rather supervenes on account of the developmental structural inefficiency than on account of "diabetes," though the symptom-complex may point to a fatal termination by the latter. The disease in childhood, in most cases, does not seem to be caused by any metabolic disorder as such, and I do not hesitate to predict that in the near future the life of many children affected with "diabetes mellitus" so-called, will be saved by simple operative procedures within the skull.

DR. LOUIS J. LAUTENBACH, Philadelphia—In connection with this paper, there is a subject which seems to me to have come up in every Section I have attended this year. It may be a fashionable complaint, or it may be a common disease which has not been generally recognized. I notice the author referred to adenoids as a possible factor in the causation of his cases. I was surprised that no mention was made of whether or not he removes them. These adenoids in the vault of the pharynx seem to be an expression of some deep general condition, probably tubercular, syphilitic or scrofulous, the exact nature of which is not apparently known as yet. In all of the cases of phlyctenular conjunctivitis I have seen there have been adenoids in 40 per cent. or more of the cases. This may give us a clue to the nature of the disorder. We specialists take the ground that whenever we know adenoids are present it is our duty to remove them promptly. Whether adenoids are a part of a strumous condition or not I do not know, but I do know that their presence does induce a series of other phenomena which should be avoided whenever possible, and the only thing to do is to remove the postnasal obstruction at once.

DR. LOUIS FISCHER, New York City—I had the pleasure of seeing at my clinic several months ago the cases reported in the paper. I examined the urine and found a large percentage of sugar. A very important point, it seems to me, and one brought out in the paper, is the fact that those cases which are tuberculous or of a type formerly called scrofulous, are the ones in which we look for these abnormal conditions. It was only two or three weeks ago that I undertook to examine a series of urines from children in private practice, and in the urine of 61 children I found one case that showed a transitory glycosuria. Sugar was found in the urine from this child on three different occasions, though the child presented no symptoms whatever pointing to ether diabetes mellitus or insipidus. The child has a little athrepsia and general malaise, yet the urine contained 1 per cent. of sugar. It would be well, I think, for us to make it a rule to examine the throat and the urine of every child coming to us for examination.

DR. HEINRICH STERN—May I ask by what method the uranalysis was performed?

DR. FISCHER—By Fehling's test and the fermentation test.

DR. EDWIN ROSENTHAL, Philadelphia—I have only seen two cases of diabetes mellitus, and the impression that I gained was that it was the first beginnings of life that started diabetes. In one of these children, 5 years of age, having a good family history, the diet had been an artificial food. The child died at the age of 5 years. The second case was the grandchild of a professor of obstetrics in Philadelphia. The child was a girl. The prognosis in these cases is invariably bad. The question of consumption, syphilis and similar disorders does not enter at all into these cases. I myself thought the diabetes in these cases was the result of the method of bringing up the children. One of the children had never been nursed, having been entirely fed artificially.

DR. J. C. DE VENNEY, Harrisburg, Pa.—I wish to state that I have seen a case similar to the one mentioned by the last speaker. It had been fed on malted milk and an artificial food until 4 years old. It was a well-marked case of diabetes.

Pathogenesis of Acute Deafness.—Baginsky relates in the *Arch. f. Kindhk.* xxviii, that a girl of 13 presented the clinical picture of cerebrospinal meningitis with complete deafness in two days. The latter persisted after recovery. She succumbed three months afterward to sepsis from a malignant carbuncle on the lip. At the autopsy no traces of the supposed meningitis could be discovered, but indications of a bilateral lesion of the labyrinths were unmistakable. The organ of hearing had been completely destroyed by endostitis ossificans, which had manifested itself clinically as cerebrospinal meningitis.

MALARIAL HEMOGLOBINURIA.*

WILLIAM BRITT. BURNS, M.D.
DECKERVILLE, ARK.

We are accustomed to using this term—the principal symptom of the disease—because it overshadows all others. Under ordinary circumstances it makes the diagnosis, and the layman knows its import.

The synonyms are, black-water fever (Das Schwarzwasser Fieber), hematuria, hemoglobinuric fever, swamp fever, icterohematuric fever; fièvre bilieuse hématurique, first described by French naval surgeons stationed at Nossibe, a French settlement off the northwest coast of Madagascar.

The pathology and pathologic anatomy of malarial hemoglobinuria is in all essential particulars the same as—and in fact is—that of a malignant malaria, with the addition of hemoglobin in the urine and rapidly increasing jaundice. There is always a history of one or more paroxysms—chills and fever—with insufficient or no treatment.

A cachectic may have had no active manifestations of malaria for months, and after undue exposure or fatigue, have a violent hemoglobinuria and die in twenty-four hours. Usually, however, there are two, three or four, or even more paroxysms, immediately prior to the hemoglobinuric symptoms. There may or may not be a cold stage; my cases have all shown a short mild cold stage with distressing prolonged fever, sometimes very high temperature, more often not to exceed 103-4 F. When the fevers have been of short duration the urine has cleared up rapidly; this has been no assurance though that at the end of twelve or twenty-four hours longer an exacerbation of all symptoms would not come on in an aggravated form.

The general appearance of the patient, if seen early after the coloration of the urine, is one of more or less excitement; the face is blanched as if in extreme shock; the eyes follow every movement; the speech is catchy and respiration sighing; he may be fairly quiet, more often there is restlessness, at times tossing over the bed, seeking a comfortable place; yet he seldom complains of pain. Indeed, the rule seems to be that there is very little pain, and if asked will answer that there is no suffering; but he goes on sighing and groaning. I have seen persons with a notably clear skin and scleræ pass black water with a first or second paroxysm and in twelve hours present the color of saffron. The spleen and liver are nearly always palpable below the costal margins and are extremely tender; some tenderness exists over the kidneys, sometimes extreme backache. Nausea and vomiting are usually present, but it is only rarely that it is so prominent that the stomach has to be abandoned in treatment altogether. These symptoms obtain far more often in acute malaria than in hemoglobinuria. The vomited matter may be at first a straw color, but it soon becomes green, dark-green, brown, almost black, blue-black and grumous, sometimes offensive. After icterus comes on, the saliva, perspiration and lachrymal fluids are loaded with bile pigment, which responds to bile tests. The urine is from a poke-berry juice to a black-coffee color. All of these fluids, and the feces when diluted, show a saffron stain and deposit of bile salts on linen. Consequent upon repeated and accumulating infection we have the destruction of red corpuscles by the malarial parasites, the liberation of hem-

* Presented to the Section on Practice of Medicine, at the Fifty-first Annual Meeting of the American Medical Association, held at Atlantic City, N. J., June 5-8, 1900.

oglobin along with the malaria toxins into the blood plasma.

