of cases. The patient died October 10, 1909. No post-mortem was obtained.

Case III—Geo. M. (508), admitted from Accomac County April 10th, 1887; white; age 46; single; occupation, farmer; habits not given; no history of heredity; suicidal tendency. Duration of insanity previous to admission four months; cause unknown.

Upon admission mental symptoms indicated dementia præcox. There was nothing of special interest in his history since admission, except a chronic ulcer over the left mastoid region, until last spring, at which time dementia became more marked. A search for hookworm was negative. During June and July the irregular diarrhœa became worse, appetite fickle and the skin on back of hands red and indurated. Over the forehead and portions of the face the skin was covered with dirty brownish looking scales about 1-8 inch in diameter. The removal of these scales with soap and antiseptic solutions

left that portion of the skin covered with dark liver-colored spots over which new scales soon formed. The sebaceous glands were hypertrophied and occasionally covered with a fine mealy looking substance. The reflexes were diminished; pupils sluggish; gums red and spongy while the flow of saliva became increased. This man rarely ever spoke or took any interest in anything around him. About the first of October he became much weaker, refusing both food and medicine. At this time a systolic murmur could be heard over the mitral area.

On October the 5th the abdomen became distended and three days later there was positive evidence of fluid in the abdominal cavity. On the morning of the 16th he was in a state of profound shock. I tapped him that afternoon, withdrawing a portion of the fluid, about 95 ounces, which showed nothing unusual under the microscope. He continued to grow weaker, dying at 11:45 p. m. Post-mortem not obtained.

In conclusion, I wish to enter a plea in behalf of those already affected and of those who are liable at any time to become the victims of this disease, that this conference, representing as it does every, or nearly every State in the Union, and many countries instruct each State committee to request the Legislature of their respective State to pass and rigidly enforce such laws relative to the method of cultivating, harvesting, maturing, preserving and milling Indian corn as seem practical and necessary.

I do not, gentlemen, wish to appear an alarmist, but why wait to appeal to the public to do what it can to protect us from this invading enemy until thousands and millions of useful lives are sacrificed and the influence of heredity defies the nation for hundreds of years?

PELLAGRA AT EAST MISSISSIPPI INSANE HOSPITAL

J. M. BUCHANAN, M. D.

Superintendent State Hospital

MERIDIAN, MISS.

At the request of Dr. Babcock I am presenting short histories of the cases of pellagra at the East Mississippi Insane Hospital, but they do not present any points of special interest, and I do not feel that I can add anything new to the question under consideration.

These cases do establish the fact that pellagra existed in our State for several years, unrecognized, for they give histories of two to ten years' duration. However, I do not think pellagra cases occurred in bunches prior to 1906, and the first known case in Mississippi was observed at the insane hospital in November, 1907, although it was not recognized as pellagra until after the appearance of Dr. Babcock's report, which was several months after the death of the patient.

I am sure this was the first case in the hospital, for with the small number of patients (500) I have been able to be familiar with every case for the last twenty years, and a retrospection fails to show a suspicious case.

In April, 1908, Dr. W. R. Card made a report of two cases before the Mississippi State Medical Association, but that report did not command a passing notice. Since then seven other cases have been received. No case has developed in the hospital.

I have no data as to the number of cases in Mississippi, but as many reports have been made from various sections, I feel safe in saying the number has passed the 100 mark.

I believe in the zeist theory, and tried to lay the cause to imported Western meal, but three of my patients came from interior counties, and their people state that all the meal used was from home-grown corn, ground at nearby mills.

I have known pellagrins to be made worse by eating good corn bread, and it seems that there may be something in the bread itself that causes the trouble, and it may not be altogether due to the supposed toxine.

Careful inquiry fails to find a local cause. All were country people—fairly well-to-do, living in open houses, and using good spring or well water. While they did not come from any particular section, all came from the hilly and sandy regions, and none were from the delta or prairies.

In some cases skin lesions were the first symptoms noted. In others, general bad health and diarrhœa were first noticed—the skin lesions appearing later. In one case there was a recrudescence of the symptoms for ten years, with mental symptoms appearing last, but this may have been a coincidence, as she has a brother who is insane.

Our treatment was practically the same for all, and consisted of tonics and special diet. Atoxyl was given by deep injections, beginning with one grain, and increasing up to four or six grains per day. For the bowel trouble beta napthol in combination with astringents seemed to give best results. Z. O. ointment was used as a dressing for the skin lesions.

Some showed marked nervous troubles, possibly spinal, but as no post-mortem could be had, we have nothing definite as to out lesions. Others presented only mental symptoms. The mental manifestations were of the manic depressive type, and were quite variable.

In three cases there was known hereditary taint.

Of the nine, one made a good recovery both mentally and physically. Two recovered from somatic troubles, with little improvement of mind. Three died, and three are still under observation with no improvement.

Case No. 1.—J. E. B., No. 2292. Male; age 44; farmer; was admitted from Clark County, Miss., Nov. 7th, 1907.

There was a marked erythematous eruption on his face, back of neck, hands and forearms—having the appearance of being sunburned. Patient was suffering from a persistent diarrhœa—was very anemic—quite thin and weak. Slept but little and had a poor appetite. No temperature.

The nervous manifestations were very marked—being quite restless and excitable, but did not complain of any pains. Reflexes exaggerated.

His mental symptoms were those of acute melancholia—said to be of four weeks' duration. He was immediately put to bed and given treatment for diarrhœa, and also put on tonic treatment. The skin lesions were treated with Z. O. ointment. The patient improved somewhat mentally, and the eruption on face and hands improved under treatment. Diarrhœa persisted.

Patient died from exhaustion in three weeks after admission. Case was not diagnosed as pellagra until after death—consequently no history of the early stages were obtained.

Case No. 2.—Mrs. C. J., No. 2311; age, 22; married three months; admitted from Wayne County, March 3rd, 1908.

This patient was seen in consultation about last of February on account of mental troubles. A diagnosis of pellagra was made, and on account of mental disturbance, it was advised that she be sent to the insane hospital.

First symptoms appeared on face and hands in small splotches three months prior to admission. The eruption soon spread over face, neck and breast, also affected the hands, elbows and shoulders. The skin lesions had been treated with crysophanic acid by family physician.

There was no diarrhœa and bowels were irregular. Appetite poor —refusing food—and was wasting rapidly.

Reflexes lively. Complained of pains in feet and legs on pressure —not able to walk—feet would draw and could not stand.

Tongue red—mouth sore with excessive salivation. Pupils dilated—slept but little.

Six months prior to admission had been frightened by some one trying to enter her room. Since that time had been nervous and hysterical. When first seen she had lucid intervals, but had characteristic hysterical mania, with periods of extreme excitement—crying and noisy.

On 10th high temperature developed, and she gradually grew worse and died from heart failure on next day.

Was fond of corn batter cakes, but did not eat more than other members of family.

Case No. 3.—Wm. C., No. 1984; admitted from Lamar County May 12th, 1909.

This man was a former patient of this hospital, having been treated for acute mania from March 3rd to June 6th, 1905, at which time he escaped. Mental condition was greatly improved. During the stay here, there was no eruption on skin nor other somatic disorder.

He remained at home and farmed until May 12th, 1909, when he was returned to the hospital.

When received second time there was extreme emaciation, offensive diarrhœa—erythematous exudative lesions on back of hands and forearms, legs and shins. The face was rough and scaly, and there was a V-shaped mark on the neck and breast where the shirt had been opened. On account of extreme weakness, patient was unable to walk.

There were no nervous manifestations. Reflexes normal. The mental symptoms were melancholia, stupor and apathy.

Was treated with deep injections of atoxyl and tonics. Beta naphthol in combination seemed to control the diarrhœa. Eruption yielded to Z. O. ointment.

Patient still under observation—pellagra entirely cured. Has gained in weight and is robust looking. Mind much improved.

Case No. 4.—Mrs. B. J., No. 2470; age 37; widow; admitted February 23rd, 1909, from Chickasaw County.

Family history not good. Several members reported to be very nervous. Patient was extremely thin. Diarrhœa—red tongue—offensive breath with no salivation. Marked mental depression, with occasional outbursts of excitement; destructive and untidy.

Duration of mental disturbance, one month. This appears to have been a case of "pellagra *sine* pellagra," as she presented all characteristic symptoms of pellagra, except the eruption.

About six months after the admission characteristic pellagrous erythema appeared on hands and arms confirming the diagnosis.

Unable to obtain any definite history as to mode of living, but is from well-to-do people. Patient still under observation, and has been given the usual treatment for pellagra, without any apparent benefit.

Case No. 5.—Mrs. A. J., No. 2511; admitted June 2nd, 1909, from Jasper County, Miss.; married; farmer's wife. Family history bad—grandmother and one first cousin insane. Duration of attack, three weeks; acute melancholia—extreme emaciation.

History by family physician very unsatisfactory. Patient's condition too grave to make an examination. Characteristic eruptions of pellagra on her hands—with bowel symptoms. Patient's condition grew rapidly worse, and died on the 8th.

Case No. 6.—Mrs. Sallie R., No. 2531; admitted June 2nd, 1909. from Itawamba County, Miss.

Age 43; widow; four children; former family history bad. Has one brother in hospital. Has been having eczema for ten years during the spring and summer while working in the field. Always got better in winter.

Health began to fail in early spring of 1909, but continued to work in field until May, when the eruption returned. Diarrhœa and mental symptoms developed at this time. Reported to have had a

spell of some kind of paralysis—could not walk or talk, and lasted about a week, and was then sent to hospital.

On admission her physical condition was fair, appetite good—stout and apparently well nourished. Had some diarrhœa, which had been worse. Tongue red—patches in mouth—increased flow of saliva—erythematus exudative lesions on back of hands and both breasts, scattering on abdomen and thighs—no pains. Reflexes normal—mental state that of melancholia—talks but little—has had two attacks of lethargy, with mutism, etc.

Patient still under observation with but little improvement, excepting skin lesions, which are about well.

Ate corn bread, meal made from home-grown corn, ground at nearby mill.

Case No. 7.—Mrs. A. M., No. 2534. Admitted from Lowndes County, July 30th, 1909.

Physical condition good—nothing abnormal—characteristic pellagrous eruption on face, hands and breasts, which was almost well on admission. Family history bad—uncle and aunt insane—patient has acute mania of four months' duration.

Was committed by jury on account of mental disturbance, and no history accompanied patient. Patient still under observation. Eruption entirely healed—no improvement in mind.

Case No. 8.—Mrs. Sallie H., No. 2537. Admitted from Union County, Miss., August 3rd, 1909; age, 53; married.

No history accompanying patient. She says there has been an eruption on hands for three years during the spring—diarrhœa was very bad at times.

Was convalescent on admittance. Hands and arms presented well-defined discoloration and the demarcation between affected and healthy skin was perfect and characteristic.

Had used corn bread made from home-grown corn.

Mental state, excitement, alternating with depression.

Discharged restored October 19th, 1909.

Case No. 9.—Mrs. Mollie L., No. 2571. Admitted from Clark County, October 14th, 1909. Age, 33; married; four children; farmer's wife; family history good; father died of tuberculosis.

Personal history states that in spring of 1907 she had severe diarrhœa lasting until July. In April, 1908, diarrhœa returned and there was a skin eruption. These symptoms continued until July, when she got better again. In March, 1909, practically the same

conditions as the previous year developed, but did not get better in July. On September 23rd she developed a temperature and soon became flighty. This was thought to be a simple delirium from fever—and severe headaches. After this she began to talk incoherently with only occasional lucid periods—not sleeping much. When first admitted there was extreme emaciation and weakness. Characteristic eruption on hands, face and neck. Profuse salivation —tongue red and mouth sore. Bowels constipated. No pains in head or legs. Reflexes exaggerated—pupils dilated.

Urine showed some albumen and casts.

Blood examination—Color index by Talquist scale 60 per cent.; red cells, 3842400. Slight Poykelocytosis, white cells, 12860. Thirty per cent. of which were large Lymphocytes.

Patient has failed to respond to any treatment, and is gradually becoming weaker. Mental symptoms more pronounced, and death does not appear to be far away.

REPORT OF A CASE OF "PELLAGRA UNIVERSALIS."

J. RODDEY MILLER, M. D.

ROCK HILL, S. C.

