showed hemoglobin 60 per cent., red cells 3,000,000, white cells 4,600, normal differential count, no plasmodia. Urine was normal. Stools large, acid, frothy, putty-colored, no parasites, no occult blood, Schmidt test positive. Microscopically, undigested food, fat droplets, and fatty acid crystals made up the picture.

The patient was put on a milk and monosaccharid diet, without any medication, and a month later eggs, cereals, baked apples, potatoes, butter, and white meat added. She was permitted to go home under observation in September, and finally discharged in December. At this time she weighed 136 pounds, had a systolic blood-pressure of 119 mm. Hg, felt perfectly well and was having one or two stools a day.

In the treatment of sprue a great number of remedies have been recommended, such as potassium chlorate with morphin and bismuth,⁶ powdered cuttlefish bone,⁷ colloidal silver,⁸ phenyl salicylate (salol), betanaphthol, mercuric chlorid, cyllin, izal (cresol preparations), acetozone and ipecacuanha.⁹ A few years ago Begg¹⁰ brought forward yellow santonin as a specific remedy, but Rogers, Manson and Castellani, working in India, have all failed to confirm Begg's results, and Ashford¹¹ working in Porto Rico, says, "Santonin, therefore, if it has any such specific action as that intimated by Begg, does not seem to be superior to a carefully supervised diet, especially a milk diet."

Rest in bed is obviously essential. The use of any drug as a specific is at present purely empirical. Moderate use of alkali seems rational. In severe cases the exclusive meat diet, or exclusive whole milk diet seems to us irrational. The fruit diet is simply a monosaccharid diet, and this in conjunction with skimmed milk seems to us to be the best diet to use in severe cases. The advent of cold weather, particularly cold nights, has apparently been of greater benefit to our patients than any other single factor. We know that the best treatment for pellagra is removal to the mountains during the hot months, and for the same reasons we think that in active cases of sprue the patients should not attempt to remain in the tide-water or piedmont regions during June, July or August.

Stuart,¹² Burnet,¹³ Jackson¹⁴ and others,¹⁵ maintain that sprue and pellagra are the same disease, or in other words that sprue is "pellagra sine pellagra." While our experience with sprue is very limited, our experience with pellagra has been considerable, and we see no reason to confuse these two diseases. They have in common sore mouth, diarrhea, secondary anemia, loss of flesh and strength. Sprue has none of the nervous, skin, uterine, vaginal and rectal symptoms of pellagra, while pellagra is not characterized by the distention, and usually not by the early morning type of diarrhea found in sprue. The stools in the two diseases are totally different; the pellagra stool is large and liquid but composed of food remains hurried through the intestinal canal; the intimate admixture of fine bubbles of gas, the intense acidity and the characteristic picture of partially split fats are entirely wanting. We have elsewhere warned

against the diagnosis of pellagra on the grounds of sore mouth and diarrhea.¹⁶

SUMMARY

Cases of sprue are constantly sifting back into this country and should be recognized and treated as such. Sprue should not be confused with pellagra. Rest in bed, a monosaccharid and skimmed milk diet and cold weather seem to be the most important factors in the treatment. Sprue may very possibly be or become endemic in the Southern states.

ABSTRACT OF DISCUSSION

DR. ROGER S. MORRIS, Clifton Springs, N. Y.: Did Drs. Hiatt and Allen make gastric analyses in their cases or not? The diarrhea which the patients had is described as a morning diarrhea, and it occurs to me that this may have been associated with conditions in the stomach.

DR. H. B. HIATT, High Point, N. C.: We made gastric analyses in all cases and nothing in particular was obtained from them. Sometimes we met achlorhydria and sometimes the conditions were normal. The morning diarrhea we considered to be the result of the low blood-pressure. The object of reporting the cases was to call attention to this subject and to utter a warning. If sprue should become endemic in the South its spread might be as rapid and the disease become as common as pellagra.