Under ordinary circumstances the liver cares for the hemoglobin by converting it into bile pigment, and the remnant corpuscles by elaboration into young corpuscles, or the complete destruction of same. But there are metes and bounds beyond which the liver will not stand. Besides its metabolic functions, constructive and destructive, its other functions must be safeguarded, and it must also protect itself from a flank movement from the intestinal tract, from which source come our intestinal toxemias. One-sixth, or even one-third, of all the red corpuscles may be destroyed by one pernicious paroxysm, this coming when the hemopoietic function is overthrown; the liver now fails to care for the free hemoglobin here liberated, and a hemoglobinemia obtains. It must not be understood that each parasite-destroyed cell contributes its normal quota of hemoglobin to the existing hemoglobinemia; it is considered that the greater percentage is consumed and converted into proper tissue and pigment by the parasites.

It is suggested that there is set free at sporulation a certain hemoglobin-dissolving substance, which hitherto has acted as a digestive agent in the assimilation of the coloring matter of the red cells and which retains after liberation its digestive function, continues to act as a hemoglobin solvent, and so dissolves out hemoglobin from healthy cells, or at least, probably, unstable young, and vulnerable red corpuscles. May it not be possible to recover some of the hemoglobin from the malarial pigment thus set free by the same agent?

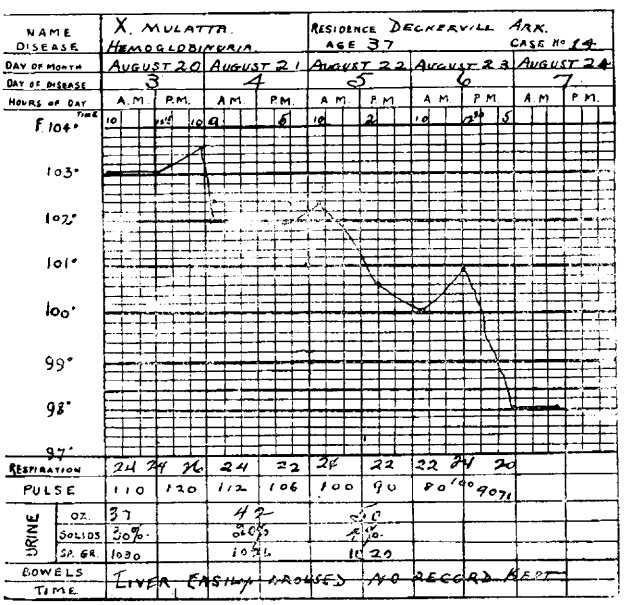
We know that there is a distinct poikilocytosis; at times there does not appear to be a sound corpuscle in the patient's body. The erythrocytes are terribly misshapen, crenated, spiculated, tailed and buckled, shriveled and otherwise deformed; there are microcytes, megalocytes and shadows, and after the second or third hemoglobinuric paroxysm, the vessels are empty and relaxed, and the blood so deteriorated that it is a difficult matter to get a smear. The blood does not exude, even after large punctures. The adhesive quality is lacking to the extent that the ordinary adhesion of the lens through immersion oil drags the cover-glass from the slide. Here the field is practically colorless, save the débris and a few ghost-like corpuscles; the wonder is that there should ever exist a hemopoietic power sufficient to restore such a blood to normal. There is now added to the destruction wrought by the plasmodia *per se,* their toxins. Moreover, the presence of bile salts and bile acids induces a cholemia, which latter condition, says Oliver, comes on before the appearance of pigment or icterus in all forms of jaundice.

If we now reflect upon the toxicity of the bile salts, we may be able to explain in part the appearance of such a quantity of hemoglobin in the urine, which is not satisfactorily accounted for by parasitic destruction *per se.* We find that even in aqueous solution of 2 per cent. bile salts kill one kilogram of body-weight; the chlorate of sodium in dose of 54 cg. and chlorate of potassium in dose of 46 cg. Bouchard says that during twenty-four hours a man makes by the activity of his liver alone, enough poison to kill three men of his own weight.

Indeed, a cholemia alone may produce a hemoglobinuria; injection of bile salts into the circulation has produced hemoglobinuria in animals. Moreover, there are various solvents which find their way into the blood-current and which will take from the erythrocytes their hemoglobin, or a part at least, without accomplishing the destruction of the protoplasmic body. The extreme pallor of the uninfected red cells is a striking evidence that there are hemoglobin solvents in the circulation which do not otherwise injure these bodies.

Maragliano explains paroxysmal hemoglobinuria on the idea of a lessened resistance to the ordinary environments, and especially to the toxic plasma.

The effect on the liver of this great hemolysis may be polycholia—filling of the gall-bladder; injection of the bile capillaries, even to their finest rootlets. The liver cells are thinned, capillaries are dilated and in places



This mulatto had been passing claret-colored urine for over forty-eight hours before I was summoned. He was very weak, his skin saffron-colored, and his eyes injected with bile. Fever and hemoglobinuria were continuous. I at once gave calomel gr. xvi, turpentin, gtt. xx, to be repeated gr. ii and gtt. ii, respectively, every two hours until the urine should clear up. I also gave quinin dihydrochlorate, gr. xv, hypodermically at 10 a. m. and 2 p. m. on the 20th.