Patient: S. F. R., white, male, age 57 years. Native of this State, has lived all his life in the Piedmont region of South Carolina. His life has been spent upon a farm, where he did general farm work. Six years ago, when 51 years of age, moved into town and began work as a carpenter.

He is a man of limited means, but has always lived well or had good food and been comfortably clothed and sheltered. Has never been intemperate in mode of living or in drink.

Family History: Father and mother were natives of this State. Father died, aged 54, of apoplexy. Mother died of pneumonia, aged 67 years. Has four brothers—ages 54, 59, 65 and 70 years. Has three sisters—ages 69, 71 and 73 years. One sister died at 12 years of age of measles. He is the father of six children. All healthy.

Personal History: Patient has always been rather thin and of spare build. Weighed before illness 125 pounds—5 feet 8 inches in height. Had typhoid fever when 25 years of age and pneumonia at 30. Made complete recovery from both illnesses.

When about 20 years of age was "salivated" after taking calomel.

Present Illness: During summer of 1906 patient became much debilitated, weak—had loss of appetite, had no pain except occasional headache, no diarrhoea. He spent two weeks at a mineral springs, which he says helped him. The approach of cold weather gave him strength, so that he was able to continue his work during the winter months of 1906 and 1907. In June of 1907 became weak—no appetite—about the same as the previous summer. This continued until October.

The following summer, July, 1908, again became debilitated; could take only limited amount of exertion; had loss of appetite; no pain; was obliged to give up his work.

During October, 1908, diarrhœa of moderate intensity developed, which continued a greater portion of the winter. Patient had not consulted a physician until in December, 1908, when he began to have "indigestion and diarrhœa," as he expressed his condition at the time.

Patient came to my office for advice April 3d, 1909. Temperature normal, pulse 76—tongue coated slightly; no pain; complains of uncomfortable feeling about stomach; has "indigestion and diarrhœa," as he says. An examination fairly complete revealed nothing.

At the time of second visit patient no better; diarrhœa more troublesome; has from three to six movements during morning hours. Movements are loose and watery; very little pain. April 15, feet and legs swollen. Œdema is in feet and legs, half way up to knee joint; both feet and both legs have the œdema.

Frequent examinations of heart, lungs and kidneys revealed nothing. Urine was always found in good condition—no albumen, no sugar, slightly acid in reaction. Some days the quantity of urine less than others. This time swelling of feet continued only one week. On the 18th of April, just two or three days before swelling of feet disappeared, an erythema developed upon back of hands and over thumbs. The skin appeared as if slightly burned, swollen, painful on pressure. On second or third days afterwards a few vesicles came which contained small quantity of yellowish fluid. This erythema extends only to the wrist; same on both hands. This condition of hands continued only about ten or twelve days, when they had healed and dried; skin became rough, cuticle pealed in places. In short time skin on hands had become almost natural; remained little rough in places.

During the period when patient had the swelling in feet and the erythema on hands, he says he did not sleep well; was nervous. Knee jerked or tendon reflex Sometimes seemed exaggerated, at other times no more than natural.

In the month of May patient began to improve or get stronger, better appetite; slept well; no swelling of feet, no rash.

This condition of improvement continued for eight weeks—part of May, June and into July. Patient began to do light work and said he felt as well as he had in years.

July 25th to 30th patient began to feel unwell again—had headache every day; appetite failed; diarrhœa returned.

July 30th, examination of heart, lungs and kidneys revealed nothing abnormal.

Tongue red, gums swollen, saliva greatly increased, but no odor about the discharge. In a few days, on August 4, mouth gave a great deal of pain, tongue red, papillæ prominent, superficial ulceration on inner side of lips and under tongue.

About this time (second week in August) a rash began upon back of hands. This is the second time the erythema appears. The eruption began on back of hands and forearm—here the skin has dark, reddish appearance, at first smooth, on second or third day is puffed, with few scattered vesicles containing yellowish fluid, in a few days vesication disappears, leaving a dry, scaly surface of dark red appearance. The eruption first appeared upon hands and forearm, but by the end of first week had appeared upon arms, face, chest, head and back. From 10th of August to 25th of August more and more surface of the body developed the rash, until it covered the entire body. Patient's mind clear at all times. The only mental involvement was a far-away expression and general apathy, so that patient seemed dull or listless. Exposed surface of body no more affected than unexposed. There were a few spots, or patches, where the skin was not so red and the vesicles were less in number; this was the case upon the under surface of forearm, under the Axilla and upon the calf of the legs. The vesicles were not numerous in any one place, but scattered, nor did they become very prominent or large, but were rather small vesicles upon a broad base; vesicle would dry in two or three days and skin become scaly and harsh.

The vesication was not so marked a condition as the dark reddish appearance of the skin of the entire body, first smooth but in a few days became rough and dry.

The skin on arms, hands and face had become scaly, rough and thickened by the time the eruption appeared upon the lower limbs and abdomen.

At no time has patient had elevation of temperature over 99 degrees F. Several times during the eruption and ptyalism temperature was registered at 99 degrees F. Pulse generally 74 to 80 per minute.

Some weeks pulse ran 76 per minute, then for few days would be 80 per minute. From the appearance of the erythema the second time and the ptyalism, the patient was confined to bed most of the time; preferred to lie in bed because the feet were tender and pained when pressed to the floor. First September—Vesicles have all disappeared. Scaly, rough, red condition of skin continues. Patient gaining strength and appetite improving, but the exfoliation is marked.

September 10—Patient gaining in strength, good appetite, bowels act only once a day, skin dry, scaly, thickened, dark reddish, covered

with whitish scales, except where the dry scales have recently been rubbed away. After rubbing away of scales, surface of skin would in few days again be scaly.

September 30—Patient walking around the house, good appetite, constipated, sleeps well; dry, scaly, thickened condition of skin about the same as has been for two weeks.

Treatment: Tonic remedies, as iron, quinine and strychnine, etc., astringents, bismuth, etc., were given at various times during illness with little apparent effect. Diet most carefully regulated.

Atoxyl given for six weeks by mouth in one-third grain dose, four times a day. At first this seemed to be of benefit, but later of no service whatever.

Later began atoxyl, two-grain doses hyperdermically every four days and solution of arsenite of iron sixteen minims daily given hyperdermically—deep injections. This is the treatment (modified) of Dr. Wood, of Wilmington, N. C. This has been of very great help to this patient. He is stronger, more like himself; the eruption is fading. Within five days after the commencement of the injections of arsenite of iron a different expression on patient's face, more like himself in every way and began to improve in all respects.

Conclusions.

I. No history of maize diet for past six years. This food product is not to be classed as cause of illness of this patient.

II. Nothing in family or personal history or habits to point to a cause of the trouble.

III. The history indicates the trouble to have commenced two and a half years before consulting a physician for treatment.

IV. Atoxyl and arsenite of iron given hyperdermically were the only remedies used with any degree of benefit. These remedies have greatly relieved the unfavorable conditions, added to the comfort and strength of the patient. Whether they will prove a cure, time and continued use can only tell.

REPORT OF A SPORADIC CASE OF PELLAGRA

JOHN LUNNEY, M. D.

DARLINGTON, S. C.

On the 11th day of August last I was called to see a young white married woman, age 23, named Mrs. C. S., who resided at the mill village in close proximity to the town of Darlington. She was afflicted with what appeared to me a very mysterious disease, unlike anything I had ever seen before in a practice of over forty years.

Symptoms.—The symptoms on examination revealed a slightly accelerated pulse and a temperature not exceeding 101 degrees F. There was a squamous eruption on the forehead and face as far down as the anterior nares. There were multiform lesions on an inflamed base extending all around the neck, shoulders, arms, forearms and hands. The backs of the hands where the disease originally commenced were slightly swollen. The upper part of the chest as far down as the mammory glands was also involved in the trouble. The affected parts were covered with papules, fine vescicles, scales, scabs and crusts with a secretion of pus under some of the scabs. In front the neck had a glazed appearance. The tip and edges of the tongue and mouth were ulcerated and bled a little occasionally. The nasal passages were also ulcerated and inflamed. The disease evidently extended through the whole alimentary canal and other mucous passages. There was considerable salivation with expectoration of a bluish, foamy, watery discharge. During the latter part of her illness a towel had to be placed under the side of her head to absorb it. The eruption was so severe and painful that the patient had to sit in a rocking chair day and night until within a few days of her death, when, from sheer exhaustion, she had to lie down. There was complete anorexia, loose bowels, loss of flesh and a neurasthenic condition amounting to melancholia which persisted to the last. On the lower extremities there were small scaly spots, and also small purpuric spots extending down to the ankles.

Etiology.—The etiology of this disease is still a matter *sub judice.* It is supposed to be brought about by the ingestion of spoiled maize as an article of food. When corn products become decomposed by fungi, fermentation or otherwise, poisonous properties are developed which probably bring on ptomaine poisoning. There are doubtless other contributory factors in the causation of the disease, which, as yet, are not fully understood. Formerly the disease was

found in Italy, France, Spain, Roumania and other countries. It is now, however, becoming alarmingly frequent in this country. Dr. J. W. Babcock, physician and superintendent of the State Hospital for the Insane at Columbia, S. C., together with Dr. J. J. Watson, recently visited Italy for the purpose of studying pellagra, and to their intelligent investigations we are indebted for a clearer insight into the disease. The investigations of Dr. C. H. Lavinder, of the Public Health and Marine Hospital Service, have also thrown a good deal of additional light on this hitherto obscure malady. Cases of this disease have have been found in South Carolina, Georgia, Alabama, Florida, Tennessee, Texas, North Carolina, Arkansas, Illinois, and other States.

Pathology.—In most cases the degenerative changes which take place are caused by the deleterious influence of the toxins developed in the system during the progress of the disease, the nature of which, as yet, is not fully understood.

Diagnosis.—The diagnosis to one who had never seen a case of the kind before, is quite difficult. In this case (my first) I had to arrive at a diagnosis by exclusion; that is, I had to find out what the disease was by finding out what it was not. I compared the symptoms and lesions with other well known skin diseases and found that they did not coincide with anything that I had previously seen.

Treatment.—In this case the constitutional treatment consisted of liquid food with stimulants, quinine, iron, strychnine, arsenic, nasal douches, mouth washes and gargles of glyco-thymoline properly diluted. The first thing I did was to thoroughly cleanse the surface, (which had been covered with gunpowder), with castile soap and warm water followed by a thorough further cleaning and disinfection with liquid green soap, and afterwards powdering the raw surface with boric acid. The remedy that relieved the burning pain best was a preparation of equal parts of linseed oil and lime water with 40 drops of carbolic acid added to the pint. This was more soothing to the skin lesions and gave the patient more ease and comfort than any other remedy employed. The case terminated fatally from exhaustion on September 1, 1909; that is, 21 days after I first saw her, notwithstanding my best efforts to save her. All through the disease she was gloomy, sad and depressed and said she was going to die. This was the first case of this kind that ever occurred in this county that I am aware of. I deemed it of sufficient interest to report it to the Darlington County Medical Society at our last meeting on September 30, 1909.

REPORT OF EIGHT CASES OF PELLAGRA

THEODORE MADDOX, M. D.

UNION, S. C.

On receiving the preliminary program I saw that there were many interesting dissertations to be presented for your consideration, with the time rather limited, so I requested that I be permitted to withdraw, but Dr. Babcock insisted, so I shall direct your attention to a report on eight cases coming under my observation during the past few months.

Case 1. T. P. M., male, age, 60. Occupation, farmer.

I saw the patient for the first time on June 9, 1906.

History: Bilious attacks accompanied with diarrhœa during the early spring months for at least eight years, which had gradually grown worse, but this was the first time patient had been confined to his bed.

On examination I found the patient extremely bilious, very nervous, moderate temperature, severe headache, pains in extremities, diarrhœa, vertigo on rising, sore mouth, tongue very red, with complete loss of epithelium, nausea and vomiting.

During the subsequent twelve days the temperature gradually increased until it reached 103 F. during forenoon, and 104 F. in the afternoon; occasionally, however, during this time there occurred rapid fluctuations in temperature, which was thought at the time to indicate a malarial complication.

From June 20th to July 10th the patient's condition was considered very critical, all the symptoms having increased in severity until death seemed only a question of a few hours. At this time there was pronounced delirium, and during rational moments suicidal mania.

