etiology of gout. In fact for ages, attention has been called to the influence of spirituous liquors on the development of this disease. Wines that contain too much alcohol, as well as strong beers, should be interdicted altogether; although Garrod has condemned cider, I do not believe that this beverage can give rise to gout; I think there may be cases in which it may be beneficial.

But if the dietary of the gouty patient needs to be carefully regulated, it is just as necessary to prescribe suitable muscular exercise of all kinds; gymnastics, fencing, pedestrianism, all should be employed, and as our immortal fabulist has said:

"Goutte bien tracassée,
Est, dit on á demi pansée."

prescribed or forbidden gouty persons. Van Helmont condemns the white and sour wines; the white wines of Bohemia and of Hungary are also forbidden. Champagne, according to Scudamore, is even more detrimental. Nevertheless, Bouchardat is of the opinion that the white and moderately sour wines, cut with the alkaline waters, are very useful in the case of polyuria.

Beer is absolutely proscribed by Garrod; Van Sweiten, however, claims that it is useful. But there is a general agreement in proscribing the alcohols, so-called, and liquors. Rabuteau has even pretended that alcohol in the blood precipitates uric acid.

[Dr. J. Mortimer Granville (*Lancet*, Aug. 16, 1884) has an interesting article on the "Mental Element in Gout," in which he sets forth the view which he says is accepted generally, now "that there is a uric acid centre, either eliminative or destructive," probably located beneath the floor of the fourth ventricle, not far from and in functional relation with the so-called diabetic and polyuric centre. An attack of gout is likely to occur in the gouty subject at either of two mental or cerebral crises. First, on the eve of a great mental effort, when the brain is charged to the highest point of tension with "nervous energy." The type of a paroxysm so occurring is likely to be "nervous," with severe neuralgic pains, if the patient be neurotic, or *visceral*, in the sense of attacking one of the large organs, if he be robust or of active habit. Second, an attack of gout is likely to occur at the end of an intellectual effort, when the centres are exhausted, and in this case it may take on the form of an epileptiform fit, or syncope, followed by more or less prolonged depression, or it may rapidly develop into a formulated arthritis of the ordinary type. In the break-down, before action, he thinks the cerebral strength itself gives way; while in the paroxysm, after exertion, the attack occurs because the inhibitory control of the cerebral centres over the spinal medullary and organic centres is suddenly suspended. The former class of cases is the most serious. and attacks of this kind go far to prove the existence of a neurosis, for which the patient will require to be specially treated. The second class is one in which the malady proper rather than the patient demands the practitioner's greatest attention. His therapeutic suggestion is that the uric acid can only be got rid of by a process of oxygenation, and that it must pass off by the kidneys, and not by the bowels. Purgatives in gout are always harmful in direct proportion, as they promote discharge of fluid from the intestinal surface, and leave the urine concentrated so that the uric acid, which requires a very large amount of fluid for its elimination, blocks the tubes of the kidneys in the form of crystals. One reason for the specially frequent occurrence of gout in warm weather is the loss of fluid by perspiration, and the consequent concentration of the kidney excretion. If uric acid can crystallize, it will. Below is Granville's famous anti-gouty mixture, which he says always immediately relieves pain, reduces swelling and raises the proportion of urea in the urine, as estimated by the hydrobromate test, from 50 to 100 per cent. in a few hours. He never starves patients or gives colchicum, believing nitrogenous food to be necessary, a vigorously trophic state being needful to facilitate the oxidation of uric acid. ℞ Ammon. chlorid, ʒ iv. Pot. chlor., ʒ ij. Glycerine, ʒ xij. Tinct. iod., ʒ ij. Aquam ad, ℥ xij. M. Sig. Take two tablespoonfuls every third, fourth or sixth hour. Granville's theory, in the present state of science, can be looked on as only an ingenious and plausible speculation, provisionally, perhaps deserving acceptance.—TRANSLATOR.]

ON THE TREATMENT OF DIABETES.