Urine.—Claret-colored; containing blood and granular casts, amorphous granular matter and hematin crystals.

Blood.—Very pale; red cells almost devoid of color. Spiculated, tailed and buckled pigmented leucocytes, crescents and ovoids. There was seen one presegmenting form.

At 10 a. m. on the 21st the urine was clearing up slowly. Calomel was replaced by sodium hyposulphite and quinin sulphate was given in 7-gr. capsules every three hours, while the turpentin was continued. Raw eggs were also given.

At 2 p. m. the urine was clear, and the blood showed pigment and pigmented leucocytes. The urine remained clear, and the temperature gradually decreased. He was dismissed on the 23d.

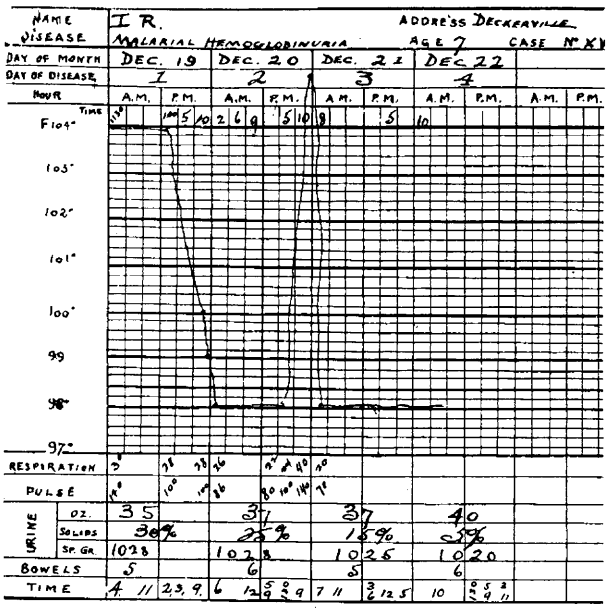
This is the first case I have ever seen before frost. Cases 11, 12 and 13 have been reported, all very much the same as this. (See Trans. Section on Materia Medica, Pharmacy and Therapeutics, Columbus meeting; *Louisville Med. and Surg. Jour.,* Aug. 1899.)

are replaced by fat drops. Necrobiotic change occupying rather extensive areas, are seen. The vessels are filled with pigmented leucocytes, dead parasites, remnants and débris, and blocks of yellowish-black pigment; Kupffer's cells and certain endothelial cells undergo multiplication by karyokinesis. Thus we have the hepatic tumor; this has a blackish, leaden appearance and is soft on section.

The splenic tumor may be merely palpable below the costal margins or it may reach below the navel and to the anterior superior spinous process of the ilium. Ordi-

narily, however, the enlargement does not assume the great proportions seen in malarial cachexia. Post-mortem, the surface of the spleen is dark, sometimes black; on section the gland tissue is also found to be dark; the parenchyma of the organ is much softened; the tarry pulp may be washed away with quite a gentle stream of water. The pigment of malaria is here found within the endothelium of the arterioles and capillaries in minute grains, often in actual blocks; we find aggregations of pigmented leucocytes, dead and breaking-down parasites forming actual thrombi and actually occluding the vessels. The spleen and bone-marrow have the distinction over all other organs of containing pigment in the cells of the parenchyma outside and away from the blood-vessels. In these latter organs pigment is contained in ordinary leucocytes, but in the splenic vein this substance is included not only in leucocytes but also in certain large white cells identical with those occurring in the spleen and evidently of splenic origin. The rôle of the spleen in infectious diseases has lately been gone over by Courmont and Duffau, Blumreich, Jacoby and Kurloff. Their deductions appear to be, that it makes no particular difference whether the spleen is present or not. Splenectomy seems to contribute to an increase in the bactericidal powers of the blood, while its antitoxin properties are unimproved. Ligation of the splenic vessels, the spleen being allowed to remain, appears to subserve the same end as does splenectomy on experimental infections. One of the striking changes in the blood after splenectomy is an increase in the number of leucocytes, especially the lymphocytes. This lymphocytosis is said to produce the protective powers of the blood seen in the cutting out of the circulation the spleen. It is explained again, that the spleen during health retains the disintegrated leucocytes and other cells. This detritus is regarded as being closely related to the alexins, and after splenectomy it accumulates in the blood and thus increases its bactericidal powers. Have we not occlusion of the splenic vessels in a hemoglobinuric spleen sufficient to act in a like manner, or in a considerable degree at least, as would a ligation of those vessels? May not this account in part for the lymphocytosis in hemoglobinuria?

The decrease in the bile pigments and the increase of the amount of bile secreted, as demonstrated by A. Pugliese, and the experiments of Tedeschi showing that the livers and bone-marrow in animals deprived of the spleen are richer in iron than the livers of normal animals, would show the close chemical relation of the two organs, and further the hypothesis of a grave occlusion of the splenic vessels in hemoglobinuria, as suggested. The condition would also contribute to the existing polycholia. The kidneys in an early stage of the disease are enlarged and congested; the tubules are blocked with hemoglobin infarcts; the cells are loaded with yellow pigment grains, and the capillaries with black malarial pigment. If the patient survives three or four weeks he is said to die of uremia. The appearances are then those of the large white kidney. The severest cases of nephritis of malarial origin are found in hemoglobinuria. The urine between the attacks may be per-



This patient had a chill, December 17. Quinin in No. 2 capsules was administered every four hours until hemoglobinuria came on.

Dec. 19: At 11.30 a. m., the patient was comparatively comfortable, and slightly drowsy. The urine was dark. A smear of blood was taken and while examining it, I was hurriedly summoned. I found the urine the color of coffee. Both this and the first specimen responded to the guaiac-turpentin test.

The first blood showed plasmodia. A second smear taken after the urine colored up showed: plasmodia, estivo-autumnal parasites in all stages of development, moderate poikilocytosis, a number of lymphocytes, leucocytes greatly increased, polymorphonuclear and mononuclear phagocytosis.