The stools resembled those of typhoid, the odor was extreme, and blood occurred very early.

While the diagnosis was typho-malaria, still I was not satisfied, for I felt that there was something more in the case.

The following spring the patient had an attack similar, but much less severe than the previous one.

During the past spring the patient had only the vertigo, darting pains in the head and mild gastro-intestinal disturbance.

Each spring since coming under my observation the patient has had the characteristic symmetrical pellagrous lesions on the hands.

These were, in 1906, supposed by the writer to have been due to the action of the actinic sun rays on a very much debilitated skin.

With the discovery of pellagra in South Carolina it was an easy matter to make a diagnosis in this case, as pellagra often resembles typhoid.

Treatment: Guaiacol Carb, quinine and arsenic were the principal drugs used in this case.

Case 2. Miss L. M., female, single, age, 25. Occupation, stenographer.

This was the most typical type of the spirilla pellagra I have ever seen.

I saw her first June 14, 1909. Chief complaint, diarrhœa and irregular menstruation.

History: About thirteen years previous she had suffered severely with diarrhœa during the early spring. Had had periodic attacks during each succeeding spring, which increased gradually in severity. On returning from a visit to the Isle of Palms during the summer of 1908, the characteristic pellagrous eruption occurred on hands, face and neck, with decided nervous symptoms, vertigo and gastro-intestinal disturbances. Recovery at this time was very tedious; she had more or less vertigo, nervousness and gastro-intestinal disturbance all through the winter, and early in the spring there was an exacerbation, about two weeks after which I was called.

Examination: Uncontrollable, occasionally slightly bloody diarrhœa, sore mouth, very red, clean tongue, with loss of epithelium; irregular menstruation occurring anywhere from two to four weeks, pronounced anemia, very nervous, vertigo, photophobia, marked debility, loss of memory, pronounced anorexia, languid, morose, despondent, discouraged, insomnia, reflexes extreme, especially vaginal.

She was given the usual remedies for gastro-intestinal disturbances, but recovery was tedious. In two weeks she felt well enough to return to her work. I saw her on the following day, as she was not doing quite so well. At this time I observed the symmetrical lesions on hands, which had occurred while walking from her residence to the office, a distance of four blocks. It occurred to me that these lesions were too severe for the distance traveled and time exposed, also their symmetry caused me to consider the case more carefully. At this time it occurred to me that she was a pellagrin, and her father and brother were so informed.

Inasmuch as I had never seen a pellagrin, I advised that Dr. Bab-

cock be called in consultation. This not being convenient at the time, patient was placed on Atoxyl and Blaud.

In July the uterine hemorrhage became so severe that she was given the usual acid drink, without any appreciable benefit, after which ergot was tried with the same negative result.

Early in August her condition became very alarming. On August 9th patient was anesthetized and curettage performed with the hope of checking hemorrhage. The os uteri was found soft, flabby and patent, and the endometrium was loaded with debris. Immediately following this slight operation improvement was very rapid and decided, and it appeared to the inexperienced that she would soon be restored to perfect health.

During September she suffered a relapse. Not only did the old symptoms recur with increased severity, but excruciating pains developed in the lumbar region and extremities. Spinal lesions were doubtless occurring at this time.

Suicidal mania, with a loss of confidence in attendants, was at this time occasionally manifested.

Forty-eight hours before death symptoms similating meningitis occurred. Muscular contractions of spine and extremities were very marked. Although narcosis was complete, opis-thotonus was extreme. Occasionally at short intervals I had the nurse to bring the patient to a semi-narcosis, during which time there occurred the most pitious ravings and horrible seizures.

According to current literature, this is the spirilla type of pellagra.

Case 3. Mrs. H., age, 50, married. Multipara.

Called to see patient for the first time May 25, 1909.

Principal Complaint: Indigestion with frequent attacks of diarrhœa.

History: For the past twelve or fourteen years had suffered with a chronic gastro-intestinal disturbance, with frequent exacerbations, especially during the spring and autumn months, which gradually have increased in severity, until now she is unable to perform any of her household duties.

Examination: Sore mouth, very red tongue, with prominent papillæ, and complete loss of epithelium, photophobia, vertigo, fleeting pains in head more or less all the time, but were increased in severity during the spring and autumn months; usually diarrhœa with an occasional bloody stool, occasionally contipated, during which time there was much flatus and discomfort, which caused an increase in severity of all the symptoms.

Emaciation, anemia and anorexia were very pronounced.

Treatment: Inasmuch as the gastro-intestinal disturbance was the most complained of, as well as the most prominent symptom, she was given test breakfast of Ewald-Boas, consisting of a breakfast roll and a glass and a half of water. An hour later an effort was made to evacuate stomach contents. As undigested particles would occlude lumen of tube, only ten C. C's. could be withdrawn.

Examination of Contents:

Digestion, practically nothing.

Consistency, bread and water.

Expressed contents, 10 C. C's.

Residual contents, not estimated.

Mucous, X.

Appearance, bread and water.

Free HCl., nothing.

Acidity of contents, 10.

Combined acidity, not estimated.

Acidity of washings, not estimated.

Organic acids, acetic.

Odor, yeast.

Relative quantity of solid and liquid portions, half and half.

Owing to small quantity, expressed ferments were not estimated at this time.

There being a large quantity of mucous present, and a complete failure of digestion, she was given antiseptics and the bitter tonics in order to increase digestion so that a larger quantity could be expressed later and a more thorough examination of the contents made.

Lesions occurring on the hands within a few days, a diagnosis of pellagra was made, the patient considered hopeless and a further examination unnecessary. She was placed on Fowler's solution for a short time, then Atoxyl and Blaud. These remedies were given for a period of twenty days, then an intermission of ten days.

The patient has neither lost or gained to any appreciable extent since coming under my observation.

Case 4. Mrs. N. K., age, 38; married; six children. Occupation, domestic.

Saw her for the first time October 10, 1909. Chief complaint, nervousness, indigestion, diarrhœa.

History: Has been in poor health for a long time, but noticed that she suffered more in the spring and autumn, and especially has she suffered during her past three parturient periods, which occurred in

1905, 1906, and 1908 respectively. She said that it would be utterly impossible for her to describe her sufferings during these periods, and that she prayed constantly that she should never again go through another. She has been growing gradually worse during the past four or five years.

Examination: Characteristic symmetrical pellagrous lesions on hands, arms, sides of neck and across lower portion of nose; vertigo, headache, indigestion, bloody diarrhœa; appetite poor, anemia, nervousness; occasionally constipation.

Treatment: Atoxyl and Blaud principal remedies used. Decided improvement.

Case 5. J. P., female; aged, 35; colored; single. Occupation, domestic.

Applied for treatment on April 16, 1909; wanted to know if she did not have the new disease.

History: Lesions first occurred on hands in 1908. She has been growing gradually worse since.

Examination: Characteristic symmetrical pellagrous lesions on hands and arms; periodic attacks of indigestion and diarrhœa, which is occasionally bloody; vertigo, fleeting pains in head, red tongue with prominent papillæ and partial loss of epithelium, anorexia and anemia.

Treatment: Blaud and Atoxyl.

Case 6. W. G., age, 34; male; married. Occupation, mill operative.

Applied for treatment September 26, 1909. Complaining with weakness and diarrhœa.

History: Health had been gradually failing for the past year or more, but diarrhœa had occurred only about two months previous. Had gone the usual rounds at the drug stores, and during the past month had been under the care of two physicians at different times without any appreciable benefit, except to get diarrhœa checked, during which period he was considerably worse. I would like to state that this has been so with all my cases. They would be very miserable until diarrhœa recurred.

Examination: Uncontrollable bloody diarrhœa, mild headaches, slight vertigo, chronic gastritis, sore mouth, red tongue slightly denuded; much debility, anemia, anorexia, emaciation and much drowsiness.

Treatment: Guaiacol Carb. et Atoxyl. Recovery very rapid.

Case 7. N. M., female; age, 20. Occupation, school teacher.

Applied for treatment September, 1909, complaining with a gastro-intestinal disturbance.

History: Had been well until some six or eight weeks previously. At this time she had a severe bilious attack, with the usual malaise; headache, vertigo, drowsiness and impairment of appetite. Periodic attacks of diarrhœa, with constipation during the interim, during which time all symptoms were increased in severity.

Examination: Mild pyrexia, diarrhœa, sore mouth, red tongue, indigestion with much flatus.

Diagnosis: Simple biliousness.

Treatment: Eliminative and antiseptic. Recovery was tedious and incomplete. Within a few days there was an exacerbation, at which time pellagra was suspected and patient given Guaiacol Carb. et Atoxyl. Recovery rapid and seemingly complete. Her rosy cheeks returned and she not only regained lost weight, but now weighs several pounds more than at any previous time during life.

Case 8. Mrs. W.; age, 32; married; prinupera. Occupation, housewife.

I saw her for the first time November 3, 1909, at 9 A. M.

History: Has suffered since her fourteenth year with rheumatism and dysmenorrhœa. About ten years ago ovaritis occurred. During a period extending from 1902-6 three os uteri dilitations were performed with negative result. In 1907 abdominal section was performed with removal one-third l. ovary. Improvement temporary. Indigestion during the past twelve years. Early in September, 1908, she had an attack similar to the present one, since which time there has occurred periodic attacks of mania. She has been under medical treatment more or less during the past eighteen years, and during the past two years continuously under treatment.

Examination: Sore mouth with scalded burning sensation extending from lips to stomach, tongue red, with prominent papillæ and loss of epithelium extending along its border; reflexes exaggerated; insomnia, photophobia, anorexia, pains in head and mild constipation; pellagrous lesions on hands.

Vaginal Examination: Extreme hyperesthesia, fiery redness, vesicles on vulva extending to rectum with tendency to pustulation. Examination rectum same as vagina.

In conclusion will state that an early diagnosis in pellagra stands in importance next to preventive medicine.

If the disease is curable at all it is prior to the occurrence of the lesions, for at this time the nervous system is irreparably damaged.

IS PELLAGRA COMMUNICABLE OR HEREDITARY?

H. H. GRIFFIN, M. D.

Assistant Physician, State Hospital for Insane

COLUMBIA, S. C.

Medical opinion in Italy seems to be unanimously against the theory that pellagra is contagious or infectious. As the question, however, is today one of the most frequent brought to the attention of the general practitioner, and asylum physicians, I thought it worth while to review this particular phase of the pellagra problem, and I do this more particularly because of the action of the health authorities of certain States that pellagra cases should be isolated.

To begin with, let me call to your attention the summary of the opinion generally entertained in Italy, about pellagra as given by Tanzi, who observes: "In short, pellagra is a disease of man and other vertebrates which in turn is derived from a disease of Indian corn. The disease of the animals is an intoxication, that of the maize is an infection, the parasites of maize is not infective in animals."

All of us who have studied available literature, and it may be one hundred or more cases, must admit that as yet we Americans are not in a position to dogmatize about pellagra. We constantly see cases which raise the question of its communicability whereas usually only one case develops in a family, yet instances have previously and during this Conference, been reported in which several members of the same family have developed pellagra. The members of a family are generally placed under exactly similar circumstances, those very ones which probably engender the disease. Furthermore, unquestionably, cases of pellagra develop in asylums in patients who have been long residents therein who have undoubtedly been associated with pellagrins, so it is pertinent and important not only to ask, but to determine the origin of this pellagrous syndrome. Unfortunately the very large proportion, if not all of the cases, have eaten products of Indian corn. So that unless we deny in toto the influence of maize as a probable cause of pellagra, we are forced to consider it as a possible factor.

A most admirable study of pellagra by Nicolas and Jambon of Lyons, France, concludes that pellagra attacks three classes of individuals, namely:

1. Those who eat corn. The malady here arising from the poor food.

2. The insane. The pellagrous syndrome in these cases being assigned to psychic depression. (As well as the food ration).

3. Those coming under neither of these groups—"sporadic" cases and "pseudo-pellagra," in which alcoholism is the most common contributing factor.

Sandwith calls pellagra non-contagious in his definition of the disease. He further says that he is not converted to the Italian belief that pellagra is hereditary, excepting in the way that alcoholism is now believed to be so. Calderini noticed in 184 familes comprising 1,319 members, inheriting predisposition, that 648 were diseased, 671 healthy, practically equally divided.