SUMMARY.—Concerning Diabetes—Its Frequency—Its Pathogeny—Physiological Glycæmia—Theory of Diabetes—Alimentary Theory—Nervous Theory—Theory of Disturbance of Nutrition—Glycosuric Urine—Tests for Glycose—Heller's Process—Boetger's Process—Trommer's Process—Dosage of Glycose—Duhomme's Method—Prognosis of Diabetes—The Diabetes of Fatty People—The Diabetes of the Lean—Grave Diabetes—Diabetes of Medium Intensity—The Mild Form—Hygiene of the Diabetic—Alimentary Hygiene—Bases of the Alimentation of the Diabetic—Regime of Cantani—Regime of Bouchardat—Regime of Seegen—Gluten Bread—Soups—Legumes—Fruits—Pastries—Alcoholic Beverages—Beverages in General—Glycerine--Resumé of the Alimentary Hygiene—Exercise—Results of Treatment—Pharmaceutical Treatment—Anti-fermentescible Medicaments—Lactic Acid—Narcotics—Valerian—Ergot of Rye—Iodine and the Iodides—Alkalies—Their Action in Glycosuria—Arsenic—Bromide of Potassium—Thermal Treatment of Diabetes—Electricity—Hydrotherapy—Local Treatment of Diabetes.

GENTLEMEN: Diabetes[1] is a very common affection, which you will often be called upon to treat when you shall have entered upon your practice; I pro-

[1] Cantani has given a full history of diabetes, which he divides into four periods:

The first, which commences with Aretæus and Galen, and during which the presence of sugar in the diabetic urine was recognized though ill-understood.

The second, which commences with Willis, and in which the symptomatology of diabetes was accurately established.

The third, which is associated with the name of Rollo, who, more than any of his predecessors, pursued a practical end while discussing, with more method than they, certain theories which were largely adopted.

The fourth, which commences with Claude Bernard, and in which diabetes is studied by the aid of the experimental method from the standpoint of pathogeny and therapeutics at the same time.

Diabetes was known from the most remote antiquity by the physicians of India, where it was described under the name of *sweet urine*, or urine of honey. In two books translated from the Sanskrit, diabetes is called *madume'hé*, which means *urine of honey*.

The ancients knew little about diabetes; at the same time, Cornelius Celsus mentions it, and Aretæus gave it the name of *diabetes*, from the Greek word διαβαινω, implying that the sweet drinks of persons so affected *passed through* the body unchanged.

Galen also considered diabetes as a disease of the kidneys attended with elimination of sweet beverages unaltered, and these ideas were adopted by Willis. Moreover, Vittorio Trincavella, in support of these Galenic notions, cites a case where the urine of a diabetic patient had the same taste as the drinks which he imbibed. During this period Paracelsus alone took a stand against this theory of Galen, and affirmed that the cause of diabetes was in the blood, and not in the kidneys.

In 1675 Willis detected sugar, or rather honey, in the urine, and thenceforth the symptoms of this disease came to be better known. Sydenham thought that it was a disease of malassimilation; and Dobson proved, by the fermentation test, the presence of sugar not only in the urine, but in the blood. Lastly, Rollo inaugurated the dietetic treatment of diabetes, which hygienic regime has been crowned by the labors of Bouchardat dating from 1841; and within the present epoch Claude Bernard has given to the world his valuable discoveries concerning glycæmia and the glycogenic functions of the liver. (*a*)

(*a*) Cantani on Diabetes. Paris, 1876.

pose, therefore, to devote a lecture to the therapeutics of this disease. So frequent, in fact, is this complaint that Bouchardat affirms that out of every twenty men between forty and sixty years of age, and pertaining to the wealthier classes, you are sure to meet with at least one diabetic patient.[1]

Whence comes this really large nnmber of glycosuric patients? It results, first of all, from the fact that we are better acquainted with the affection, and, moreover, with the conditions of modern life, which, in exciting unduly the digestive functions and those of the cerebro-spinal axis, predispose notably to this disease. There is, then, a preponderating interest in knowing the therapeutic rules which ought to be observed in such cases, especially since, when these rules are followed, they cause disappearance of sugar from the urine, and all the troubles which rise from glycosuria. But before entering upon the main part of my subject, there are two preliminary questions which must be decided: the pathogeny of diabetes, and the tests which determine the presence of sugar in the urine.

Pathogeny plays an important part in the therapeutics of diabetes; it constitutes ætiological therapeutics, and we cannot discuss scientifically the influence of medicaments and medications till we have examined the mechanism of glycosuria. Thanks to the brilliant researches of Claude Bernard—researches which constitute, perhaps, his best title to glory—we know to-day what is the origin of the sugar which is found in the normal state in the blood of animals.[2]

[1] Bouchardat affirms that in every twenty men from forty to sixty years of age, belonging to legislative assemblies, the great learned societies, elevated positions in commerce, finance, and even in the army, you are sure to find one glycosuric patient. As for the frequency according to age, he adds that glycosuria is the more formidable the younger the subject, and that the epoch when it is most often observed is between the ages of forty and sixty years. Griesenger's statistics give the greatest frequency between the ages of twenty and forty. This is perhaps true as far as hospital practice is concerned, but it does not represent the frequency of this disease in private practice, and especially among persons in easy circumstances. Andral's table, which concerns 84 cases of diabetes, gives the following figures:

AGE.	CASES.
From 0 to 5 years	2
" 10 " 20 "	3
" 20 " 30 "	12
" 30 " 40 "	20
" 40 " 50 "	20
" 50 " 60 "	13
" 60 " 70 "	12
" 70 " 80 "	2 (*a*)

[2] Claude Bernard has dwelt particularly on the origin of the glucose which is found in the blood. This glycæmia comes from two sources—from alimentation, and from glycogen formed in the liver.

The glucose furnished by food is a product of the digestion of amylaceous matters by saliva and the pancreatic juice, and also of saccharine articles of diet which are transformed from saccharose into glucose by the action of the intestinal juice; this juice contains a ferment, which Claude Bernard calls *inversive ferment*. The rôle of the liver in this case is

(*a*) Bouchardat, On Glycosuria and Saccharine Diabetes.

This physiological glycæmia has two origins. Either it is a fact of alimentation—the feculents and cane sugar being transformed into glucose by the intestinal juice, the salivary disatase and by the pancreatic juice, constitute the first of these sources—or else it is a product of the glycogenic functions of the liver. Claude Bernard has, in fact, shown us that there exists in the liver an animal amyloid principle, glycogen,[3] and that this starchy principle, under the influence of an hepatic ferment, furnishes glucose. Notwithstanding the vehement attacks which Pavy[4] and Lussana have made upon the doctrine of the normal glycogenic action of the liver, pretending that this sugar formation was simply a pathological or post-mortem phenomenon, the views of Claude Bernard are none the less admitted by the majority of physicians.

Rouget, on his part, generalizing the glycogenic functions, has strongly maintained that all parts of the body, and particularly the muscles, possess glycogenic properties. But we are not obliged to enter into the discussion of all these objections; it suffices us to know that glycosuria, or the passage of sugar in the urine, is the exaggeration of a normal phenomenon, physiological glycæmia, that is to say, the presence of glucose in the blood. The conditions which, exaggerating this physiological glycæmia, transform it into a persistent glycosuria, are numerous; and, according as they have been exclusively adopted to explain the mechanism of diabetes, a great many theories have been proposed.[5]

said to be that of a reserve depot for this alimentary sugar, and to supply it to the blood in proportion as it is required. The glycogen or animal starch (which forms a singular explosive substance with nitric acid, takes a violet color with iodine, and passes to the state of dextrine) in presence of a hepatic ferment is transformed into glucose.

Claude Bernard insists that the formation of sugar in the liver is a normal fact. The liver, then, is the seat of two orders of phenomena—phenomena of assimilation, and phenomena of disassimilation. The first correspond to the formation of glycogen, the latter to its transformation into dextrine and glucose. The more active the life, the more pronounced the phenomena of disassimilation. (*a*)

[3] Glycogen is a hydrocarbonaceous substance isomeric with starch; has for formula $C^6H^{10}O^5$; dissolves in water forming a milky opalescent liquid with right-handed polarization. Under the influence of dilute mineral acids, saliva, pancreatic juice, blood serum, and of hepatic extract (prepared cold), glycogen is transformed first into a variety of dextrine, then into glucose. This transformation demands for its production a temperature of 30° C. Cold nitric acid transforms it into xyloidin, and warm nitric acid into oxalic acid.

[4] Pavy maintains that the sugar produced by the liver results from a cadaveric transformation, or from pathological conditions. These pathological conditions have their source in the non-fixation by the liver of an excessive quantity of sugar derived from food, and we ought to distinguish here the amylaceous aliments and the azotized aliments. As for the former, we are concerned with the product of an incomplete digestion; as for the latter (the azotized aliments), three conditions may aid their transformation into sugar: (1) general venous stasis, from exaggerated muscular movements, from asphyxia, etc.; (2) troubles in the portal circulation; (3) section of the filaments of the great sympathetic, disturbing the hepatic circulation. (*b*)

[5] The number of theories put forth as to the cause of diabetes is considerable. As

(*a*) Claude Bernard on Diabetes and Glycogenesis, in Revue des Cours Scientifiques, 1873.

(*b*) Pavy on Diabetes, London, 1862. 1868. Also lectures in the Lancet.