The lips and gums were pale, also the tongue, which is large and flabby, with a thick, white coat and a tinge of brown over the back part. Icterus notably mild.

At 1:30 p.m., calomel gr. x and turpentin gtt. xx—turpentin in a beaten egg—were administered, to be repeated every two hours in doses of gr. i and gtt. ii, respectively, until the urine cleared up. Quinin dihydrochlorate, gr. viiss, hypodermically, was given at 1:30, 5 and 10 p.m., adding strychnin, gr. 1/120 to each injection. At 10 p.m. the urine was still black.

Dec. 20: At 8 a.m. the urine was clearing up nicely. Calomel was replaced by sodium hyposulphite solution, gr. xx, every two hours. Beef juice was ordered—a half teaspoonful every two hours. Quinin bisulphate in hot solution was ordered—gr. x every four hours.

At 5 p.m. the dihydrochlorate, gr. viiss, was given hypodermically to avoid paroxysm. A tepid bath, containing a little sodium bicarbonate for a cleanser, followed by hot whisky and quinin, was given. Sponge and normal salt enema every four hours.

At 11:30 a.m. the urine was clear; turpentin was discontinued and at 5 p.m. the condition was practically normal.

At 10 p.m. the urine was quite dark.

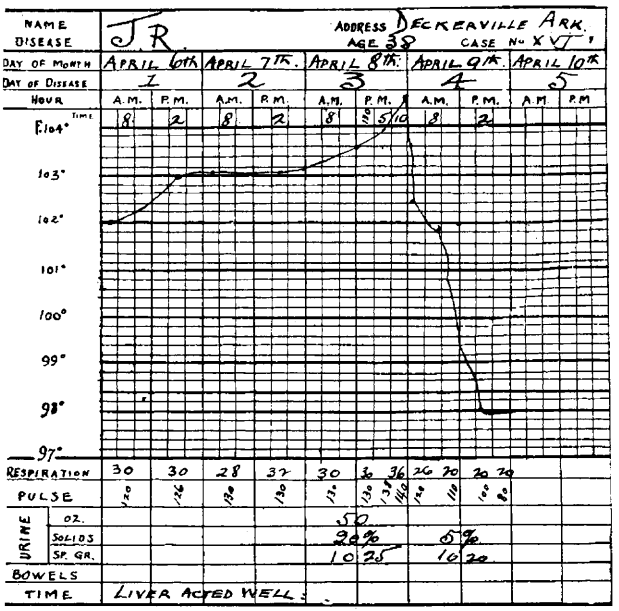
The blood contained numerous hyalin bodies, crescents and round bodies; leucocytosis was marked. There were a few lymphocytes; phagocytosis was marked, and there was an abundance of pigment and pigmented leucocytes. Quinin dihydrochlorate, gr. xv, was given in solution. This was vomited and repeated immediately and retained. At midnight the fever was subsiding rapidly, and the urine clearing up slightly. Turpentin, gtt. xv, was given at 10 p.m. and repeated in 2-drop doses every two hours until urine cleared.

Dec. 21: At 8 a.m. the urine was clear, and there was no fever. The blood contained free pigment, pigmented leucocytes and debris, also two old crescents. Quinin bisulph., gr. v, and strychnin nitrate, gr. 1/200, in solution was ordered. Sodium hyposulphite and beef-juice to be given every two hours and a bath and normal salt solution every four hours.

Dec. 22: At 8 a.m. patient was put on tonic and light diet.

It will be noted that at 5 p.m., Dec. 20, quinin, gr. viiss, was given hypodermatically, yet the paroxysm came on at 10 p.m., at which time quinin, gr. xv, in solution by the mouth was administered and vomited, repeated at once and retained, and cinchonism was profound. This may be evidence of precipitation of the alkaloid by the alkaline tissues.

fectly or practically normal, but with the attack it becomes, as before mentioned, from a poke-berry juice to a black-coffee color, somewhat turbid and smoky in appearance, and when allowed to stand precipitates an abundant chocolate-like sediment. This sediment is chiefly amorphous granular matter, disorganized corpuscles, with minute hematin crystals. Urea is generally increased; albumin is in abundance and globulin may be seen on close testing. The specific gravity ranges between 1015 and 1030, is usually acid in reaction, but oftentimes faintly alkaline, and the volume



Patient was a cigarette smoker since 10 years of age. He had had bronchitis for several days, together with some pain in the chest. There were no other signs of pneumonia, and hot applications to the chest relieved the pains. Quinin, gr. v, every hour did not control the fever, but a purgative acted well.

On the morning of the 8th the urine was of a pokeberry-juice color, and responded fully to the guaiac-turpentin test for hemoglobin. The blood showed poikilocytosis, leucocytes increased, estivo-autumnal parasites in all stages of development. The red cells crenated, tailed and buckled; phagocytosis. The urine showed a large quantity of granular and blood casts; amorphous granular matter, hematin crystals, squamous and columnar epithelium. Icterus was intense. Quinin dihydrochlorate, gr. xv, and strychnin, gr. 1/40, were given hypodermically at 8:30 a.m., and the same was ordered by the mouth at 4 and 10 p.m.

At 9 a.m. on the 9th the urine was clear and the fever rapidly abating. Quinin, gr. v, in solution was ordered for 10 a.m. and gr. x for 10 p.m. At 2 p.m. fever had disappeared.

Here quinin internally failed to control fever, which responded at once to it hypodermically. Icterus disappeared rapidly. The patient was up on the morning of the 10th for breakfast, smoked several cigarettes and went to bed at noon with pain in the chest. I was called and found what developed in a few hours into a violent pneumonia, from which he died on the 15th. There was no return of the hemoglobinuria.

may be increased. There are casts, principally dark granular ones, though hyaline casts may be found. Many of the casts are made up of hemoglobin. Rarely a few scattering blood-corpuscles are noted.