Says Nicolas and Jambon: "The hypothetic role of heredity admitted by Strambio and Calderini was proven false by Roussel and Bouchard, false so far as including the transmission of a germ from parents to their children. But if by heredity one means only a debility of constitution of pellagrous infants, their pathological predisposition, this hypothesis is tenable and is verified by observers in countries where pellagra is frequent. (Here we might draw the parrallelism between pellagra and tuberculosis).

"Bouchard admits a morbid tendency, a predisposition and further says this predisposition is of less importance without exposure to the sun and poverty without which it would be incapable of itself of producing pellagra."

Scheube (disease of warm countries) says heredity plays a part in the etiology of the disease, as the acquired predisposition of the nervous system to contract pellagra can be transmitted to posterity, Lombroso is even of the opinion that the disease itself is hereditary, especially from the grand-parents. (Here evidently atavism is a more important factor in producing pellagra than immediate heredity).

Further studies of etiology have appeared to show heredity predisposition.

The investigation of Probizer among school children shows the offspring of parents afflicted with pellagra to be quite generally of poor development, both physical and mental. They are usually anemic and subject to various nervous and digestive disorders as headache, dullness, listlessness, dyspepsia, gastralgia, enteralgia, nausea and vomiting.

The mental symptoms manifest themselves especially at puberty.

(R. A. C. Wollenberg, Assistant Surgeon U. S. P. H. & M. H. Service on pellagra in Italy.)

The disease is sometimes hereditary, the children of generations of pellagrins are frequently feeble in resistance and of lowered physical vitality and hence fall easy victims to the disease. (Marie).

From Babes and Sion I further quote: "Lombroso distinguishes two forms of hereditary pellagra: a somewhat more severe form and a very mild form. This is so far interesting as it deals with the so-called abortive forms, which Roussel will not recognize as pellagra In such cases Lombroso found bad formation of the skull, extraordinary brachia-cephalia or dolicho-cephalia, retreating forehead, bad setting of the external ears, asymetry of the face, anomalies of the genital organs, many of them showing, according to Lombroso, a true pellagra sine pellagra, in that they are merely single symptoms, as burning of the feet, pains in the back, leucorrhea, amenorrhea, vertigo, etc., while desquamation and delria are wanting.

"We can not, however, unconditionally approve of this view." We have not ourselves observed anything similar and it is difficult to refer such vague symptoms to a disease, which is characteristic in its entire symptom-complex. Only in such places where misery, heredity and nourishment with spoiled maize are shown to exist, can we assume that the injurious poisons cause some of the pellagrous symptoms, whereas we would by no means recognize pellagra cases with such single symptoms in places where pellagra is not found, and maize is not eaten.

"In the pellagra of small children exists probable always a congenital or hereditary element to which the poor nourishment and neglect make an essential contribution.

"It was indeed early recognized and especially emphasized by Lombroso that cretins and epileptics are predisposed by nature to pellagra."

Predisposing causes—Innate weakness as manifested in cretins and epileptics, and influence inherited from drunkards, pellagrins, syphilitics, and malarial ancestry.

These conditions involve the nervous system so that in the next and succeeding generations this vulnerability is even more marked.

Sambon says pellagra is not transmitted by means of lactation. He further says: Until quite recently, the majority of physicians believed in the hereditary transmission of pellagra. Contagion was not admitted but cases of conjugal pellagra have been reported.

Procopiu discusses at length both of my queries. He says:

"The non-contagion of pellagra is proven. The contrary opinion has been sustained by Saloman, Titius, Hameau and Casal. The peasantry who live in towns and come in intimate contact with an urban population have never exhibited the appearance of a single case of pellagra; furthermore the people who live in the country among pellagrins, but without using corn for nourishment never become pellagrous themselves. Boniva has tried to inoculate this disease with the blood and with the saliva, but without result. All these facts prove that pellagra is not contagious, as would be expected since it is an intoxication."

This non-contagion is an argument against the microbian theory of pellagra.

Heredity—The heredity of pellagra has been sustained by Odoardi, Calderini, Landouzy, Ballardini and a number of others.

"Pellagra is not hereditary although children of pellagrins develop it frequently. One almost never sees pellagrous nurslings. The children of pellagrins, if they do not eat corn, never become pellagrous, but the influence of pellagra makes itself felt from generation to generation becoming for the race a real cause for degeneracy.

"Boudin rightly says: 'Pellagrins transmit to their children an evident predisposition to this malady. This predisposition shows itself by their inferior physique which lessens their resistance to the toxin. If many members of a family are attacked, the fact is very natural, all being exposed to the same cause.' "

Sacchi says: "The offspring of pellagrins are recognized by their uncertain gait, by their yellowish eyes, by their jaundiced complexion, by their fissured lips, by their coarse hair, by their puny, dull and apathetic appearance.

"Lombroso specifies predisposition to microcephaly, the absence of hair, atrophy of the genital organs and the hypertrophy of the abdominal ganglia.

"We have met with microcephalic offspring of pellagrins, but we have not noticed the absence of hair, on the contrary, we have sometimes marked its abundance. We have seen idiots born of pellagrous parents, and also dwarfs and cretins.

"Ordinarily the children of pellagrins are anemic and apathetic. Boudin believes that if the father is pellagrous, the predisposition to pellagra is transmitted to the boys, and a pellagrous mother transmits this predisposition to the girls. He supports this affirmation by a table of cases of pellagra which he had met in Italy. This assertion has not since been confirmed."

The Italian school denies the communicability of pellagra, but admits that the descendants, especially the grandchildren, of pellagrins are particularly susceptible to the unknown poison. In the face of long years of observation by innumerable observers, such evidence is not lightly to be set aside. What American would presume after a few years study of a limited number of cases, to set up his opinion against Lombroso, who said that he had studied pellagra all his life. He certainly had studied it for forty years in Lombardy, and who shall undertake to estimate the number of cases he saw during this long period. While the French school does not admit the theory of communicability, they seem especially inclined in explaining their cases of "pseudo-pellagra" and "sporadic" pellagra to emphasize the influence of heredity and the role of alcoholism.

The German and Roumanian schools, if one may conclude from Babes and Sion, agree in the main with the Italians.

I draw briefly two conclusions, namely:

1. That pellagra is not communicable, basing this conclusion upon the authority of many great minds who have spent a lifetime in the study of this disease. This seems to be the universal conclusion of those who by virtue of a large experience are in a position to speak and, from the one hundred or more cases that have come under the observation of the staff of this institution.

2. That pellagra is hereditary—that is in the form of a predisposition or a morbid tendency, such a tendency as we now believe to be inherited in tuberculosis or even in insanity itself.

The hereditary pellagrin is especially vulnerable, not only to pellagra but as well to physical and mental degeneration, including insanity.

Granting this double hereditary weakness as proven to exist in the offspring, that is the children, and especially the grandchildren of pellagrins—and further admitting as proven the prevalence of pellagra in our country among the white and black races today and possibly existing for thirty or forty years past, are we not brought face to face with a tangible explanation of the reason for the prevalence of insanity today especially the increase of mental disease that has been steadily going on in the negro race since emancipation? If the pellagrous heredity from the past is a factor in producing insanity today, what have *you*, the medical members of this conference, the guardians of the public health in many commonwealths, to say is one of the most tangible means of preventing insanity for the future? There can be but one answer, "Establish

beyond doubt the real cause of pellagra and remove it, and save humanity from two of its greatest curses: pellagra and its congener, insanity."

It has been well said by Dr. Zeller: "When we know what pellagra is we shall be much nearer understanding what insanity is."

DISCUSSION ON THE PAPER OF DR. GRIFFIN

Dr. John Forrest, of Charleston, S. C.: If I may be allowed to express my opinion on this subject I should say that pellagra is neither contagious nor hereditary any more than any specific poison is, any more than ergotism is, and indeed the analogy between ergotism and pellagra is remarkable, although not a word has been said here with regard to the analogy between these two conditions. I have heard it said on the floor today that corn is not the cause of pellagra. I did not suppose anybody thought it was. Corn is not corn when it is poisoned, it is not corn when it is damaged, it is not corn when it has turned into fungus, neither is rye. Who ever heard of ergotism being charged to eating rye? You may eat as much rye as you please and never be poisoned, but if you eat ergot, which is a fungus that replaces the grain of rye, you will be poisoned, and as ergot is the cause of ergotism, so, a fungus replacing the grain of corn is, I believe, the cause of pellagra. Almost all fungi are poisonous, and if we want to get at the true cause of this disease we must study the corn itself that is damaged and that has taken on a fungus growth.

Dr. Watson (closing the discussion): In this institution (South Carolina State Hospital for the Insane) it has been observed that the nurses can make a diagnosis of pellagra before the skin eruption appears by the offensive odor and characteristic appearance of the stools.

PELLAGRA IN CHILDREN

M. B. YOUNG

ROCK HILL, S. C.

Dr. Lavinder has said that the task remains to the Southern physician to ascertain whether genuine pellagra exists in the South, and whether the remedies employed are properly applied.

My limited opportunities for studying the malady prevent my giving you any satisfactory conclusions on this point, much as I should like to throw some light on the question. My paper will be limited to the report of four cases in children.

PATIENT ELLEN BOON, born in Chesterfield County, where she lived until seven years old, then moved to Lancaster, where her people worked in a cotton mill for three years. Afterwards the family moved on a farm where they lived for two years. They then moved back to the mill and lived a year, going thence to a cotton mill at Camden and there remaining five months. From Camden they moved to Rock Hill.

Family History.

Father and mother both living. The patient has two brothers and three sisters living. There have been no deaths in the family. I found no history of consumption, cancer or rheumatism in family.

Personal History.

Patient has had mumps, whooping cough and measles. Her bowels have been loose most of her life; otherwise fairly healthy. I am unable to find out what quality of food the patient has had, but now she seems to have an abundant supply of fairly nutritious food.

Present Illness.

The patient noticed a red rash on the back of her hands in April while living in Camden and thought it sunburn. The rash disappeared in forty days, but in July this red surface began to crust and peel off, the patient's bowels became worse and she grew very weak and nervous. Her mouth was never sore. Her tongue was moist and red and the papillae were enlarged. Patient uses snuff.

Examination.

Patient's height is 5 feet, weight 55 pounds. She is very anemic and thin. Her skin was rough and peeled off on the back of hands, on arms, shoulders, forehead and back of neck, front of chest, thighs, knees and instep of foot. The patient's hair is falling out. The papillae of her tongue are enlarged and pigmented, and her knee jerk is very much exaggerated. The patient moves about very slowly and before treatment was begun she was unable to get out of bed. She was very nervous. She answered questions in a slow, indistinct tone. Pupils react to light, and there is nothing abnormal about the heart or lungs. No examination of stool or blood has been made.

JOHN MOORE, three years and five months of age, born and lived at the house where he died. The house is surrounded by a farm and there are only two houses within half a mile of the place. His sisters and mother work in a cotton mill.

Family History.

Father and mother still living. The father is worthless and deserted the family some time ago. I don't know how his health is now. The mother is very stout and healthy. The patient has three sisters living, and they are strong and healthy, with excellent complexions. He had no brothers.

Personal History.

The child was well grown to his age, looked perfectly healthy and was very ruddy. So far as I could see he had an abundance of nutritious food and good sleeping quarters. He was very fond of corn bread and buttermilk. Had an attack of whooping cough last winter, for which I treated him. Had a few attacks with bowels while teething. I treated the child in June for what I thought to be malaria, at which time I overlooked the condition of his hands. This is all the sickness the child ever had until the attack from which it died. Present illness commenced in May with a red rash on back of hand and dorsal surfaces of feet. The mother thought this was caused by a scouring soap which the child had been accustomed to bathe its feet and hands with. The redness soon faded and the child had no further symptoms, save occasionally loose bowels. On September 12th I was called to see the child, when I found a dark desquamating crust on the dorsal surfaces of feet and hands, and on nose and back of neck. The child was very nervous and demented.

It could hardly be kept in bed. September 13th I was unable to get to the child, as I was quite busy, but was sent for several times on account of the restless condition of the child. The knee jerk was very much exaggerated, mouth was very sore and the papillae on tongue were enlarged. On September 12th, the child's bowels were very loose with a good deal of mucus in the stools. On September 14th the stools had become bloody and increased in number and the child became maniacal. On September 15th the stools were almost entirely blood and the child was no longer able to get up from the bed. I took sick this day and was unable to see the child any more and turned it over to a young practitioner who watched it until it died on September 18th. I have been unable to get any definite information from this young man.

