"anterior metatarsalgia," etc., are not, strictly speaking, of diagnostic significance, as they refer to symptoms only.)

With regard to the terms "flaccid" and "rigid," this distinction is important, but serves only to describe one aspect of the physical process, irrespective of origin; it does not of itself suggest the pathological basis of the trouble.

The same holds good for "flat foot." If the term is used in the manner above described, it is simply a description of a physical picture. It is, therefore, seen that the term "flat foot" may apply both to feet which are not diseased and to those that are diseased, but it is also plain that if a foot is apparently diseased as shown by the history, the swelling, the muscular spasm, the pain, the disability, etc., then the lowering of the scaphoid tubercle by indicating the mechanical relation between the bones becomes an important additional piece of evidence, in many cases suggesting the severity of the process as well as the residual strength of the foot.

Medical Progress.

PROGRESS IN PHYSIOLOGICAL CHEMISTRY.

BY J. BERGEN OGDEN, M.D., NEW YORK.

CHEMICAL INVESTIGATION CONCERNING THE ETIOLOGY OF ECLAMPSIA.

B. J. DRYFUSS¹ has made an extensive study of this subject, and the results of his research are briefly stated below.

Fresh normal placentas were divided into two parts, one half analyzed immediately and the other half after seven days' autolysis. The total solids increased during autolysis from 0.132 to 0.266%. In the normal placenta the total nitrogen was distributed as follows: Amide nitrogen, 4.3%; diamino nitrogen, 68.3%; amino acid nitrogen, 46.2%. The comparable figures for the autolysis specimens were: Amino nitrogen, 9.2%; diamino nitrogen, 40.0%; amino acid nitrogen, 61.3%. A comparison of the normal fresh placenta with the eclamptic placenta showed a total soluble nitrogen content nearly twice as great, 0.2493%; whereas in the normal placental autolyses the non-coagulable nitrogen became doubled, the amide nitrogen was increased 4.5 times. The de-aminization was, therefore, more than twice as active as the proteolysis. In comparing the normal fresh placenta with the fresh eclamptic, the de-aminization in the latter is not so great as in the former. The formation of amino acids in autolysis has not occurred as rapidly as in the autolysis of the normal fresh placenta. The diamino acids show little change after autolysis. The injection of powdered normal or eclamptic placentas, suspended in water, into the peritoneal cavity of pregnant rabbits was without results in so far as the detection of differences was concerned. Intraperitoneal injections of eclamptic serum and extracts of eclamptic placentas were also negative. Experiments tending to prove the presence of an endotoxin in the placenta from eclampsia were somewhat doubtful.

The opinion is expressed that lactic acid stands in no causal relationship to eclampsia; it must be considered rather as a result of the diminished oxidation which accompanies the seizures, etc. The exclusive placental origin of eclampsia has never been demonstrated.

THE HEMAPHEIC REACTION OF URINES.

Dufare² has found that a urine gives the hemapheic reaction if, when it is placed in contact with HNO_3 , one obtains in the vicinity of the plane of separation a brownish-mahogany coloration, of which the intensity is more or less marked and decreases gradually toward the outer layers. The author confirms the statement of Engel and Kierner, that it is impossible to obtain the mahogany tint with urobilin alone, as maintained by Florence. If defecation of the urine be accomplished with $\text{Pb}(\text{OAc})_2$ or $\text{Ba}(\text{OH})_2$, instead of with H_2SO_4 , one obtains with HNO_3 a sort of disassociation of the mahogany coloration, i. e., a red ring of variable intensity, and immediately below it a yellow ring, but this may be obtained also by simple dilution with water, less sharply, however, than if lead or barium be employed. While hemapheic urines always contain urobilin, they also always contain appreciable quantities of indoxyl, and the author believes that it is this indoxyl which, in the form of indirubin, regularly intervenes to form the red, but it is also probable that skatoleic red plays an important rôle. The hemapheism of Gubler is, therefore, not characterized by the presence in the urine of any particular pigment, and hemapheim must be withdrawn from the list of urinary pigments. The rôle of urobilin is then only to temper the red coloration produced by indoxyl.