From the exclusive point of view on which I am placed, that is, the standpoint of treatment, all these theories may be referred to three heads: the hepatico-intestinal or alimentary theory; the nervous theory; and, lastly, the theory of nutritive disturbance.

According to the first theory, the glycosuria results either from the too great abundance of saccharine and starchy aliments, or from an excess of activity of the digestive ferments, or from exaggerated action of the functions of the liver. In the normal and physiological state, the glucose which has been formed in the intestinal tube passes into the circulation, and the liver becomes the regulator of this normal glycæmia; when the glucose is in too great quantity, it stores it away; when deficient, it furnishes this principle to the economy, thanks to the glycogen which its cells contain. But let some circumstance arise to trouble this harmony,[6] and we pass from the physiological to the pathological state, and glycosuria appears. This glycosuria then entails a series of modifications, more or less grave, in the economy, and we soon have all the symptoms of the confirmed disease. This theory is one of the most seductive,

many as fifty have been designated, but all may be referred to five principal theories, which are the following:

I. The gastro-intestinal theory.
II. The hepatic theory.
III. The nervous theory.
IV. Theory based on disturbance of nutrition.
V. Pancreatic theory.

I. *The Gastro-intestinal Theory.* This is the theory of Bouchardat. It is divided into two parts, the alimentary theory and the digestive theory. In the first, the sugar of diabetic patients comes from a dietary overcharged with feculent and saccharine matters; in the second, the habitual action of the ferments of the digestive tube is too energetic.

II. *Hepatic Theory.* This theory is supported by Claude Bernard. The cause of diabetes consists in a circulatory trouble, in a secretory trouble, or in a simple exaggeration of the function of the liver. It is supported by clinical observations in which diabetes has been observed to arise from hepatic derangements, such as congestions, or even cirrhosis.

III. *The Nervous Theory.* This theory is based on physiological, anatomical, ætiological phenomena. Patho-anatomical observations have found, in diabetic patients, lesions of the floor of the fourth ventricle, congestion, ramollissement, sclerosis of the spinal cord and of the brain, alterations more or less profound of the great sympathetic. From an ætiological standpoint, contusions of the encephalon and excesses in the functionating of the nervous system have produced diabetes.

IV. *Theory of Nutritive Troubles.* This is the theory which has recently received the support of Bouchard. The glycæmia of diabetes depends on a fault in the consumption of sugar by the tissues, due to a diminution of their nutritive activity. The cause of this retardation is a vicious habit, congenital or acquired. Gout and a sedentary life are the most important factors in diabetes. Jaccoud also admits that the cause of this disease is an alteration of nutrition, over-production or lessened destruction of sugar.

V. *Pancreatic Theory.* This theory is based exclusively on pathological anatomy. Lancereaux is its chief supporter. Diabetes is due to lesions of the pancreas, such as atrophy, fatty degeneration, cancer, obliteration of the pancreatic duct.

[6] Colrat and Couturier, taking as their basis certain experiments of Claude Bernard—who showed that when the portal vein is tied the glucose passes directly into the blood, and produces an alimentary glycosuria—have noted that, in animals affected with total or partial obstruction of the portal vein, from any cause whatever, glycosuria always exists.

and has for its consequence therapeutic applications which dominate in the treatment of diabetes. I refer to the alimentary regime. My venerated master, Bouchardat, has won the claim to public gratitude by founding, on this theory, that admirable hygienic treatment which enables us, through its rigorous application alone, to cause the sugar to disappear from the urine of diabetic patients, at least in the great majority of cases; and when this hygienic treatment fails, be assured that the disease will almost invariably resist all other therapeutic means.

The nervous theory is also based on experimentation,[1] on clinical experience, and on the results of therapeutics. We know, in fact, since the celebrated experimentation of Claude Bernard, that we can in animals cause a temporary glycosuria by irritating or wounding the rachidian bulb.

Schiff, by traumatic lesions of the entire cerebro-spinal axis, and even of the sciatic nerve, Pavy, Eckhard, Cyon, Aladoff, by traumatisms of the ganglia and filaments of the great sympathetic, have arrived at the same result, and have in this way determined a glycosuria equally transient.

I insist on the word *transient*, for it shows us that, unable to produce true diabetes in animals, we cannot practise on this disease experimental therapeutics, for the glycosuria which results from our experiments gets well of itself in the course of several days.