BERTHA AND GRADY NEAL, white, age 7 and 6 years respectively, were born in Rock Hill and lived here except about three years when they moved to Chester and from there to Union and then to Yorkville, then back to Rock Hill. They have always lived about cotton mills and have been pretty good livers for mill people.

Family History.

Mother and father both living. Father's health good; mother has pellagra also. They have one brother one year and four months old, who has had a sore mouth and loose bowels. They have had no deaths in the family. The family is not of a nervous temperament, and they keep an exceptionally clean house. There is no consumption, cancer or rheumatism in the family.

Personal History.

Bertha has had measles, whooping cough and mumps. She has had loose bowels most of her life and has a marked kyphosis. She is fairly well nourished and not extremely anæmic. Grady has had mumps and whooping cough and this last spring. He is rosy and extremely healthy looking. He had the dermititis last spring, which disappeared during the fall, but was never noticed by a physician.

Present Illness.

I treated these children and mother most of the spring and summer for severe diarrhoea and stomatitis without once thinking of pellagra. Nearly everybody else at the same village was suffering with same complaint. This ran on with severe exacerbations at times until August, the girl and boy both developed a severe dermititis on the

feet, hands and necks. Then my eyes were opened. They are much improved now but their bowels are still loose. Their mouths are still sore and the papillae are enlarged but not pigmented. The mother did not have dermititis, but there was a distinct bronzing of skin when exposed to sunlight. Bertha has been extremely nervous and showed some signs of dementia. Grady, the boy, never showed any nervous symptoms. Both suffered from severe gastritis with nausea and vomiting on appearance of the dermititis.

Examination.

Both have a rusty appearance of skin on hands and feet, also on back of neck, which comes off in large crusts. Bertha for awhile was scarcely able to get about and hardly gave an intelligent answer to questions. Bertha has increased knee jerk, the tongues of both are red and moist and papillae are very distinct.

A CASE OF LABOR IN A PELLAGRIN, WITH SUBSEQUENT HISTORY OF MOTHER AND CHILD

CROWN TORRENCE, M. D.

UNION, S. C.

As a part of the report in this case of labor in a pellagrin, it seems advisable to preface my remarks with a brief history of the mother previous to the accouchement. White, native of South Carolina, in good financial circumstances, was 42 years of age at the time of confinement and had borne nine children, the youngest being then five years of age. Of these nine children four were living and in splendid health, while five had died during the first two years of their lives, one from diphtheria, one from convulsions, and the other three from cholera infantum. She was married at 19 years of age; all of her confinements being normal, and the first occurring in her 20th year. The menstrual discharges had been regular, but with a tendency to be too free and exhausting. Two years before the birth of the child in this report she developed a serous diarrhœa which attended her through the summer despite treatment. No other symptoms were noticed until the following summer, when there was a return of the diarrhoea, some digestive disturbances, much lassitude and a decrease in body weight. No regular medical service was had except as some pronounced exacerbation of these symptoms would occur. In November of this year, 1907, she missed her regular menstruation, and on account of her weakened condition she felt relieved over this, not atrributing it to pregnancy, but to some beneficent oversight of nature. In December, though, she became satisfied that she was pregnant, and from then until her confinement, the 13th of July, 1908, her condition to her became an obsession. The first five months of her pregnancy during the winter months were attended by much nausea, digestive disturbances, insomnia and great mental apprehension. The buccal mucous membrane during this stage had a raw and inflamed appearance, while the tongue seemed denuded of epithelium. During the last four months the nausea and stomachic disturbances were greatly abated, and the patient was hungry, but, because of a return of the serous diarrhoea, was in much fear of all food, and could hardly be induced to take any nourishment. At times this diarrhoea seemed to almost

threaten the onset of premature labor; but at no time during the most severe spells of diarrhoea was any rise of temperature noted. The insomnia grew worse and the mental condition became pitiful; the daily cry was, "I would rather be dead, I know I am going to die." Opiates alone gave any temporary comfort, and because of an idiosyncrasy, the after-effects were worse than the original condition. Albuminurea, with swollen, bloated face, and dropsical swellings in the feet, legs, thighs and up to mid-waist, appeared at end of fifth month, and continued until delivery, despite treatment.

No erythema was noticed, although the patient complained of the skin over the feet and ankles being on fire. This was passed over as being due to the œdema. The patient was confined to her room during the entire time of this pregnancy, and this may have some bearing on the non-appearance of any erythema. She was delivered on the night of the 13th of July, 1908, of a full sized girl baby. The labor was much easier than was expected, the placenta coming away clean, and the womb contracting down hard, without any manipulation. When the placenta came away a teaspoonful of F. E. Ergot with 1.-40 grain strychnine was given by the mouth, simply as a routine. The trained nurse I had secured for the occasion felicited with me over the result, the outcome of which we had been anticipating in such great dread.

After the toilets of the mother and child had been made and I had finished drying off my instruments (which I fortunately had not needed), and had eaten a little luncheon in an adjoining room, and was ready to leave the house, in all about one and one-half hours after the placental membranes were delivered, I was hastily called by the nurse to the mother's bedside. The patient had just vomited the Ergot and she was ghastly pale, yawning and gasping for breath. Underneath the bed a little stream of blood was beginning to trickle to the floor. Fortunately a fountain syringe which we had neglected to take down was suspended by the head of the bed. Quickly emptying this of its half tepid water and filling with water from a kettle that was nearly boiling, and assisted by a colored servant, I inserted the nozzle up into the womb, holding it there with one hand and with the other hand kneading and squeezing the abdomen with all the strength of desperation. The nurse in the meantime had been ordered to secure Ergotole from my bag, and to use hypodermically. In all I think she used five hypodermics as fast as she could fill the syringe and inject. At first I could feel no uterus under the abdominal wall. Everything was relaxed and

flabby, as so much soft flesh. After a few minutes, which to us were interminable hours, I felt the contractions start; and in a quarter of an hour the womb was back again, hard and firm as a croquet ball. There was no return of the hemorrhage, the lochia being about natural and continuing about 14 days. We injected some normal salt solution per rectum, but as to how much was retained or the beneficial effect derived, I was never able to say. There was never any rise of temperature. The dropsical condition disappeared, as did the diarrhoea and digestive disturbances.

The patient had seemed anaemic before labor, but now she looked as if there could not be a drop of blood in her body. There was no lactation. Outside of anaemia, non-lactation and extreme exhaustion, the patient made a comparatively uneventful slow recovery. She was not able to walk around in the house until nearly three months after labor. She passed through the winter of 1908-'09 in fairly good health, so far as comfort was concerned, but with the advent of spring, 1909, she again developed the diarrhoea and all the whole train of nervous symptoms, insomnia, loss of appetite, vertigo, raw mouth and tongue, and she was forced to take to her bed. It was just at this time that a positive diagnoses of pellagra was made. The patient had gone out into her vegetable garden and sat in the sun watching the weeding of some of her plants; becoming faint she was assisted back to the house, and after resting had a lunch of warm corn muffin bread and butter, of which she was very fond and accustomed to eat regularly. In a short time she became very ill, exclaiming, "That muffin has made me sick, I never want to taste any again." Then looking at her hands she called attention to them, saying, "And here I have gotten my hands all sun-burned out in the garden today also." These incidents were related to me in the same afternoon when I was called in; and it was then that I first allowed myself to realize that it was a case of pellagra. This was the first appearance, to my knowledge or to her memory, of any erythema or sun-burn.

The diarrhœa, at first serous in character, became sanguinolent, and so continued for over a month, then became serous again. The use of large quantities of the bromides were necessary to get even three or four hours sleep in the twenty-four. The erythema was marked and uniform over the dorsal aspect of each hand, and from over the elbow down the ulnar surface of the fore-arm. Pustules appeared upon the face, and several good-sized boils came upon the hands and fingers. The feet were again on fire and required con-

stant hot salt baths, and even carbolized ointments were needed to give temporary respite.

The patient seemed to brood over the fact that she was going crazy, and would continually say to me that she must be losing her mind. She developed a morbid antipathy toward her child, not wishing to see it or have it where she could hear it, attributing to the child her lamentable condition.

She was placed on Donovan's solution about the last of May, 1909, and maintained a dosage of five drops three times a day until the middle of October before any puffiness appeared under the eyelids; as this puffiness apeared the dosage was decreased to 2 drops three times a day, until it disappeared, then increased to a three-drop dosage three times a day, which is now her only medicament. She was confined to her bed from May until the last of July, since which time she has gradually improved in every way. She has gained 10 pounds over her April weight, her color is fairly good, and it takes close observation to detect the scar cuff around each wrist. The erythema in this case was dry and scaly, and under advice from Dr. Babcock, who saw this case with me, three applications of tincture of iodine removed all the scales and left a soft smooth skin.

I wish to call attention to the fact that this patient menstruated slightly six weeks after the birth of her child, and has been regular each month since. The discharges are not free and are very black and last only two or three days. She now sleeps from five to six hours each night, and attends to much of her house work, but has trouble sewing on account of her eyes, which she says have failed very noticeably. She is still easily upset, and is extremely careful with her diet, which is chiefly eggs and sweet-milk. By-the-way, she says that a number of the breakfast foods and cereals will upset her bowels and make her feel almost like the muffin did.

Now a few words as to the child: It was brought up on the bottle, and, though for the first year it was simply a scrawny little assortment of bones covered with skin, it all at once took on a notion to live, and became fat and is now a very well nourished 16-months-old girl with four teeth and a lusty yell. When about 8 months old it had some 20 boils and furuncles to appear at different times and places about over the body. Since then, nothing has disturbed her growth, except the pitiful fact that she has spina bifida, and the lower extremities from the hips down are poorly developed.

DISEASES OF THE EYE IN PELLAGRA

A. B. CLARKE, M. D.

PLANTERSVILLE, S. C.

In diseases of the eye in pellagra I find no difference in the symptomatology, characteristic appearance nor complications, save only they are very prone to exhibit the same cycle, as it were, a recrudescence in early spring-time and as summer progresses make recovery. My observations and findings have primarily been upon the rice field negro, who has been inured to hardship, poorly educated and equally poorly nourished, seldom able to read, and when he has disturbances attributes it to local conditions. My patients have invariably given history of damp meal, grits in lumps in bags, which have been wet carrying home in cotton sacks, openly exposed to vermin and dew, many times sour and eating sour corn bread. My attention was first directed to this disease in 1906. The patients gave me history of rheumatic pains, generally malaise, sensation of heat esophagus and in the region of the kidneys, radiating to the hands and feet, noticeably the palms and soles. (One child had spastic paralysis.) All complain of vertigo, neuralgic pains in the posterior aspect of neck. The dorsum of their hands and feet they said were "sun-burnt," skin swollen tense, burn and itch, severe stomatitis petallar reflexes exaggerated; insomnia, anorexia and erythema with petchiæ occurring at times. Also small oval blisters, even bullæ, which, upon breaking down, leave indolent ulcers. The chief physical symptoms delusions, apathy, melancholia disorientation with symptoms of dementia præcox. In two of my cases between the fingers cracked, extending to metacarpal bones and exposing ligaments. The tongue and mouth red inflamed and sore, gums swollen at times and present appearance of salivation. Lips swell, and they appear almost scarlet, crack and become denuded. The tongue has presented three varieties to me, heavily coated with brown streaks in centre, notched around the edges and very red. Again, smooth and denuded of epithelium, and in another instance sleek with deep fissures. Have never seen any changes in the hair and nails of my patients. All give digestive disturbances and lack of appetite. As the disease progresses the mental disturbances increase from extreme dull apathetic conditions, slow in speech, listless, tired and ennui, melancholy and dreads of