A NEW BILE ACID REACTION AND THE DETECTION OF THE BILE ACIDS IN URINE.

A. Jolles³ describes the following: If 2 to 3 ccm. of a 0.1% solution of taurocholate and glycocholate or pure cholic acid are boiled for a short time with 1 to 2 drops of a 5% rhamnose solution and 2 to 3 ccm. concentrated HCl , a red color is produced followed by a beautiful green fluorescence. A concentrated cholate solution gives a red-brown color by transmitted light, and a malachite-green fluorescence in reflected light. From 0.0005 to 0.0001 gm. of cholic acid can be detected. The fluorescence is caused by the methyl furfural derived from the rhamnose. The reaction applies only to cholic acid derivatives and not to protein, urea, carbohydrates, CS_2 , or the acids of the aliphatic or aromatic series. To detect the bile acids in urine, mix 50 ccm. urine with 15 ccm. of a 3% casein solution, and add 10% H_2SO_4 , drop by drop, shaking until the casein is entirely precipitated. Filter, and to the precipitate add 10 ccm. absolute alcohol and leave at room temperature about one hour, stirring frequently. Filter, and to 4 to 5 ccm. filtrate add one drop of a 5% rhamnose solution and 4 to 5 ccm. concentrated HCl ; heat to boiling and boil gently one minute. Cool, add 2 ccm. ether and shake. If bile acids are present, there is fluorescence. As little as 0.05%

sodium taurocholate can be detected. In concentrated urines, and especially in those rich in indican and aromatic hydroxy acids, the test is less satisfactory, for the alcoholic filtrate after boiling with HCl is so deeply colored that the detection of the fluorescence is more difficult. This difficulty may be obviated by sending a ray of light through the solution with a lens.

THE NECESSITY OF EXAMINING THE URINE AT DIVERSE PERIODS OF THE DAY, ESPECIALLY THAT OF CHILDREN AND OLD PEOPLE.

While it is ordinarily advisable to examine a sample of the mixed twenty-four-hour urine in every case, A. Richoud and A. Bidot⁴ have pointed out that it is at times advisable to examine specimens of urine voided at different times of day in order to get a clear picture of the case. The authors show that if an examination of urine be made from the whole twenty-four-hour sample, as is usually the case, and necessary in most, a very serious error in diagnosis may result. The case, a child of eight years, was admitted to the hospital suffering from apparent nephritis, the diagnosis resulting from the detection of albumen in the twenty-four-hour sample of urine. The patient did not improve after six months' treatment. The urine was then examined at different periods of the day and that passed immediately after waking was found to contain no albumen. Based upon a new diagnosis, resulting from this discovery, another system of treatment was inaugurated and the patient improved.

AN INVESTIGATION OF THE FOLIN METHOD FOR THE DETERMINATION OF CREATININ AND CREATIN.

In a research by Weber⁵ it was found that, in the perfused mammalian heart, the active muscle gives out more creatin and creatinin to the blood than the inactive muscle. In dogs with paralyzed muscles, as compared with those which have cinchonine spasms, one finds, in paralyzed muscle, a decrease in the content of creatin; in passive muscle after section of the nerve, a very marked decrease. The muscle with cinchonine spasms forms more creatin than does passive. Muscle convulsions in starving dogs produce a distinct absolute increase and a decrease in relation to total nitrogen. Violent muscular exercise decreases the creatinin output in fed animals. The authors do not agree that creatin and creatinin are a separate compound generally, as Folin has suggested. The increase in creatinin in the urine after the administration of meat extract is greater than the amount of creatinin in the extract.

THE ORIGIN OF ENDOGENOUS URIC ACID.