In support of the nervous theory of diabetes, clinical medicine furnishes a great number of observations where we see violent blows on the head, or great cerebral excitation, the consequence of too prolonged intellectual labors, painful emotions, chagrin and disappointment, become the starting-point of diabetes. This it is which explains why this disease is so common among certain classes of the population, such as literary men and persons devoted to scientific and academical pursuits. You will not, then, be astonished to see a considerable number of authorities claiming that diabetes is always of nervous origin.

As for that theory which refers diabetes to disorders of nutrition, it has been quite recently defended, and with considerable success, by Professor Bouchard,[2] who has classed diabetes with affections due to retardation of

[1] Claude Bernard showed, in 1849, that pricking or puncture of the fourth ventricle below the origin of the vagi causes glycosuria. Section of the splanchnics after the puncture does not modify the glycosuria, but it prevents it when made before the puncture.

Schiff produced glycosuria by section of the optic thalami, crura cerebri, pons, middle and posterior peduncles of the cerebellum, also by section of the spinal cord on a level with the second dorsal vertebra, by extensive lesions of the anterior and posterior columns, and even by section of the great sciatic nerve. Richter has shown that lesions of the sympathetic have the same effect; Pavy, that section of the superior cervical ganglion occasions glycosuria. Eckhard, Cyon and Aladoff have attained similar results with sections of the inferior cervical and superior thoracic ganglia. (*a*)

[2] Bouchard shows the difference which exists between arterial and venous blood *à propos* of sugar. In animals this is 40 centigrammes; that is, one kilogramme of arterial blood in becoming venous blood loses 40 centigrammes of sugar. When you take into

(*a*) Schiff, Jour. d'Anat et de Physiol., 1866. Cl. Bernard, Arch. gen. de Mec., 1849. Bouchard, Maladies par Relentissement de la Nutrition, Paris, 1882.

nutrition. Here also we can deduce facts in support of this theory from physiological and clinical experimentation. The glycogenic function is not, in fact, exclusively a function of the liver; all the tissues participate in it. Have we not seen Rouget demonstrate this power of sugar-production in all organs of the fœtus? Have we not seen Schiele detect glycogen in stratified pavement epithelium? In fine, do not the muscles themselves participate in this glycogenic function, as Boehm[3] has pointed out? We can, then, say, with Bouchardat, Jaccoud and Bouchard, that every circumstance which troubles the inmost processes of assimilation and of disassimilation of all the tissues may be a cause of diabetes.

One other consequence of this theory is to call attention to the elimination of urea in diabetic patients, and to cause this azoturia to play a preponderant part as Lecorché has done.

Ought we to take sides with one or the other of these theories, and base exclusively on any one of them our therapeutic endeavors? By no means; and we ought instead to draw from all them the elements of our treatment. But I must, before approaching this main subject of my lecture, set forth the clinical means which you ought to make use of in order to determine the presence of sugar in the urine, and the quantity of this abnormal ingredient.

You should, in fact, be aware that we cannot judge of the effects of our treatment but by a daily examination of the urine; our prognosis, moreover, is based on such examination; you should then perfectly understand the modes of testing urine for glucose.

Glycosuric urine is generally abundant in quantity, of a high density, which sometimes attains even 1050; it is frothy, stains the clothing, and has the curious property of attracting flies.

To detect glucose in the urine we avail ourselves of the curious oxidizing and reducing property of this substance in presence of alkaline agents, and without stopping to consider the numerous processes which have been proposed

consideration the total quantity of blood which is transformed into venous blood, it is susceptible of demonstration that a man loses in a day, at the least, 1,850 grammes of sugar; now one gramme of sugar demands, for conversion into water and carbonic acid, 1 gr., 066 of oxygen, and as a man in health never consumes more than 850 grammes of oxygen a day, this oxygen is incapable of burning all the sugar; there remains, then somewhat more than 1,000 grammes of sugar which is not consumed by oxygen, but which is destroyed by molecular metamorphoses of the entire organism. So, according to Bouchard, the conditions which give rise to hyperglycæmia may be referred to the following heads: (1) everything which prevents the sugar and starch of food ingested from fixing itself in the liver in the state of glycogen; (2) everything which increases the formation of sugar in the liver; (3) everything which interferes with the destructive metamorphosis of sugar in the economy or its fixation in the tissues. (*a*)

[3] According to Boehm, the muscles behave with regard to glycogen like the liver; that is to say, glycogen augments in the muscular tissue after meals, and is destroyed in muscular work.

(*a*) Bouchard, Maladies par Ralentissement de la Nutrition. Paris, 1882.

by Krause,[1] Luton, Muller,[2] Maumené,[3] Neubauer, and Vogel,[4] will mention only those of Heller, Boettger, and Trommer.