persecution, even suicidal tendencies have been exhibited. Some of these patients have informed me these conditions have crept upon them insidiously, gradually becoming more severe each spring and summer. (Three of these patients have since died.) Many of them complain of pains in the eyes, one of diplopia, and others of various symptoms. I examined more carefully and present the following data: (1) Man, 42, negro. Had been working timber and living upon a diet of grits and corn meal mush, crackers and cheese. All of which he stated had become moulded and soured, as he had no place save a sack to keep it in. He was suffering from ectropion, both eyes with slight conjunctivitis. Had marked pellagra symptoms. Iris was dilated. Very little photophobia-cornæ aqueous, lens clear, vitreous, slightly hazy; choroid showing evidence of atrophy. Dark pigmented areas, the nerve head was oval, suggesting myopic conditions. Arteries pale, veins thick and heavy. The other eye was normal, consensual reaction from left to right, but not from right to left. I made several further attempts for examinations, but patient was in a cachectic condition and refused, dying in seven days after the last attempt to view fundas. (2) Child, 6½, boy, negro, epiphora both eyes, marked pellagra patient; talked incoherently all the time; had 3-5 degree fever (axilla). History positive, mouth, tongue and nares raw. Mother stated child's eyes became sore every spring, which suggested vernal catarrh. Examined for it, but was not present. Had marked conjunctivitis with small granulation, which resisted all treatment. Cornæ aqueous lens vitreous clear. Opthalmoscopic findings: Fundus dark black spots similar to retinitis pigmentosa, only limited to about four M. M. around nerve head and involving macula regions. There was no evidence of choroditis, nor were these dark areas in any other part of fundis. Mother stated child's eyes had been sore in springtime for three or four years, when his hands and feet became "sun-burnt." His vision had become worse this time. (From this child I recovered uncinarias) upon two occasions. Child's hands, feet, arms and legs and nape of neck in horrible condition, extremely emaciated. Died five days after my seeing him. They refused post-mortem, nor would they allow me to remove one of the eyes. (3) Man, 38, negro. History: Working in swamp making shingles, living on grits and condensed milk. He termed it "stir-mush." Would make enough at night to do next day. Sometimes it would sour in the pots, he said, but if boiled over it was fit to use. It had given him indigestion, he stated. Both hands, arms and legs presented distinct line of demar-

cation with round ulcers, some confluent irregular edges, and edema. Patient was painfully emaciated; complained of pains in the eyes, with burning sensation and failing vision. Upon examination I found fundus visible and appeared normal, optic nerve pale, almost white, beginning nerve atrophy. Patient was to return in fourteen days; was unable to do so, dying in nineteen days after last visit. (4) Girl, 17, negress. Anemic enlarged glands, cervical and epitrochlear for three years; hands and feet, arms and legs had become denuded. Was now raw with fetid ulcers on one leg almost to knee. Right eye cornea hazy with small ulcers, iris irregularly dilated posterior synechia, aqueous hazy, vitreous muddy, fundal lesion large hemorrhage to temporal side. Patient stated she was not aware that she was blind in that eye. Had floating bodies; was unable to get any detail of fundus. Left eye normal. In inner canthus of both eyes small papilla, which exuded watery substance upon being punctured. Patient came regularly for four weeks, becoming rapidly worse; excessive diarrhoæ and became almost unmanageable, refusing to eat. Died in eleven days after last visit. (5) Woman, 42, negress. Second year had sun-burnt hands with sore mouth, vertigo; had lost considerable flesh, had grown melancholy with hallucinations. *Iritis* in right eye three weeks; history of damp grits; bought them because they were cheap; damaged meal. Upon examination, hands and feet desquamating in layers, shelling off, leaving shiny tense beneath, and in places raw, bleeding surface. Lips and tongue denude. Suffered from insomnia and had no memory. Opthalmoscape gave no fundal lesions. Patient recovering. (6). Woman, 26, negress. First year mouth became sore and denuded, bright red in appearance, distinct erythema; complained of pains in the eyes, smarting and burning. Patient came every other day for two weeks, and each morning had a film of blueish tint, oval cornea of both eyes. At end of six weeks she stated that eyes gave her no further trouble. Physical condition improved. Mentally slowly making progress. Opthal moscopic shows nerve head normal, arteries very small, veins enlarged; one small black area between macula and nerve head of left eye. Right eye arteries very small, veins enlarged; small hemorrhage in vein to nasal size in physilogical cup. This patient gives positive history when and where she obtained the damaged meal.

In presenting these histories I have refrained from repetition as much as possible, also from citing pellagra symptoms. The class of persons I find who are suffering are they who lack nitrogenous food.

Many exist upon rice, grits and potatoes and a little cheap, poor grade pork, termed second or butts. They need meat, red meat, and a variety of vegetables. In four out of seven cases I examined, I found uncinaria, in all anemia, the treatment has been supportias and nutritious. Tonics increasing Tr. Cl. Fe. to 30 m., Tid. and Fowlers' Sal. to 24 m. Tid. Cod liver oil, eggs, bevinine and pepto manganse (Gudo's) acetate of lead and opium for diarrhœa. Phosphoric acid and fish diet, also oatmeal. Strychnine, tr. nux vomica; milk in abundance. I find local applications of practically no value.

These cases are from the extreme rural districts; hygienic conditions are very poor. Mentally, from educational standpoint nil, not one gave luetic history, all gave malaria. In one family, mother had chronic condition, son acute; both died. I have been informed that many negroes bought damaged meal which had been recovered from a flat, it having sunk. Some of the negroes became sick, had vertigo, abdominal pains and intense gastric disturbances.

From my observation I am of the opinion that pellagra is a trophic neurotic condition, involving the entire sympathetic system, not an autointoxication, but secondary to the ingestion of a fungi or ferment which causes katabolic metamorphosis.

EYE SMYPTOMS OF PELLAGRA

E. M. WHALEY, M. D.

COLUMBIA, S. C.

Gentlemen: The results reported were compiled from the examination of thirty-five pellagrins, and 50 per cent. of these were insane, thus we would expect to find the nervous elements much in evidence. The examination as to field and color it was impossible to get. It is rather disappointing that I cannot make out but one symptom that seems to be characteristic of this disease, and on account of the small number of cases, would not like to be too sanguine as to its being pathognomonic of pellagra. Still, Lombroso seemed to find it, though he did not state to what extent. This symptom appears as if the retinæ were thickened and gives the fundus reflex a peculiar indistinct yellowish color, and is not so pronounced as the senile reflex.

The skin lesion occurs on the eyelids, both upper and lower, more especially the lower; is well marked, and line of demarkation positive and definite, appearing like an echemosis of traumatic origin, but is not smooth on the surface. Two cases.

The dilitation of the pupil was not so prevalent as we expected to find from the reports of our Italian friends. This may be accounted for by the fact that observations in different localities make some variation in the symptoms, and the season has also been seen to affect the kind and virulence of the attack.

As stated, the number of cases examined was thirty-five, their ages varying from eight to seventy-six years, only two of whom showed normal eyes.

The appearance of the patients was that they did not carry their upper lids as high as they should, thereby giving the appearance of general lassitude. This dyskinesis of the upper lids in these subjects is not due to paralysis; it is voluntary and due to the fear of light. If unilateral and real, care must be taken with the examination to exclude paresis from other cause. Ptosis, due to ptomaine poisoning, fungi, lead, etc., must be eliminated.

Congenital ptosis will be confusing unless we bear in mind that it is permanent, usually bilateral, and often incomplete.

If you can make the pallagrin look up he will not correct the lid-

drop by throwing the head back, unless there be other cause present and an involuntary ptosis exist.

Frequently on raising the patient's head the lid or lids, as the case may be, will descend lower.

All pellagrins are unresponsive, and no field examination could be made. With few exceptions, the examination had to be made while the patient was in bed. The dilated pupil occurred bilaterally in three cases only; unilaterally in two cases, and one of the bilateral cases was myopic. Two cases resisted the action of homatropine for two hours, four resisted it less strenuously, while the others reacted in the usual twenty minutes. Hypersensitiveness to light with contracted pupils was the rule. (This contraction was not meant to indicate that they were pin-point pupils, but were smaller than normal.)

Shallow anterior chambers were found in 33 per cent. of the cases. Strabismus could not be detected when there was no other evident cause.

Where the gastro-intestinal symptoms were very pronounced and the inflammation extends to the mouth and post-nasal space, we find an obstruction of the lachrymal duct due to continuity of surface. This was noted in five cases, all of which had the mucous membranes very much affected by the disease. Photophobia of slight degree, without the inflammatory changes which usually accompany this condition, was present in six cases.

The findings, as tabulated, are as follows:

Lids—Paretic, 1; lachrymation, 2; dachryocistitis, 2; conjunctivitis, 2; muddy conjunctivæ, 2; jaundiced conjunctivæ, 3; obstruction of lachrymal duct, 5.

Corneal Abnormalities—Ulcer, 4; superficial inflammation, 2; increased sensibility, 2; subnormal sensibility, 7.

Muscles—Paresis (Rt. rectus), 1; mystagmus, 1.

Anterior Chamber—Shallow, 12; deep, 1.

Iris—Iritis serous, 1; sluggish reaction to light, 6; hypersensitive, 4; photophobia, 6; reaction to homatropine, slow, 4; prompt, 1; spastic reaction to light, 2.

Pupils—Unilaterally dilated, 2; bilaterally contracted, 3; bilaterally dilated, 3; Argyle Robertson, 1.

Tension—Plus bilateral, 1; plus unilateral (O. S.), 2.

Fundus—Retinitis, 2; detached retina, 1; optic atrophy, 3; optic neuritis, 3.

Lens—Cataract bilateral, 3; unilateral, 2; cloudy lens, 1.

Arteriosclerosis, 15.

After examining somewhat over half of these cases I found that there was most common a dilation of the retinal veins and a somewhat yellowish reflex from the retina that I do not remember seeing elsewhere. This appearance is hard to describe, appearing as a thickening of the retina itself.

The arteriosclerosis which appears in so many of the cases is of every stage and occurs in the young as well as the older.

Five additional cases examined gave the following:

No. 1. L. B.; bilateral ptyrigiums, pupils quick and contracted, arcus senilis; no inequality. Rt., reacted less promptly to homatropine. Optic neuritis; veins as usual.

No. 2. L. M.; corneal sensation subnormal; anterior chamber shallow; retinitis O. S.

No. 3. Dr. P.; corneal ulcer left eye.

No. 4. R. A.; sensitive to light, muddy conjunctivæ O. D.; iris slow to homatropine.

Opaque nerve-fibres, O. S., temporal side.

Veins; atony.

No. 5. D. S., pupils sluggish; photophobia; sensory reflex absent. Chorio-retinitis; arteriosclerosis.

Veins; antony.

The following report is extracted from "Pellagra," by Prof. A. Marie, of Paris. Prefaced by Prof. Lombroso. Authorized translation from the French by C. H. Lavinder, M. D., U. S. P. H. and M. H. service, and J. W. Babcock, M. D., Physician and Superintendent State Hospital for the Insane, Columbia, S. C.

Eye Symptoms—Remarkable peculiarities are found in the eyes of the pellagrous; a falling of the supercilliary fold is very frequent (28 times). In many cases is found also a marked unilateral injection of the conjunctivæ. These are observations which remind one of general paresis, and show, along with other manifestations, how frequently the lesions of the nervous system may be unilateral, especially lesions of the sympathetic system.

Very often also (74 cases) mydriasis of the two sides is found. Miosis is more rare, and when found is more usual in the aged. Cases of blepharitis are not rare, as was shown by the Piedmont commission. Often also diplopia, photophobia and synchysis are found. Many pellagrins remain for years with their eyes closed for fear of the light. Early cataracts are found among the pellagrous;

and pterygium is not infrequent. Dr. Ottolenghi, with Professor Manfredi and Dr. Flarer, have made ophthalmic studies on pellagrins. Their results are given in the following table:

Number examined	36
Depth of eye normal	12
Changes in the retina	15
Atrophy of arteries	12
Anomalies in fundus of left eye	1
Anomalies in fundus of right eye	6
Atrophy of optic nerve	3
Increase of pigment	3
Dilatation of the veins	1

Fifteen of these cases showed retinal changes by a yellow or gray reflex in one or both eyes—a sign of precocious senility; it is of interest to note that there were three cases of white atrophy of the papillæ, among which was one case of retine-choroiditis in an advanced stage. Ottolenghi found also in three pellagrins one light case of papillitis, more pronounced in the left eye; in the second case pronounced gray atrophy and diffuse retine-choroiditis of the two sides; the third was normal. It is of interest to note the observation that in several individuals the ocular fundus differed on the two sides. This, however, cannot be given as a reason for the numerous pupilary inequalities, since these are noted in individuals who show a normal fundus. However, the unilateral anomalies of the fundus, as well as those of the pupils, predominate in the right eye and consist in lesions of the arterial vessels with papillary and retinal changes. Rampoldi observed pellagrous ocular troubles principally in the autumn or the spring, and found that they consisted of organic lesions rather than functional disorders. The retina and optic nerve show more than any other part of the eye the pellagrous cachexia, next come the cornea and lens; finally the choroid and vitreous body. Hemperalopia and pigmentary retinitis are not rare. Torpid ulcers of the cornea are found with essential hypotonus of the bulb and scintillating synchosis of the vitreous."