As a result of the experiments of E. B. Cathcart, E. L. Kennaway and J. B. Leathes,⁶ a marked increase in the output of endogenous uric acid was found to occur in fever, exposure to cold and after severe exertions. The increased output coincides and terminates with the febrile rise of temperature, coincides with and outlasts by many

hours the exposure to cold, follows the exertions and lasts for many hours after them. It is suggested that in all these conditions the uric acid has its origin in metabolic processes occurring principally in the voluntary muscles and not immediately related to voluntary contractions and work. The daily variation in uric acid excretion, high in the morning, low at night, is not due to retention of uric acid formed during the night, nor to the fact that the digestive organs are inactive at night, but it is due to the quickened activity of all functions, especially those of the voluntary muscles, which results from the rest of sleep. Generally speaking, the more lively the performance of the functions of the body as a whole, the greater will tend to be the amount of uric acid produced.

THE EXCRETION OF UROCHROME IN THE URINE OF NORMAL INDIVIDUALS AS WELL AS IN CERTAIN DISEASES.

St. Dombrovski⁷ has pointed out that the copper reagent employed in the Krüger-Wulff method precipitates urochrome together with the purin compounds. This varying factor, at first unknown, rendered this general method of doubtful value. The fact of the precipitation of the urochrome with copper renders a quantitative determination of this pigment comparatively easy. It is only necessary to determine the nitrogen of the copper precipitate in the Krüger-Wulff method and then to subtract from it the nitrogen of the purin compounds as given by the Camerer-Arnstein or Huppert-Haycraft methods. The difference indicates the nitrogen of the urochrome. By this method, four samples of normal urine gave a twenty-four hours' elimination of urochrome equal to 0.458 to 0.741 gm. The copper salt of urochrome is easily soluble in ammonia and in such solution is not precipitated by silver nitrate. This affords a means of separating the pigment from the purin compounds. Employing copper acetate as a precipitant for purin compounds and urochrome, the total nitrogen of these can be determined. Employing ammoniacal silver nitrate, they may be separated and their nitrogen determined. In five cases of typhoid the urine examined by this method showed 0.779 to 1.057 gm. of urochrome, or nearly three times the normal amount.

THE RELATION OF NITRIFYING BACTERIA TO THE UROSEIN REACTION OF NENCKI AND SIEBER.

In the study of the urine of a child seven years old, suffering from a peculiar variety of intestinal decomposition, due to an abnormal type of intestinal flora, Herter⁸ noticed that the urine had the urochrome reaction with hydrochloric acid, but that the reaction developed only in such samples as had stood in the laboratory at least twelve to twenty-four hours. Fresh urine was negative. The onset of the reaction coincided with the development of a turbidity due to bacteria. Urines protected from contamination failed to develop the reaction, but if the sample was inoculated with bacteria from the urine which had already

developed the reaction, the color changes were obtainable in twelve to twenty-four hours. These organisms were obtained in pure culture and, among other characteristics, formed nitrites. Sterile urines developed nitrites after inoculation with these bacteria. It was further found that fresh urine to which was added a few drops of 0.2% solution NaNO_2 gave the reaction immediately in a characteristic fashion. Nitrates were negative. Normal urines plus nitrites never gave the urochrome reaction either immediately or upon standing. The second factor in the reaction was the presence of some chromogen which gives the reaction with nitrous acid. Other oxidizing agents gave a color closely resembling that of urochrome, although the tendency to over-oxidation was then present. Sometimes the color of the urochrome reaction with nitrite was a purple or violet, according to the mode of oxidation. This violet color is not removable by chloroform as is the case with indigo in the indican reaction. There is no relation between the urochrome coloring matter and skatol red, although in amyl alcohol solution their spectroscopic appearance may be indistinguishable. Skatol could not be obtained from the intestinal contents of the patients. Through the action of the intestinal bacteria from the case under observation it was possible to obtain a coloring matter having the chief characteristic of urochrome by subjecting sterile whole milk to the action of the fecal bacteria. In a note it is stated that this chromogen is indol acetic acid.

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- ⁴ Jour. Pharm. Chim. (6), xxviii, 199-201.
- ⁵ Arch. Exp. Path. Pharm., lviii, 93-112.
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- ⁷ Zeitschr. Physiol. Chem., liv, 390-397.
- ⁸ Jour. Biol. Chem., iv, 239-251.