Hemoglobinuria occurs in the old residents, usually after the second or third year, rarely in new-comers, more often in males than in females. This is probably on account of the greater exposure of the male. Out of 16 cases seen by me, 13 were males and 3 females, the oldest being 38 and the youngest 5 years of age. Dr. McElroy, of Stovall, Miss., shows a record of 40 cases, of these there were 30 males and 10 females.

Hemoglobinuria is extremely rare in infants. Fish reports one case in a child of 14 months; McElroy, one at 12 months. It is said that the full-blooded negro is immune to the disease. Easman, Eyles and Quartey-Papafio have recorded hemoglobinuric attacks in native Africans; Krause, of Memphis, reports one case in a "pure negro." Of my 16 cases, 3 were very light mulattoes; of Dr. McElroy's 40 cases 13 were negroes, 2 of which he writes to me were full-blooded negroes. There are, however, observers of decided repute who maintain very stoutly that the disease does not obtain in negroes of full blood.

The parasitology of hemoglobinuria, so far as my observations go, is estivo-autumnal pure and simple; and I believe this is the consensus of opinion; there are those, on the other hand, who hold to a special parasite, and others appear to have observed a special bacillus in the blood and urine.

Paroxysmal and toxic hemoglobinemias aside from the parasitic agent are accounted for in the same manner as that of malarial origin, i. e., any agent that will set free more hemoglobin in the circulation than the liver can care for will set up a hemoglobinemia —this in turn is thrown off at the most convenient outlet by the renal epithelium—a hemoglobinuria. Paroxysmal hemoglobinuria is precipitated by exposure to cold, or to the application of cold to the hands, etc.

Toxic hemoglobinuria is said to be produced by certain drugs, among which are cited: chlorate of potassium, carbolic acid, naphthol, carbonic oxid and quinin. To all of these, save quinin, I objected in my paper on hemoglobinuria, read before the Section on Materia Medica and Therapeutics, of this Association, at Columbus, Ohio. Chlorate of potassium is being used in large doses by my confrères and myself for pytalism, as recommended by the elder Gross; hemoglobinuria does not follow, nor does any other toxic effect. Carbolic acid, because a poisonous dose produces a coagulation of albumin and such a rapid and violent inflammation of the mucous lining of the stomach, that absorption is prevented to any great extent, and death is so sudden that hemoglobinuria could hardly obtain. Naphthol: Bouchard says it would require a half-pound to produce death in a healthy man weighing 150 pounds. Carbonic oxid combines with the hemoglobin of the red cells and renders them unfit for conveying oxygen, but does not disturb the continuity of the cell—no hemoglobinemia, no hemoglobinuria.

TREATMENT.

Of my first 6 cases, 2 died, 1 of these latter never having had a movement from the bowels, despite large doses of calomel and frequent enemas. Whether this was a paresis of the bowels, due, as Dr. Jones, of Memphis, thinks, to malarial toxemia, I am not prepared to say. Calomel, turpentin, eliminants, hot applications and supportives formed the treatment. I am persuaded that this gave a good percentage of recoveries. No. 7 bade fair to recover; urine was clear for 72 hours; but the patient relapsed after gormandizing; collapse followed with suppression of urine and passing of feces; yet his kidneys partially recovered, and he became strong enough to start on a journey home, but died on the way. No. 8 got on nicely without quinin. Nos. 9 and 10 died. No. 11, a little girl of 5, had no chill in two or three months; then she had a chill at 3 a.m. I was

not called until 8 a.m., at which time she passed the first black water. Calomel, strychnin, hot applications and mustard bath were employed. Urine cleared up perceptibly and symptoms subsided, but at midnight urine darkened again, and remained so the following day, though voided in large quantities. Icterus became marked. Vomiting was distressing, and restlessness pitiable; delirium came on at 5 p.m.; another chill occurred and urine became black; there were plasmodia present. I finally gave quinin, also normal salt solution by hypodermoclysis. It was too late to be of benefit. Nos. 12, 13, 14, 15 and 16 were given the same treatment plus quinin. All recovered nicely. Nos. 12 and 13 were the same person in two separate attacks. I now depend more on quinin. Nos. 12, 13, 15 and 16 took quinin in large doses before the hemoglobinuria came on. Larger and better cinchonism, however, proved to me that quinin is not such a destroyer of red cells, and the decided phagocytosis would appear as tolerable evidence that the movement of the white cells is not very unfavorably interfered with, but that it does rapidly destroy and banish from the circulation the malarial parasite, and thus closing out the hemoglobinemia and necessarily the hemoglobinuria. Of course the eliminants throw off the toxic environment, which contribute very considerably to the hemoglobinemia. Other agents are methylene blue, nutmeg and tannic acid. The first I have used in one case, but very little of it was retained; vomiting was excited every time it was given. I regard, though, methylene blue quite favorably in cachexia.

DISCUSSION ON PAPERS OF DRS. LAZEAR, WOLDERT, CRAIG, JONES AND BURNS.*

DR. WILLIAM KRAUSS, Memphis, Tenn.—I must say that the five-minute limit cripples me completely and it is impossible to say much or do justice to the discussion of all these papers. I had desired to discuss some of the earlier papers, but since they are mostly in line with my own findings, I will leave them to take issue with my friend Dr. Burns. I feel a sense of humiliation for the writers of my adopted country, except the Johns Hopkins School, when I read the allusions, and incorrect allusions at that, to this syndrome of malarial origin—I object to its being called a symptom. It is sincerely to be hoped that they will revise that part of their books in future editions. The fatal dictum "malaria ergo quinin" has sent countless hundreds to their graves. I have attempted to harmonize existing views, not having one of my own to defend, and think I have constructed a working hypothesis of the origin of the "disease," if you please. There is not the faintest analogy between this and the hemoglobinurias due to the ingestion of drugs in non-malarial subjects. Dr. Burns has given us its symptomatology. What are the principal known facts in regard to malarial methemoglobinuria? 1. Hemolysis can not be caused by adding quinin to the blood of a hemoglobinuric, in vitro. 2. Hemolysis never occurs in acute malaria. 3. Active infection by malarial organisms bears an indifferent ratio to the severity of the attacks; indeed, it may be absent in fatal cases. 4. Quinin may, in some cases, produce hemolysis in an individual to-day and not to-morrow; while in others the hemolysis is permanent, and if quinin is persisted in it will destroy the patient. 5. Hemolysis may be present in rare cases in the absence of a quinin history. 6. We know, as a positive fact, that in all cases where the plasmodia existed early in the attack, they rapidly disappear from the peripheral circulation under the influence of the hemoglobinuria; post-mortem, the parasites are always entirely absent. 7. Occasionally we find mild cases in which the plasmodia survive the attack. 8. The constant evidence of the estivo as the only parasite. 9. The entire absence of any evidence of a specific malarial toxin.