The numerous pupil inequalities in the above report are probably due to trophic disturbances, and this will also account for the slow reaction of the iris to drugs that are dependent upon the local nerve endings. We must also take into consideration that this report does not mention the number of cases examined when seventy-four dila-

tations were reported, and consequently we cannot formulate any percentage as to these findings. It seems that the thirty-nine cases examined by opthalmologists were only considered from the anomalies and abnormalities in the interior of the eye. The pupilary reaction and external eye diseases were not sufficiently prominent or intentionally not reported.

Two additional cases reported to me by Dr. E. N. Carpenter, one with atrophy and one complaining of a continuous green light before the eye. E. M. WHALEY.

We are looking for light, and I hope this will stimulate research and assist in further investigation.

COMMENTS ON TWENTY-ONE CASES OF PELLAGRA

WILLIAM F. DREWRY, M. D.

Superintendent Central State Hospital

PETERSBURG, VA.

The first case diagnosed as pellagra in this institution, and I believe the first in Virginia, was in September, 1908. Dr. Rea Parker, then one of our assistants, first observed it. Since then twenty other cases have been observed and studied here. In addition to these about a dozen cases have been seen and reported by physicians in various sections of the State.

Of the cases occurring at this hospital all were negroes—16 black or brown and 5 mulattoes. Age of youngest at time of onset of disease, or rather full development, was 20, oldest 60; average 35. Nine were under 30, only 3 over 40. Ten were residents of towns or cities; 11 lived in the country. Their homes were not confined to any particularly geographical section of the State. With three or four exceptions all were poor and illiterate. Some had fairly comfortable homes and lived regular, industrious lives. Three or four and probably others, were dissipated, or habitually immoral, and in as many more there were indications of syphilis and in a majority there were symptoms indicating tuberculosis. One has had epilepsy for years. In one of those of the better-to-do class corn was seldom if ever used as a part of the diet. In all the rest corn doubtless constituted an important item of food. In those living in towns or cities the meal was doubtless a Western product, and the same thing probably applies, in a large measure, to those from the country districts. In this connection I may be pardoned for saying that the diet at the hospital is wholesome, nutritious and ample, and while much of the corn meal used is a Western product it has seemed to be of excellent quality.

Eleven of the cases have been received into the hospital within the past fourteen months and the symptoms existed at the time of admission or developed soon afterwards. The others (10) had been residents of the hospital from a year and a half to ten years, and in nearly all these there had been a history of recurrent diarrhœa and progressive mental deterioration, but as far as the histories show, no cutaneous manifestations were observed. There has been notable uniformity in the chief physical, cutaneous nervous and mental

symptoms. Muscular weakness, emaciation, anemia, more or less incoordination, uncertain gait, tremor, slow and slurring or thick speech, usually mutism in the advanced stages, headache, back-ache, anorexia, insomnia, have marked, to a greater or less degree, every case. The sensory abnormalities and the disturbance of eye and patella reflexes have varied. No convulsions except in one case. No paralysis. There has been variation in the several cases as to the pulse, temperature and respiration. In most of the cases the gastro-intestinal symptoms have been practically similar, there being remissions in practically all. The symmetrical skin lesions, too, have been about alike in all cases, simply varying in degree or extent. In some the lesions did not exist on the feet, neck, face or elbows, but in every instance the hands were involved. In only one or two cases was the face exempt. Hypertrophy of the sebaceous glands over the nose was present in every case.

The mental symptoms have run a remarkably uniform course, not conforming, however, to any recognized classification of psychosis. The symptom-complex approaches perhaps a toxic-exhaustion psychosis, of depressed type, terminating in more or less dementia. In all our cases there were mental apathy, despondency, emotional irritability, stupidity, disorientation, defect or loss of memory, confusion, disturbance of attention, clouding of consciousness, usually insomnia, etc. In some there was occasionally motor restlessness and mental excitement, and in some hallucinations and delirium of a terrifying or depressed nature. Depression, confusion and finally aementia or dementia have been notable in every case. In all the cases the mental and nervous deterioration was more rapid or pronounced after the gastro-intestinal and cutaneous symptoms set in; and on the other hand, when these improved the mental condition usually improved.

As to the termination of the cases: seven died during the full development of the triad of symptoms, in a state of physical collapse and mental disintegration. In three the symptoms have improved, in four the gastro-intestinal and skin symptoms have entirely disappeared, and in the rest these symptoms are improving. There has been no very material change in the mental condition of any of the patients, save one or two.*

Dr. J. C. Bardin, pathologist and bacteriologist at the hospital, who is carefully studying this phase of the cases, that is, pathologi-

*By January 1, 1910, there had been a total of nine deaths, and in all the other cases all cutaneous and gastro-intestinal symptoms had disappeared.

cal, reports that the blood showed a slight anaemia in every case; in some cases it appeared markedly concentrated and poor in fluid, doubtless due to the depleting diarrhœa. The leucocyte counts were generally about normal; some were slightly below normal. Differential counts have been repeatedly made, and in practically every case there has been a change in the lymplocyte and eosinophiles content, presenting certain features apparently constant in this affection. Dr. Bardin is not yet ready to announce the full results of his observations, as they have not yet been fully worked out. Mophologically the red blood cells show some variation from the normal size, tending to be larger than usual and in some cases poikilocytosis and polychromatophilia were observed. There were no abnormal leucocytes.

The blood pressure in every case but one has been below normal. In one case it averaged about 180 m.m. from day to day, while the other cases averaged about 112 m.m.

The urine presents nothing abnormal, save a diminution in chlorides in every case.

Post-mortem examinations in four cases showed the following general features: In three cases there was intestinal tuberculosis; in two, marked pulmonary tuberculosis; in one, old healed foci in the lungs and no tubercular lesions in the gut; and in one there was no lung involvement, though there were eight annular intestinal ulcers. The presence of some form of tuberculosis in every case that came to post-mortem is interesting; it probably is a terminal infection.

The heart in every case was atrophic and fibrous. Myocarditis was a marked feature. Microscopically, the atrophic muscles were in every case pigmented, the pigments being situated about the nuclei—the so-called "brown-atrophy" of the heart. The liver in each case showed an irregular capillary congestion, atrophy and cloudy swelling of the parenchyma and pigmentation. Fatty degeneration has not been observed. The spleen has shown nothing save marked congestion.

The intestines showed in each case a marked atrophy. The muscles and mucosa showed great thinning and in places the entire mucous membrane had disappeared. An infiltration of the submucosa with eosinophiles was noted in two cases. There was in every case but one a pigmentation (golden brown) of the muscular coat. The kidneys have shown irregular congestion and slight increase in connective tissue, chiefly marked in the capsules of Bowman and in the

medulla. Arterio-sclerosis has been marked and the left coronary artery has shown this condition to an extraordinary degree in every case. The brains and cords of these cases have been sent to Dr. Simon Flexner, Rockefeller Institute, for examination. His report has not yet been received.

The general line of treatment followed has consisted in special attention to the dietary, daily baths, personal hygiene, modified rest and out-door life, attention to the gastro-intestinal disturbance, together with eliminants, tonics, arsenic, &c.

Presented at the Conference on Pellagra held at Columbia, S. C., November 3rd and 4th, 1909.

PELLAGRA, THE CORN CURSE

J. S. DE JARNET

Superintendent Western State Hospital

STAUNTON, VA.

I offer no excuse for writing this article, even though the medical and lay presses are publishing literature on this subject daily. I believe unless the most drastic steps are promptly taken to stop the consumption of diseased Indian corn in our Virginia corn-eating State, we will suffer the most terrible curse that has ever befallen our fair land. The demon, I believe, lurks in a green fungus, and there lies our danger.

The majority of lexicographers give pellagra, as derived from two words, *pellis,* meaning skin, and *agra,* a seizure; others give *pelle,* skin, and *agra,* rough.

The synonyms of pellagra are: Italian leprosy, Lombardy leprosy, Alpine scurvy, Asturian rose, Maladie de las Teste, Mal de la Rosa, Mal de Sole, Vernal Insulation, Hydro-mania, and Mal de Miserere, etc.

DEFINITION.

Pellagra is an endemic skin and spinal disease of Southern Europe. Now found in Virginia and fifteen other States of the Union. It is said to be caused by eating diseased or damaged maize, also dependent upon bad hygienic conditions and exposure to the sun. It is marked by increasing erythema of the exposed skin—worse in spring, fading away in winter.

Exfoliation of the skin, weakness, spinal pain, digestive disturbance, brain and cord lesions, mental depression, great emaciation in last stage, marked tendency to insanity, immobile expression, and usually little or no fever.

ETIOLOGY.

The eating of diseased Indian corn is almost universally believed to be the cause. This theory is ably maintained by Lombroso and Balardini on account of the greenish color produced by the fungus. This was called the verdet theory. Belmondo believed pellagra to be due to a specific organism. The disease is never found except among a corn-eating population, and, in Italy and France, where

pellagra is prevalent, it increases in proportion to the amount of musty corn products consumed.

The fungus may develop before the harvest if a wet season, especially if the corn is late and does not have time to ripen. It may attack the grain at any stage, in the field, in the corn crib, at the mill, before and after grinding, and even after cooking, if left for any length of time. The fungus will grow on the bread, or breakfast food, etc.

In Southern France and Northern Italy, where a week's supply of the corn meal gruel or polenta is all cooked at one time, the fungus grows rapidly. These countries have heretofore been the most pellagrous in the world. The disease is not contagious in any sense.

HISTORY.

Pellagra has been known since about 1750, and has been, and is, endemic in Southern France and Northern Italy. From 1830 to the present time it has been thoroughly studied by Strambio, Brierre de Boismont, Baillarger, Billod, Sacchi, Gintrac, Lombroso and others. Mental derangement frequently accompanies the disease. According to Billod, three-fifths of the insane in the asylum at Astino were pellagrins; in Senarra one-third, and the same proportion in Servolo at Venice. Assistant Surgeon Lavinder, U. S. M. H. S., states in his able article on pellagra, in 1907 there were 100,000 pellagrins in Italy, and upwards of 50,000 in Roumania.

In pellagrous countries about 10 per cent. of pellagrins are in hospitals for the insane. Assuming this to be true in Virginia, we have thirteen reported by Dr. W. F. Drewry, of Petersburg, Superintendent of the Central State Hospital for the Insane; four in the Western State Hospital at Staunton, Va. Total, 17. Therefore we would be justifiable in believing we have about 170 pellagrins now in Virginia.

SYMPTOMS.

The symptoms of pellagra are too numerous to mention. The most prominent are: recurring erythema in the spring on parts exposed to the sun, subsiding during the winter; ravenous appetite, sometimes horror of food; diarrhœa, usually persistent; great psychomotor retardation, vertigo feeling of heat and pain along the spine, burning of skin, slight losses of consciousness, extreme apathy, indisposition to do any kind of work or take exercise, intense debility, numbness of lower limbs, staggering gait, upper extremities ataxic,

lower spastic, hallucinations of sight and hearing, mutism, immovable attitude—according to Sandwith—the "Smile forgotten face;" slowness of speech, incoherence of ideas, sad delirium and a fixed idea of despair, stuperose melancholia, delusions and suicide by drowning. The hydromania is so fearful that one cannot conceive of its intensity. Strambio says: "The intense heat of the skin excites not only directly to immersion in water, but also gives rise to delusions of fire, both in this world and in the next, and the miserable victims plunge into the water to extinguish at the same time the real and the imaginary fire."