The first of these processes is based on the oxidation of glucose by potassa, which by heat generates glucic and melassic acids which give to the liquid under examination a more or less black color according to the quantity of sugar in solution. Bouchardat has substituted for the potassa a chemical which is much more easy obtainable, slaked lime or milk of lime.[5] This test, which is a good one when the urine has a large amount of sugar, loses its value when the quantity of glucose is small; then there is liability to several sources of error; the coloring matter of the urine, for instance, may turn brown under the agency of alkalies. Moreover, albumen and mucus may give a dark discoloration, and even an impure preparation of potassa may give the same color.

The process of Boettger is altogether different; it consists in heating urine supposed to contain glucose with a mixture of subnitrate of bismuth and carbonate of soda; in this white liquid thus formed there is soon seen a black precipitate of oxide of bismuth.[6] Although this test has been modified by my master, Behier, and more recently by Primavera, it is subject to numerous fallacies, and the reduction of bismuth may be accomplished by substances other than glucose. Attfield affirms that almost any kind of urine will effect the reduction of bismuth. Therefore this test is inferior to those which have for basis the cupro-alkaline liquors.

Trommer was the first to recommend testing for sugar in urine by utilizing the reducing properties of glucose on salts of copper, and it is upon this

[1] Krause and Luton employ the same test as for the detection of alcohol, namely the solution of bichromate of potash in sulphuric acid; this solution, of an intense red color, becomes emerald green in the presence of sugar.

[2] Muller uses the following test: To the urine heated over a spirit lamp he adds a solution of indigo-carmine made alkaline by bicarbonate of soda. If glucose be present this mixture passes from blue to green, then red, purple, and finally yellow. Jaccoud recommends this process.

[3] In Maumené's process, strips of woolen cloth are soaked in a solution of perchloride of tin; then moistened with the urine to be examined and heated over a spirit lamp. If sugar be present the strips are blackened.

[4] This test consists in adding to urine supposed to contain sugar, a few drops of solution of ammonio-nitrate of silver; if glucose be present there is a black precipitate of reduced silver.

[5] Saccharine urine when mixed with an equal volume of liquor potassa and heated, turns yellow, and, if sugar be abundant, brown; a molasses-like odor is at the same time observable.

[6] Boettger's test, which is much in use in this country, is practiced in the following manner: A few cubic centimetres of the urine are mixed in a test tube with an equal volume of solution of sodium carbonate (one part crystallized carbonate and three parts water) a few granules of bismuth subnitrate are added, and the mixture boiled for some time. If sugar be present the bismuth turns brown or black by reduction to elementary bismuth. (Witthaus' General Medical Chemistry.) Witthaus affirms that no other normal constituent of the urine reacts with this test, but there may be some substance present which, by giving up sulphur, may cause the formation of a black sulphide of bismuth.—TRANS.]

basis that a number of test liquids have been devised, Barreswil's and Fehling's solutions for instance.[1]

Fehling's solution is the most used at the present day; the reaction is very pronounced, and it suffices to heat in a test tube a mixture of glycosuric urine with one of those cupro-alkaline solutions to see the liquid change from a beautiful blue to a pale yellow, then a lively red, in consequence of the precipitation of the oxide of copper, which gradually settles down to the lower part of the test tube.

At the same time with this method, which is much the most certain, all chances of error are not avoided. Uric acid, urinary pigment, tyrosin, may cause the precipitation of oxide of copper, but this precipitation is much less clear and pronounced than that with glucose. Moreover, albumen prevents this reaction from taking place, therefore it will be necessary to have care when you are examining urine that contains both albumen and sugar (which frequently happens) first to precipitate and filter out the albumen before proceeding to test for sugar.

The employment of the cupro-sodic liquors enables us not only to recognize the presence of sugar, but also to determine the quantity, and this is a matter of great importance. I shall not here speak of the polarimetric processes while admitting that they are the most sure and precise, and that one ought always to have recourse to them when desirous of attaining mathematical exactness in the dosage of glucose, but these are methods of the laboratory, and few physicians can have at their disposal a saccharimeter, whether it be that of Soleil, or the one with a *penumbra*, or the diabetometer of Yvon. I shall only mention the clinical processes which are quite sufficient in practice.

Of all the clinical processes the most simple, the most ready, and the most

[1] There are several formulæ for cupro-potassic or cupro-sodic solutions. These are the principal:

SOLUTION OF BARRESWIL.