Now, what do we find as the determining factors in hemoglobinuria? 1, persistent neglect of an old infection; 2, superimposition of an added acute infection; 3, some gross indiscretion; 4, constipation and biliary stasis resulting in the resorption of bile; 5, change to a better climate, better location, better surroundings; 6, anti-malarial treatment. Here, then, we have two opposing factors producing similar results. Noeggerath's dipping experiments with Texas fever cattle had to be abandoned because a large percentage of these developed murrain with destruction of the animals. This is quite a parallel case. We must suppose then, that, after a while, in persons living in malarial climate a symbiosis is established, which is an exact balancing of two opposing factors to the extent of producing apparent health, a disturbance of which precipitates a paroxysm; if the potential is high, quinin or some other agent will produce a grave paroxysm; if low, it may not do much or any harm.

Bignami tries to make the point that probably hemoglobinuria is produced by the precipitation of the pigment in the epithelium of the convoluted tubules, the epithelium breaking down and causing a retention of hemolytic substances in the circulation. I have here some photo-micrographs apparently refuting this idea. They show absolutely no pigment in the epithelium of the convoluted tubules; it is in a state of cloudy swelling or even further degenerated; there is stasis in the vasa recta, the glomeruli and Bellini tubules are the seat of the pigment, which is in coarse particles; there is obstruction of the loops of Henle. In none of my preparations is there anything which can be regarded as even resembling a plasmodium.

Hemoglobinuria is probably an exaggeration of the tissue reaction which ordinarily is conservative and salutary. Proof is shown in the destruction of the parasites. All this is in accord with the clinical proof. I have seen 13 consecutive recoveries under the "eliminative" treatment; no such record can be shown under the quinin therapy. (Three more cases and no deaths at time of revision of this proof.)

Active malaria is not an essential factor. The plasmodicidal effect of the hemolysis is greater than that of any amount of quinin. No death from the "disease" can be ascribed to the malaria per se. There is no logical indication for quinin except in the rare cases in which the parasites survive the attack. Quinin is dangerous during the attack, though it is the best prophylactic.

DR. T. J. HAPPEL, Trenton, Tenn.—I could not, even if I desired to, review the ground so thoroughly gone over; others are more competent to do so than I. As a general practitioner, living in a malarial section, I desire to state that we deal with malaria nine months out of each year. I shall not attempt to discuss any of the special papers, but I do want to emphasize what was pointed out by Dr. Jones, that too little attention is paid to the clinical history of the disease and we are attempting to learn too much from discussions on theory. We are gradually turning the study of disease over to the laboratories. We will soon have no real practitioners. Soon we will have men carrying away blood to the laboratory to ask the question: "What have we got?" I think we are going to extremes. We certainly have been able during the past years to diagnose malarial fever, typhoid fever, etc., and we are able to do so to-day without the microscope. There are far abler men in the profession than I who are prepared to state that the results of treatment were as good then as under the present advanced knowledge of the disease. If the gentlemen will come South at the invitation of Dr. Jones they will find a different type of mosquito, which does not convey the disease. In the South the poison hangs on every bush. Our children do not get malaria from mosquitoes, which bite them through almost the entire year.

DR. W. S. THAYER, Baltimore—Perhaps the most important point in Dr. Lazear's communication is the fact that he has brought before us easy, practical methods of making and applying Romanovsky's stain. This is a valuable clinical advance. All who use the microscope know how hard it often is

* The papers covered by this discussion, other than that of Dr. Burns, are that of Dr. Lazear, October 13, p. 917; Dr. Woldert, October 13, p. 933; Dr. Craig, November 3, p. 1139, and Dr. Jones, November 3, p. 1148.

to find small hyaline malarial parasites when but few are present. Proper investigation of the fresh blood specimen may, under these circumstances, require a great deal of time and patience, much more than is possible for the active practitioner to give. By these methods, however, it is not difficult to make a few dried cover-glass specimens at the time of the visit, and to stain them later. The results are so clear and sharply defined that one can readily detect parasites when only few are present.

I must confess that I am not satisfied with the existence of a distinct quotidian malarial parasite. It may be possible that in some instances the cycle of our estivo-autumnal parasite may be barely as short as twenty-four hours, but I do not believe that there is a distinct quotidian species. The view which we first held, based entirely on temperature charts, that paroxysms due to quotidian parasites were common with us, I have been compelled to abandon some years ago, and I am inclined to agree with Gautier and Ziemann that the estivo-autumnal parasite is one whose cycle lasts ordinarily about forty hours. In the recent publications of the Italian observers, they speak with considerable precaution with regard to the possibility of the existence of a distinct species of quotidian estivo-autumnal parasite. It seems to me that on careful analysis of these charts one observes that the paroxysms every third day begin at approximately the same hour, while those on alternate days vary slightly more. This is extremely suggestive as indicating a double infection with parasites, the cycle of which is of about forty-eight hours.

Our observations in Baltimore have led us to agree entirely with the views of Dr. Jones concerning the frequency of malarial nephritis in the negro. The vulnerability of the negro's kidneys, as well as of his lungs, appears to be especially marked. The frequency and high mortality of pneumonia in the colored race, as well as of nephritis, as compared with the white, is striking.