Gintrac states in traveling through pellagrous districts he was informed that every year many insane pellagrins are found drowned in ponds. Miss Marion Hamilton Carter, in a carefully written article, November number of McClure's Magazine, most graphically describes the water-mania. She says: "It is largely through the burning sensations that water comes to exercise a peculiar and often fatal fascination for pellagrins. They love it—love its feel on their bodies, its flow, its shining surface; hang over it by day and dream of it by night." But with the fascination of delight runs the fascination of terror, even in the same patients. The water seems to call them, to hold them in a spell. They cannot withdraw their gaze from it, but look into its depths until nausea and vertigo come on and they fall into it. In this state, and stunned by the shock, they drown. Some, on coming to a water course, close their eyes and cling to the nearest tree until the vertigo passes, then shamble away in fright, only to be seized with the charm of the next stream and throw themselves bodily into it. Through all the disease runs the black thread of misery."

DIAGNOSIS.

The multitudinous symptoms mentioned above are very confusing, and I think the following are usually sufficient to make a diagnosis:

1. The symmetrical erythematous eruption on the back of the bands and exposed parts to the actinic rays of the sun. The eruption is first very red and the skin is exquisitely sensitive to the sun's rays, becoming blistered from the slightest exposure. Attendants in hospitals for the insane have been discharged when their patients were found with these blisters, the superintendents believing the vesicles to be the result of scald while bathing. After the acute stage wears off the eruption becomes scaly and the skin rough and dirty looking, with whitish lines running along its natural folds.

2. The diarrhœa is more or less constant, and has a peculiar odor, probably caused from an inflammatory condition of the entire alimentary tract.

3. Anæmia, immobile expression, shiny, bright eyes, and indisposition to take exercise.

4. The victims are usually among the very poorest class, who live largely on corn products. Since poverty compels them to buy the cheapest, they usually get that which is diseased.

PATHOLOGY.

Leptomeningitis is found with much thickening, and even the formation of osseous plaques; the anterior cornual cells of the cord are pigmented and atrophied; postero-lateral sclerosis of the columns of Goll and Burdach in the upper cord levels; and the lower portion of the crossed pyramidal tract sharply sclerosed. The increased symmetrical vulnerability of the skin, spasticity of lower limbs, vertigo, etc., I think clearly indicate we have a poison which attacks the trophic centres, and the insanity indicates special selection of the brain cells. The nerve and brain poisons seem to have a peculiar selection for the skin; we see it in the gin blossom of alcoholics, in the erythema of atropine poisons, and in the papular-pustule eruption of bromide of potash, and in food poisons causing urticaria, etc.

PROGNOSIS.

If the disease is treated early, recovery is said to be easily secured (Lavinder); if allowed to run on death is the rule in a few years. Seventy-eight per cent. of cases are curable when treated properly. Of the four cases I report, two are dead and the remaining two will not live very long, in all probability.

TREATMENT.

The treatment is to stop all diet of corn products, breakfast foods, meal, etc., and give mixed animal and vegetable diet. Tonics, such as arsenic, quinine phosphates and chalybeates are to be given; careful avoidance of exposure to the sun, and soothing lotions to the eruption. Atoxyl has been recently recommended (Lavinder). A serum treatment has also been suggested, the serum to be gotten from horses which have recovered from "Blind Staggers" caused by eating diseased corn. The only scientific treatment of the disease is prevention, and this should be attained by educating the laity in

regard to the dangers in the use of musty corn products. All millers who are found grinding musty corn, and storekeepers selling musty meal, should be fined and imprisoned. The school teachers in our State should be instructed to teach the horrors of pellagra and its cause. The farmers should have special bulletins sent them teaching the best methods of drying their corn, when to cut it, how to shock it, house it, shell it, send it to mill, and keep it when at home. No cold corn bread should be used as food for man, and all breakfast foods made from corn should be carefully examined before eaten, and none kept over after becoming damp. Dampness from salt water is especially favorable to the growth of the fungus.

VIRGINIA CASES.

There have been nineteen cases of pellagra reported in Virginia so far as I can collect; thirteen by Dr. Wm. F. Drewry, of the Central State Hospital for the Insane at Petersburg; one by Dr. J. H. Hewett, Lynnhaven, Va.; one by Dr. Smith, of Amherst, Va., and four by the writer in the Western State Hospital at Staunton, Va., Ten per cent. of the pellagrins being in hospitals, as I said before, we have about 170 now in the State. While this number seems small, the possibility and probability of the rapid spread of this horrible disease is fearful to consider, and now is the time to fight it.

I know of nothing, not even the great white plague, which will compare with this disease should it once get possession of our land. I say this without fear of being accused of pellagraphobia.

We have had four cases of well defined pellagra in this hospital. One male from Amherst County, who died in 1908, and his disease was not recognized until after reading the recent discussions on the subject; one male from Halifax County died in 1908, the disease suspected at the time, but not clearly recognized until a few months ago. We have now two female patients with pellagra; one has been in the hospital ten years and developed the disease during last spring. She has been demenated for years. She originally came from Louisa County. We received during this month one female from Pittsylvania County, near Danville, in the last stages of the disease. All of these cases came from among the poorest classes of people in the State, and became demented rapidly. There was nothing unusual in the symptoms of these pellagrins, as they ran the usual course. We have had two additional suspects, but as they improved so rapidly we sent them home on furlough apparently well. We will, however, keep in touch with them. One was a woman from Alexandria City,

and one a woman from Spottsylvania County. Why we have never had pellagra here in the United States before is a natural inquiry, this being the home of the maize. I believe the fungus possibly has been comparatively recently imported from the infested districts, brought in in the food of Italian immigrants, and would recommend that the government have the food of all immigrants examined immediately on arrival from pellagrous countries.

A CASE OF PELLAGRA IN ILLINOIS OCCURRING OUTSIDE OF AN INSTITUTION

GEORGE W. MITCHELL, M. D.

First Assistant Physician, Peoria State Hospital, October, 1909.

Patient was born in Braunschweig, Germany, of German parents. Both paternal and maternal grandparents were healthy individuals. The paternal grandfather was seventy years of age at death. Cause is not known. The father of the patient was considered a healthy man and died at the age of seventy-two. Patient states that there was very little the matter with him. The mother suffered an accident due to a fall and died a few years later at the age of sixty. There were four brothers on the father's side, one brother on the mother's side, who lived to an old age. Patient had two brothers and four sisters. The oldest brother lived to an old age, the younger brother dying from complications following a double inguinal hernia. One sister died in this country at the age of sixty-five from "bleeding of the lungs." He does not know if the other sisters are living or not. Emigrated to America in 1857 to Indiana and in 1873 came to Central Illinois. Has always been a farmer and a hard working man. Has raised and handled corn during his active working life until twelve years ago he moved to a city of ten thousand a few miles from his farm. Since that time he has done the work about his residence and drove back and forth to his farm two or three times a week. Has always been able to sleep well and at present sleeps exceptionally good, going to bed at nine and arising at six. Never sleeps in the day time. His appetite has always been good. He eat corn bread but did not care for mush or hominy. While living on the farm usually eat corn in some form several times a week, during the fall and winter months but not in the summer. After moving to town he eat but little corn and the last few years practically none. Has been married forty years, his wife at present being sixty-six years of age and is a well preserved, active lady for her years. Has always been in good health and was never sick in bed and does not look her age by fifteen years. She was always very fond of all forms of corn product, not only corn bread, but was very fond of mush and hominy and eat a great deal more corn products than her husband. Her memory is clear, intellect

sharp and has never had any sort of skin eruption, vertigo, sore mouth or chronic diarrhoea. Seven children were born to their union. The first one is alive, in good health and is thirty-nine years of age. The second, third, fourth and fifth died in infancy. The deaths varied from three to seven months after birth. There was an attempt to raise all these children with the bottle as the mother had no milk. All died with the "summer complaint." The sixth child was a girl, is in good health, married, thirty-one years of age, has two children, a boy and a girl, aged five and three respectively, both apparently bright and in good health. The son has two boys, aged nine and six respectively. Both are in good health and each carries his respective grade in school. The seventh child was born dead. The mother was at this time forty-six years of age. The patient does not remember of having the ordinary diseases of childhood. If so were not severe. Thirty-three years ago had two attacks of malaria. Six years ago an attack of "dysentery." There was some blood in stools and the patient was in bed for a few days. This occurred during the summer when there was a mild epidemic of diarrhoea. Patient says he has had some looseness of bowels each summer for several years but this summer has been constipated. He has been taking cathartics since erythema appeared "to get the poison out of his system." Otherwise has always been in very good health. Patient has an abundance of iron gray hair and presents the characteristic placid facies of the even-tempered Germans of his class. He is apparently fairly well nourished and looks much younger than his age. Patient articulates well and has the slow characteristic speech of his class. Eyes react promptly to light and accommodation. No paralysis of any of the eye muscles. Consenual present both eyes. The sight is good. Has no teeth. No inflammation of the mouth or gums. Tongue clear, quite broad. Hearing is good. Lungs negative. Respiration eighteen. Has cup-shaped depression over lower part of the sternum which is quite marked. Heart is normal in size and position. The first sound is rather loud and booming in character. The arteries can be palpitated but are certainly not sclerosed to any extent. Pulse is fifty-four in reclining posture. Blood pressure 160 (Shepherd). The abdomen is smooth and pliable and no pain on pressure. Spleen not palpable. The grip is firm and there is no paresis of any of the muscles.

REFLEXES.

Elbow reacts promptly. Patellae reacts promptly. Planter reacts promptly. Suggestion of a Babinski on the right side. Oppenheims phenomena present both sides. No ankle clonus elicited. No achilles elicited. Abdominal diminished. Pain sense in general slightly diminished. Cremasteric reacts promptly. Temperature sense normal. No areas of anathesia. No pain or tenderness over the spine. Romberg negative. Coordinates well in every way. Axillary glands enlarged and hard. Otherwise the glandular system is negative. Temperature normal. Skin lesions—erythema on exposed surface of the neck, with very fine bran-like desquammation, particularly pronounced along the hair margin and below each ear. Skin dry, parchment-like to touch and somewhat thickened and indurated. Involved surface behind the ear began with erythema; there was an irritating and itching sensation present, vescicles formed with some exudate and slight hemorrhage from raw surface when denuded. Patient says "skin on eye lids was red and caused some discomfort." At present the dorsum of right hand is swollen, skin dry, harsh to the touch, thickened and somewhat oedematous, light red in color. Entire dorsum surface involved extends from proximal phalangeal joint of middle ring and index fingers to a line one and one-half inches above the wrist joint. Entire dorsum of thumb involved and line of demarcation between dorsal and palmar surface extends from outer margin of thumb nail running upward and inward to middle of wrist. Anterior surface of the forearm is involved to an extent of two and one-half inches above wrist joint. Line of demarcation on forearm abrupt and distinct. The most severe involvement is over the dorsum of the thumb, the radial side of the dorsum of the hands and the radial side of both anterior and posterior involved surface of forearm. Over this surface everywhere the skin shows fissures, very dry and rough and present yellowish squamae which are at present quite firmly attached. Desquammation is proceeding. Color disappears on pressure with exception of some of the most indurated parts where crustace are present. Ulnar side of dorsum of hand and involved portion of forearm is thickened, dry and somewhat harsh to the touch but does not present the severe nature of the radial side. Ulnar side of wrist and forearm is more smooth and uninvolved. Left hand presents an involvement symmetrical with the right both in extent and degree except that fissures are not so marked. General outline does not differ decidedly anywhere at present. There is

a dry, thin and scaly desquammation of extensor surfaces of both arms to the shoulder. Skin condition first appeared about six weeks previous as a slight erythema on surface of radial side of each hand, gradually extended and became darker in color. Was washed with soap and water which caused the skin to become rough and cracked. Extensor surface of an arm and forearm involved at same time as hands. There is some hypertrophy, pigmentation and desquammation and roughening of skin over both heels, and outer surface of both great toe joints. Involved surface at first was somewhat painful to the touch and there was a feeling as if "little worms" were crawling about under the skin. This was very annoying to the patient and would disappear at times.

Blood Analysis:

Haemoglobin 80 per cent. (Dare.)
Erythrocytes 4,250,000.
Leucocytes count 11,600.
Differential count—negative.
Color Index.
Urine analysis from specimen voided at mid-day:
Specific gravity 1016.
Clear.
Reaction acid.
No albumen.
No sugar.
No acetone.
No peptone.
Trace of Indican.
Few epithelial cells.
Cylinderoids few.
No crystals.