℞.	Carbonate of soda	4 grammes.
	Cream of tartar	5 grammes.
	Caustic potash	4 grammes.
	Water	40 grammes. M.
Add.	Sulphate of copper	3 grammes.
	Water	25 grammes. M.

FEHLING'S SOLUTION.

Take the pure	Sulphate of copper	4 grammes.
	Distilled water	16 grammes.
	Caustic soda	13 grammes.
	Neutral tartrate of potas	16 grammes.
	Distilled water	60 grammes. M.

Trommer's test, the first in use, is employed as follows: Take a sufficient quantity of liquor potassæ and a solution of sulphate of copper. Into a certain amount of urine in a test tube add an equal quantity of liquor potassæ and two or three drops of the solution of cupric sulphate. If the urine contains sugar the blue mixture with hydrate of copper clears up by agitation, and if the tube is heated a precipitate of reddish cupric oxide is formed.

economical is that of Duhomme,[1] which we use every day in the hospital and in private practice, and every physician, it might almost be said every diabetic patient, ought to have one of these little instruments for measuring the amount of sugar in the urine.

It is composed of a little box which has in its interior some test tubes, a spirit lamp, two vials, the one containing a little liquor sodæ, the other Fehling's solution, and finally, two dropping tubes.[2] These two dropping tubes are graduated, the one which is reserved for urine by a mark which indicates a cubic centimetre, the other, which is destined to contain Fehling's solution, by a mark which measures just two cubic centimetres. You begin by taking up one cubic centimetre of urine of which you take care first to count the number of drops by pressure on the rubber ball. Then with the other dropper you aspirate two cubic centimetres of Fehling's solution. This you place in a test tube, and add the same quantity of solution of soda. You then heat the mixture, and after having taken up some of the urine of which you have just counted the drops, you let it fall, drop after drop, into your test liquid, heating the latter from time to time till the entire mixture takes on a reddish color. You have counted the number of drops necessary to obtain this reaction, and you have only to refer to the table herewith given (see adjoining page) to know the quantity of sugar contained in a litre of the urine. This table contains in one column the number of drops present in one cubic centimetre of urine, and in another the number of drops made use of in order to get the characteristic color.

As this method of analysis is very rapid it is desirable, before taking a definite figure, to repeat the process three or four times, in order to know exactly the number of drops necessary to obtain the red color in the mixture.

It may be necessary when the percentage of sugar is very large to dilute the urine with an equal quantity of water, which you can easily do by means of your dropper, and in estimating the result you have simply to double the figure obtained. Duhomme has, moreover, given a method for utilizing his saccharimeter for the detection of very feeble quantities of sugar. It suffices in these cases to employ only one cubic centimetre of Fehling's solution, and to count the number of drops necessary to effect the disappearance of the green color you can make use of the same table, but you must carry the decimal point one line toward the left.

With this analytical process you can sufficiently estimate the effects of your treatment. You will also have a basis for your prognosis. You are aware that there are two kinds of diabetes. The one is the mild form, *diabetes benignus*, the diabetes of fat people, an affection which permits a person to live a good many

[1] Duhomme, Clinical Saccharimetry, Bull. de Ther., t. lxxxviii, 1875, pp. 163, 214, and 261.

[[2] The dropping tubes graduated to indicate cubic centimetres can easily be obtained of instrument makers, Codman and Shurtleff of Boston, for instance.

The method of Duhomme is so handy and simple that it ought to come into general use; it is, in fact, in general use on the Continent. Besides the dropping tubes and the printed table all that is required is the ordinary cupro-sodic testing solutions.—TRANS.]

TABLE GIVING THE RESULTS OF THE RAPID ANALYSIS OF SUGAR BY THE METHOD OF DUHOMME WITH THE GRADUATED DROPPING TUBE OF LIMOUSIN.