THE BLOOD IN ACIDOSIS FROM THE QUANTITATIVE POINT OF VIEW*

W. McKIM MARRIOTT, M.D.

ST. LOUIS

Although the elimination of acetone bodies, found in the urine in various conditions, has been thoroughly investigated, we still lack reliable information as to the extent to which these substances may accumulate in human blood and tissues during life. Diminished alkalinity of the blood, such as may occur in acidosis, is a condition unfavorable to diuresis, so that it is quite possible to have only a moderately high excretion of acetone bodies in the urine of a patient who may exhibit symptoms of severe acid intoxication. It is in such cases that an analysis of the blood should yield data of diagnostic and prognostic value.

By a method, which is described elsewhere,¹ it is possible to determine acetone, aceto-acetic acid and beta-oxybutyric acid in small amounts of blood drawn directly from a vein. From 2 c.c. to 5 c.c. of blood is sufficient, and the analysis may be carried out in about the same time as is required for an analysis of the urine for the same constituents. When analyzed according to this method, normal blood from man, dog, pig and beef is found to contain less than 1.5 mg. of gross aceto-acetic acid (acetone preformed, plus aceto-acetic acid), and less than 4 mg. of oxybutyric acid per 100 gm. of blood, all results being expressed in terms of acetone.

A number of pathologic conditions associated with acidosis have been studied, including light and severe diabetes, febrile conditions of childhood, postoperative acidosis and experimental phloridzin and pancreatic diabetes in dogs. In all these conditions the acetone

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7. Cantlie, J.: *Brit. Med. Jour.*, Nov. 15, 1913, p. 1296.

8. Manson, Sir P.: *Tropical Diseases*, New York, William Wood & Co., 1908, p. 477.

9. Rogers, L.: *Dysenteries*, Oxford Univ. Press, 1913, p. 320.

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11. Ashford, B. K.: *Am. Jour. Trop. Dis.*, 1913, i, 146.

12. Stuart, C. E.: *Abstr., Jour. Trop. Med.*, 1913, xvi, 287.

13. Burnet, J.: *Are Pellagra and Sprue One and the Same Disease?* *Abstr., THE JOURNAL A. M. A.*, Sept. 16, 1911, p. 1014.

14. Jackson, T. W.: *Tropical Medicine*.

15. Tyson and Fussell: *Practice of Medicine*, p. 1164.

16. Allan, W.: *New York Med. Jour.*, Dec. 18, 1909.

* Read before the Section on Pathology and Physiology at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

1. Marriott: *Jour. Biol. Chem.*, August, 1914.

bodies were present in the blood in excess of the normal. The highest figures obtained were from the blood of a patient with severe diabetes at a time when he exhibited signs of impending coma. In this instance, the oxybutyric acid amounted to 45.2 mg. per 100 gm. and the gross aceto-acetic acid to 28.0 mg., both calculated in terms of acetone.

The accumulation of the acetone substances in the blood and the excretion of these substances in the urine did not always run parallel. Administration of alkali resulted in considerable alteration in the relative distribution of the acetone substances between the blood and the urine, the effect being to diminish very considerably the concentration of these substances in the blood, without at the same time bringing about any appreciable change in the total daily output in the urine.

Analyses of the blood for preformed acetone indicated that this substance may amount to more than 60 per cent. of the gross aceto-acetic acid, in conditions of severe diabetes. With alkali therapy, preformed acetone showed a relative diminution. That is to say, the alkali appeared to decrease the decomposition of aceto-acetic acid.

In the bloods examined the ratio of oxybutyric acid to total acetone substances ("beta ratio" or Kennaway, Pembrey and Poulton) varied from 50 per cent. to 80 per cent. The significance of this ratio is not, as yet, entirely apparent. It is possible that a high ratio may be an indication of partial asphyxia; for we know from the work of Embden, Friedmann, and Dakin and their collaborators that, with diminished oxygen supply, various organs of the body readily effect the reduction of aceto-acetic acid to oxybutyric acid, whereas the reverse change is favored by the presence of an abundance of oxygen.