With regard to hemoglobinuria, I can not speak from experience. In Baltimore malarial hemoglobinuria does not occur. From a study of the literature there can be little doubt, however, that in those cases of hemoglobinuria where active malarial parasites are present, quinin does good. There is apparently also no doubt that in certain instances where the parasites are present at the onset, the mere occurrence of the paroxysm is sufficient to destroy nearly all, if not all, the organisms present. This is in all probability due to the fact that the infected corpuscles are especially vulnerable and thus prove subject to dissolution in an attack, the parasites, which are set free, being immediately destroyed. It has further been definitely shown that some attacks of hemoglobinuria occur after an acute malarial infection has entirely passed by, when no parasites are to be found in the blood.

There is no doubt that true quinin hemoglobinuria does occur, though the direct causal action of quinin is probably rarely observed. There is also some reason to believe that in individuals who have taken quinin for very long periods of time there develops sometimes a predisposition toward this manifestation. I should give quinin in any case of hemoglobinuria where active parasites were in the blood. I should abstain from giving it if they were not present.

DR. HENRY D. DIDAMA, Syracuse, N. Y.—In 1852 we had a swampy region in the neighborhood of a salt plant, and there was a good deal of ague; there were about 200 cases in one year. The treatment, at that time, was in giving calomel and jalap to get the liver in good order. I was a younger practitioner then than now, and I thought the complaint was the result of some poison. There was a quantity of mosquitoes, but I did not blame them. The ague is not there now, but the mosquitoes are there just the same.

DR. ALLEN A. WESLEY, Chicago—In reference to the excellent paper given us by Dr. Jones, of Tennessee, in which he makes the positive statement that the negro does not have chronic malaria, I beg to disagree. It was my privilege, while in Cuba, to see over 1000 cases of malaria among the negro soldiers, and I have seen chronic malaria among them. There are now in Chicago some of these soldiers who have returned from Cuba and who are suffering with malarial cachexia. Shortly before I left Chicago for this convention a former soldier came to me presenting a coated tongue and a cold, clammy skin, which had a peculiar ashen hue. He also complained of having had dysentery and an aching throughout his body and chilly sensations, which were sometimes followed by fever. I put him upon quinin and mineral acid and he improved. Further, I wish to state that while serving as surgeon in Cuba, not having any microscopic appliances in the hospital, I sent to the United States for cover-glasses, on which I made a number of blood smears, using every care in sterilization. These specimens were sent to Dr. A. J. Coey, of Chicago, who has made an especial study, both in Vienna and at Johns Hopkins, of the parasite of malaria. His report showed that the blood in many of these specimens contained crescent bodies, and evidences of malignant and chronic malaria. Thus it will be seen from the clinical picture given, and the scientific data here presented, that it is a mistake to say the negro does not have chronic malaria.

It seems to me that all subjects coming before this body should be considered of sufficient import to demand the most careful consideration of every phase thereof and to require the careful collection of all scientific data pertaining thereto before any generalizations are made, especially when those generalizations effect a whole people. I wish to enter my protest against medical men making positive statements without any, or at most, very little scientific data on which to base the same. In regard to the negro's susceptibility to kidney disease, I have only to say that in our command of over 2100 negroes we had over 1500 cases of malaria, which predisposes to kidney disease, but we had only one death directly traceable to the kidneys.

DR. N. S. DAVIS, JR., Chicago—I should like to ask Dr. Howard whether he has information about the migration of anopheles. The disease often disappears from localities and reappears in them many years later. For instance, in my boyhood days malaria was not uncommon in and about Chicago. During the first ten years of my practice there a case of ague was rarely seen, unless it was brought to the city from a considerable distance. However, when the lagoons were made in Jackson Park and the grounds were prepared for the World's Fair, and when excavation began and was in progress for the Chicago drainage canal, malaria appeared among the laborers in these localities and gradually spread, and still clings to neighboring sections of the city. The drainage of the land about Chicago undoubtedly caused the disease to disappear. What has caused it to reappear? Was it because the anopheles were given a suitable place in which to breed, or was it due to their migration, and if the latter, what laws govern their migration?

DR. L. O. HOWARD, U. S. Dept. of Agriculture—There are no evidences to show any migration to a distance.

DR. W. R. TAYLOR, Wheeling, W. Va.—There is nothing that will live eternally but truth. Upon this rock let us build. I should like to call attention to the fact that if malaria is due entirely to the bites of mosquitoes every man who goes to the Klondike would be killed in a month. There is no region on the earth where these pestiferous insects are more numerous nor their bites more venomous. Yet, malaria does not exist there. In our city of Wheeling, during the months of August and September, we sometimes have countless hosts of these insects, and plenty of the anopheles or dapple-winged variety. A large part of the city is built on an island, surrounded by the waters of the Ohio River. The island ground is low, and part of it swampy, yet malaria de novo is never seen here at the present time. After the war with Spain many soldiers came back from the South and from Cuba literally saturated with malaria, but they did not prove to be sources of infection for our citizens, although the mosquitoes were here busily engaged in carrying the infection to our unsuspecting and uninfected citizens. Wheeling Creek runs through the central part of that portion of the city which lies east of the Ohio River. In the summer months this creek is little more than an open, stinking sewer; a perfect paradise for mosquitoes, but we have absolutely no malaria. These facts may be multiplied and elaborated ad infinitum.

What inferences must we draw from these facts. Mosquitoes may carry the plasmodium malariæ from the blood of a patient afflicted with malaria to one who is not, as flies may carry the tubercle bacilli from the sputum of a tuberculous patient to other persons and deposit it on or about them; they will not all become infected, only a very small percentage of them. Mosquitoes can not develop malaria de novo. Persons who are perfectly well may go into a malarial district in the early spring—before mosquitoes are hatched and developed—and work in the ground and contract malaria without ever seeing a mosquito. Foster defines malaria as "air tainted by deleterious emanations from animal or vegetable matter, especially noxious exhalations of marshy districts, capable of causing fever or other disease." This definition is in strict accord with all of our exact knowledge of malaria.