Number of Drops contained in one Cubic Centimetre of Urine Examined.	Number of Drops Employed.																							
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.	Gr.
XVIII.	180	90	60.00	45.00	36.00	30.00	25.71	22.50	20.00	18.00	16.36	15.00	13.85	12.85	12.00	11.25	10.59	10.00	9.47	9.00	8.57	8.18	7.83	7.50
XIX.	190	95	63.33	47.50	38.00	31.67	27.14	23.75	21.11	19.00	17.27	15.83	14.61	13.57	12.67	11.87	11.18	10.55	10.00	9.50	9.05	8.64	8.26	7.92
XX.	200	100	66.67	50.00	40.00	33.33	28.57	25.00	22.22	20.00	18.18	16.67	15.38	14.28	13.33	12.50	11.76	11.11	10.53	10.00	9.52	9.09	8.69	8.33
XXI.	210	105	70.00	52.50	42.00	35.00	30.00	26.25	23.33	21.00	19.09	17.50	16.15	15.00	14.00	13.12	12.35	11.67	11.05	10.50	10.00	9.55	9.13	8.75
XXII.	220	110	73.33	55.00	44.00	36.67	31.43	27.50	24.45	22.00	10.20	18.33	16.92	15.71	14.67	13.75	12.94	12.22	11.58	11.00	10.48	10.00	9.56	9.17
XXIII.	230	115	76.67	57.50	46.00	38.33	32.86	28.75	25.55	23.00	20.91	19.17	17.69	16.43	15.33	14.37	13.53	12.78	12.10	11.50	10.95	10.45	10.00	9.58
XXIV.	240	120	80.00	60.00	48.00	40.00	34.28	30.00	26.66	24.00	21.82	20.00	18.46	17.14	16.00	15.00	14.12	13.33	12.63	12.00	10.43	10.91	10.43	10.00

Figures indicating in grammes the quantity of sugar contained in each litre.

EXPLANATION.—The figures in the first horizontal line correspond to the number of drops employed. The Roman numerals in the first vertical column correspond to the number of drops in a cubic centimetre of the urine under examination. The table reads like the table of Pythagoras. For example, suppose that the urine which you are examining gives 22 drops to the cubic centimetre, and 11 drops are required to decolorize the cupro-sodic solution, you take the figure 11 in the first horizontal line, corresponding to the number of drops employed, and you glance down the vertical column till you come to the point of intersection of the horizontal line which begins with the Roman numerals XXII, and you find the number 20, which indicates that the urine you are testing contains 20 grammes of glucose per litre.

years. The other is grave diabetes, the diabetes of lean people, which disturbs profoundly the nutrition of the individual, and determines in quite a short space of time cutaneous, pulmonary, or other complications which rapidly prove fatal.

There has been much discussion as to whether we ought to give the name diabetes to both these affections, some maintaining that the diabetes of the corpulent is nothing but a symptomatic glycosuria, and reserving the name diabetes to the more malignant disorder of lean people; others hold that the two affections are the same, but of different degrees of intensity.

Without entering into the discussion I can affirm that from a therapeutic point of view the examination of the urine will enable you to class your patients in three principal groups: the light forms of diabetes, those of medium intensity, the grave forms. Note first of all that the quantity of sugar which you find is no criterion for prognosis; you may find as much as one hundred grammes per litre, and at the same time have a very mild case. A much more important indication is the persistence of a certain quantity of sugar in the urine in spite of the most rigid dietetic règime.

When, after having made your patient follow scrupulously a rigorous alimentary regimen, you discover that the figure of sugar in the urine keeps at from thirty to fifty grammes per litre, be persuaded that your case is a grave one, and that your pharmaceutical measures, however judicious and appropriate, will not stay the progressive decadence of the organism and the death of your patient. Generally these patients are thin, debilitated, presenting pulmonary complications of a tuberculous nature, and their forces are rapidly exhausted. I have often been called during my medical career to treat such diabetic patients, and despite all my endeavors have not been able for an instant to interrupt the steady decline of the organism.

When, on the other hand, the quantity of sugar, owing to your alimentary hygiene, has fallen to nine or ten grammes a day, you have a case of diabetes of medium intensity. These are the patients who may live many years, but in whom there supervene, when the digestive functions become enfeebled, pulmonary complications, a peculiar comatose state, or it may be cerebral ramollissement, an accident so frequent in those whom Bouchardat calls *petits diabétiques* (that is, persons slightly diabetic). These diabetic patients are amenable to a treatment which opposes to a certain extent the production of such accidents.

In the third group are cases of diabetes of feeble intensity. Here we witness the triumph of alimentary hygiene, for it alone effects very rapidly and in a few days the disappearance of the glucose from the urine, however great the previous quantity. How often have I seen such obese patients, generally arthritic and gouty, who had been voiding two hundred to three hundred grammes of sugar per day, and in whom the règime of Bouchardat had enabled us in a few days to bring down the figure to zero. Nevertheless in these patients the sugar reappears whenever they commit errors in diet.

You see, then, that this dosage of sugar, or, in other words, the determination of the quantity of sugar in urine, is a matter of capital importance to your patients both from the point of view of therapeutics and of prognosis, and I