So far I have spoken only of the accumulation of the acetone substances in the blood, and have said nothing of the tissues. The blood, however, appears to be a fairly satisfactory index of the extent to which acetone substances may accumulate in the tissues. For example, it was found that the blood and liver of a starved phloridzinized dog contained the same amounts of acetone bodies per 100 gm. and the muscles approximately one-half as much. The beta ratio, however, was higher in the muscles and liver than in the blood, and this would, of course, be expected as there would be relatively less oxygen in these organs than in blood.

It is not intended here to present a detailed discussion of acidosis but merely to indicate some applications of a method which I believe to be capable of throwing light on the subject of acetone body acidosis.

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ABSTRACT OF DISCUSSION

PROF. A. J. CARLSON, Chicago: I should like to ask Dr. Marriott for the findings in the pancreatic diabetes in dogs. It has been my experience in this experimental diabetes that there is not in the urine or in the breath of these dogs the characteristic acetone odor that is found in advanced cases of human diabetes. Was there any indication of increase of acetone in the experimental dogs comparable to that in cases of human diabetes?

DR. W. MCKIM MARRIOTT, St. Louis: I was surprised to find that in pancreatic diabetes in the dog there was only the very slightest increase in acetone bodies, nothing at all comparable to that of human diabetes or even of phloridzin diabetes in the dog; the figures were just above the normal.

DIPHTHERIA OF THE SKIN OF UNUSUAL TYPES

REPORT OF TWO CASES *

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PHILADELPHIA

Diphtheria of the skin was first recognized by Chomel¹ in 1759, and by Samuel Bard¹ in an epidemic in New York in 1771. Although the diphtheria bacillus was not discovered until 1883 by Klebs¹ and 1884 by Loeffler¹ of Greifswald, working independently, as early as 1830 Trousseau¹ wrote extensively of cutaneous diphtheria. It was not, however, until 1891 that the integumentary outbreak of this affection was studied not only clinically but also bacteriologically by Neisser.²

The invasion of the skin by the Klebs-Loeffler bacillus is of infrequent occurrence, as shown by the report of the St. Anne's Children's Hospital of Vienna.² From 1894 to 1902, out of 2,217 clinical and bacteriologic cases of diphtheria the cutaneous surface was attacked in but twenty-three instances, and the most of these were of the false-membrane type. Mallory¹ has found in a series of 251 cases of diphtheria that the skin was attacked in but one instance and the lesion was then of the false-membrane type.

Trousseau's original contention that cutaneous diphtheria was preceded by some tissue loss, no matter how slight, is the view held by the present-day investigators, notwithstanding Daviot's³ view to the contrary.

The bacillus gains entrance to the skin through some opening, in cracks, slight excoriations, erosions resulting from herpes, in the breaks in the continuity of the skin secondary to an eczema, an intertrigo or an impetigo, etc. Cutaneous diphtheria may be of primary occurrence and limited to the integument; the infection may be conveyed to the larynx, throat or nasal mucous membrane, or, as is usually the case, the skin is secondarily involved, the mucous membranes, just mentioned, showing the original lesions.

Inoculation of the skin with the Klebs-Loeffler bacillus occurs in one of three ways: by auto-inoculation, by means of some article of clothing, utensils, etc., containing the same, and from one person to another by close contact, as by kissing, coughing, circumcision, etc.

Numerous varieties of lesions have been reported in which the diphtheria bacillus has been found. The most frequent of these is the false-membrane type. The other types which have been recorded are the ulcerative, gangrenous, eczema-like, impetiginous eczema-like, pustular and impetigo-like, ecthymatous, vesicular and varicella-like, bullous, dermatitis herpetiformis-like, carbuncular-like, and the tumor and abscess.

False-Membrane Type.—The older writers on diphtheria of the skin affirmed that every case had a diagnostic false membrane. The break in the conti-

* Read before the Section on Dermatology at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

1. Quoted by Nuttall, G. H. F., and Graham-Smith, G. S.: The Bacteriology of Diphtheria, New York, G. P. Putnam's Sons, 1908.

2. Quoted by Marschalko: Arch. f. Dermat. u. Syph., 1909, xciv, 379.

3. Quoted by Jack: Brit. Jour. Child. Dis., 1913, x, 456.