DR. O. T. OSBORNE, New Haven, Conn.—I should like to emphasize Dr. Davis' point. I have had somewhat the same experience in New Haven that he has had in Chicago. Fifteen years ago there were a great many cases of intermittent fever. From that time until five years ago we had very little. In the center of the city there was almost none. Then they began to put up a number of large buildings, dug cellars, put in electric cars, which went all over the city; the streets were much dug up. From that time typical intermittent fever reappeared. New Haven is accustomed to a few mosquitoes; we have very little trouble with them and seldom do we find it necessary to screen against them.

DR. R. C. NEWTON, Montclair, N. J.—I should like to ask if it is true, as has been frequently asserted, that malaria does not originate in localities which are over 700 feet above the sea-level. And if this popular belief is well founded, what shall we say about the presence of the anopheles? Is it found at an altitude of 700 feet or over above the sea-level?

DR. W. S. THAYER, Baltimore—With regard to the effect of the mosquito bite one should remember that it is not the number of mosquitoes which bite one, but the kind of mosquito, and the result depends entirely on whether or not it is infected. The great mass of mosquitoes all over the country are perfectly harmless, as far as malaria goes. There is only one genus of mosquito which is capable of transferring the disease and this genus inhabits those regions which we know as malarious. Members of this genus are, however, present in other regions which may not generally be known as malarious but which may be shown to be potentially so, as was the case in the epidemic which has recently been referred to, following the advent of Italian laborers. And in such districts when one looks carefully into the history of the region he will probably find that malaria has, at one time or another, existed. There is really no evidence to show that malaria can be acquired through the gastro-intestinal tract. There is simply no positive evidence that any epidemic of malaria is due to an infected water-supply.

DR. L. O. HOWARD, U. S. Department of Agriculture—In answer to the question asked regarding the occurrence of malarial mosquitoes at an altitude above 700 feet I would state that they have been observed at an elevation of 1000 feet and over.

DR. ALBERT WOLDERT, closing the discussion—Experiments have shown that the length of time the anopheles remains infected after it has bitten a case of malarial fever is about three weeks. After this length of time has elapsed it is believed the insect can not inoculate man with malarial fever.

Referring to the subject of malarial hemoglobinuria, in my opinion the etiology is not definitely known. Many cases of hematuria of doubtful origin have been spoken of as malarial hemoglobinuria. A distinction should be made between hemoglobinuria and hematuria by the use of the microscope. At one hospital I know of, over 2000 ounces of quinin had been given to patients in which the clinical diagnosis of malarial fever had been made, without producing hemoglobinuria. Of five cases of supposed malarial hemoglobinuria all took quinin and all recovered.

DR. WILLIAM BRITT. BURNS, Deckerville, Ark.—I came to this meeting to especially make a plea for harmony in the treatment of hemoglobinuria. As my reports show—of course, 16 cases does not show a large clinical report—more than one-half the cases died under the treatment, without quinin. It is the custom for many physicians in the southern states to use no quinin in the treatment of hemoglobinuria. One year I was losing all my cases and I determined to try one case, and see if he would live if I used quinin. This patient had considerable jaundice and an enlarged spleen, a typical case, in short, wherein those, who fear quinin, would have withheld that agent. He had been given quinin sulphate in large doses by the mouth, and on the third day developed hemoglobinuria. How easy it would have been to have arrived at a quinin-hemoglobinuria. Quinin saved that man's life, I believe; I began at once, not to withdraw, but to get quinin into the circulation, giving 7½ grain doses every four hours of the hydrochlorate, intra-gluteally, and to my surprise he got well; moreover this same person was treated successfully with quinin in a second attack some months later. Ever since, I have treated every case with calomel, turpentin, hot applications and beef-juice I have also been using the normal salt solution, per rectum, which I find replenishes the blood and allays thirst. This mode of treatment, with quinin applied, in the past five cases has proved successful. I object to the term post-malaria in any form; I know of no post-malarial condition, except convalescence and sound health. I find estivo-autumnal parasites in all cases of hemoglobinuria, and knowing the great hemolysis wrought by these agents, I would doubt the wisdom of withholding the one agent which would eliminate the plasmodia from the blood. I no longer fear quinin in hemoglobinuria.

MASSAGE OF THE EYEBALL.

PRESENT STATUS AND VALUE, WITH THE CONSIDERATION OF WHAT DISEASES OF THE EYEBALL MAY BE FAVORABLY INFLUENCED BY THIS THERAPEUTIC MEASURE AND WHAT ARE THE BEST MEANS OF ITS APPLICATION.*

CASEY A. WOOD, M.D.
CHICAGO.

Massage is one of the oldest remedies known to ophthalmic surgery, and it may well be believed that a method of treatment not only in vogue thousands of years ago, but also adopted and retained by almost every school of medicine, must have special virtue.

Having used ocular massage extensively for many years, I gladly avail myself of this opportunity of exchanging with my colleagues opinions as to its efficacy.

First, I wish to speak particularly of the simple and not of the instrumental variety, such as the direct rubbing with pieces of cotton wool or some fabric mounted on a convenient shaft, the *tapottement* (or tetanization) of Maklakow, the use of sounds, with or without buttonheads, and other devices. In the main, indirect massage (Pagenstecher, Michel) with the pulp of the finger placed on the skin of the lids is to be preferred. Costomiris, formerly of Athens, who speaks enthusiastically of massage, believes that the best results are obtained by the direct rubbing of the finger tip or tips on the exposed cornea and conjunctiva. So far as the latter membrane is concerned, I believe direct massage is valuable, and in some instances decidedly preferable to the indirect method, but the stroking movements, with slight pressure on the exterior of the eyelids, are usually sufficiently efficacious, are certainly less difficult to carry out in practice, and are more readily borne by the majority of American patients.

It is necessary to say that the amount of force used, the length of each sitting, the frequency of the appli-

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