(To be continued.)

Reports of Societies.

THE AMERICAN DERMATOLOGICAL ASSOCIATION.

THIRTY-THIRD ANNUAL MEETING, HELD AT THE BELLEVUE-STRATFORD HOTEL, PHILADELPHIA, PENN., JUNE 3, 4 AND 5, 1909.

SECOND DAY, FRIDAY, JUNE 4.

(Concluded from No. 17, p. 594.)

THE ETIOLOGY OF PSORIASIS. ITS PRESENT STATUS.

DR. SIGMUND POLLITZER, of New York, opened a general discussion on this subject. He reviewed at length the various hypotheses that had been advanced concerning the nature of psoriasis and said that our knowledge of the subject at the present time might be summed up in the following conclusions: 1. That rheumatism, gout, neuroses and heredity were not direct etiological factors in the production of psoriasis, but in the present state of our knowledge it could be neither denied nor affirmed that they might have some bearing on the obscure conditions of the system

which rendered it more or less susceptible to infection. 2. That psoriasis was one member of a group of parakeratoses to which seborrhea corporis and (in part) eczema seborrhoicum belonged. 3. That it was most probably due to an external microbial infectious agent.

THE QUESTION OF THE PARASITISM OF PSORIASIS.

By DR. JAY FRANK SCHAMBERG, of Philadelphia.

In discussing this subject, the author said that three propositions might be formulated for consideration: 1. That psoriasis might be the result of the circulation in the fluids of the body and deposition in the skin of a micro-parasite, analogous to what was observed in syphilis and variola. 2. Psoriasis might be due to the implantation upon the skin of an exogenous parasite, as was observed in ringworm, favus and tinea versicolor. 3. The disease might be due to one of the common facultative parasitic organisms ever present upon the skin in individuals in whom a constitutional predisposition rendered the soil favorable. After discussing each of these propositions in detail, and reviewing the literature and experimental work that had been done on the subject, Dr. Schamberg said it might be argued that the failure to auto-inoculate psoriasis in the vast majority of subjects operated against the assumption that it was a local parasitic disease due to exogenous organisms. It might furthermore be argued that if the lesions of psoriasis were due to the implantation of a parasite upon the skin of a susceptible individual, inoculation with lymph, blood and scales from a diseased patch upon the healthy skin in the same patient might be reasonably expected to reproduce psoriasis lesions. Our lack of knowledge, however, concerning possible conditions of immunity in this connection made it unwarranted to deduce any positive conclusions of the character indicated. The inability to auto-inoculate psoriasis patients would not at all affect the hypothesis that psoriasis was a constitutional, infectious disease. From what had been stated, it was obvious that no definite conclusion as to the parasitism of psoriasis was warranted. The mode of growth and configuration of the patches, the occasional instances of apparent communication of the disease and the Destot inoculation were arguments in favor of parasitism which could not be decisively brushed aside. On the other hand, the general behavior of the disease, the common experience of observers as to the absence of contagion, and the almost uniform failure of inoculation experiments constituted strong evidence against parasitism. Wisdom should prompt us, in the light of our present knowledge, or, more accurately speaking, in the dimness of our inadequate knowledge, to commit ourselves neither to positive affirmation nor to uncompromising negation.

DR. JAMES C. WHITE said that psoriasis might be called a family disease, or, to a certain extent, a hereditary disease. We knew that it succeeded quite a number of other cutaneous diseases, but that did not necessarily imply any etiological relationship between the two. When we mentioned these two factors as having some direct influence on the production of psoriasis, he thought we had told the whole story, so far as we had any direct knowledge of the nature of this disease.

DR. LOUIS A. DUHRING, of Philadelphia, said that up to the present time we had no convincing proof of the parasitic origin of psoriasis.

DR. L. DUNCAN BULKLEY, of New York, said that all were ready to acknowledge that there was something back of psoriasis that we did not know anything about. There was no question that there was something on the skin which produced these lesions on a favorable soil. He had some patients under his care for many