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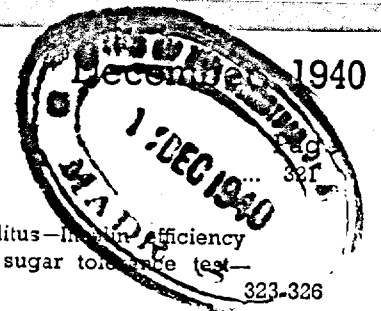
Journal of the All-India Medical Licentiates' Association

(With which THE AGRA MEDICAL CLUB JOURNAL is incorporated)

DIABETES MELLITUS

Vol. 34, No. 12

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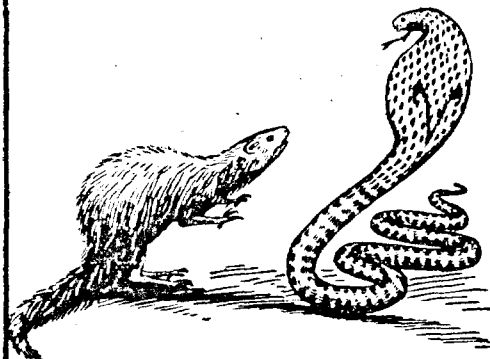
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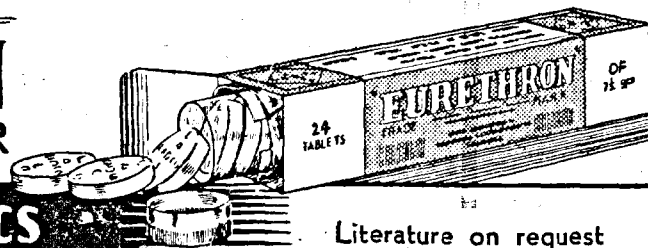
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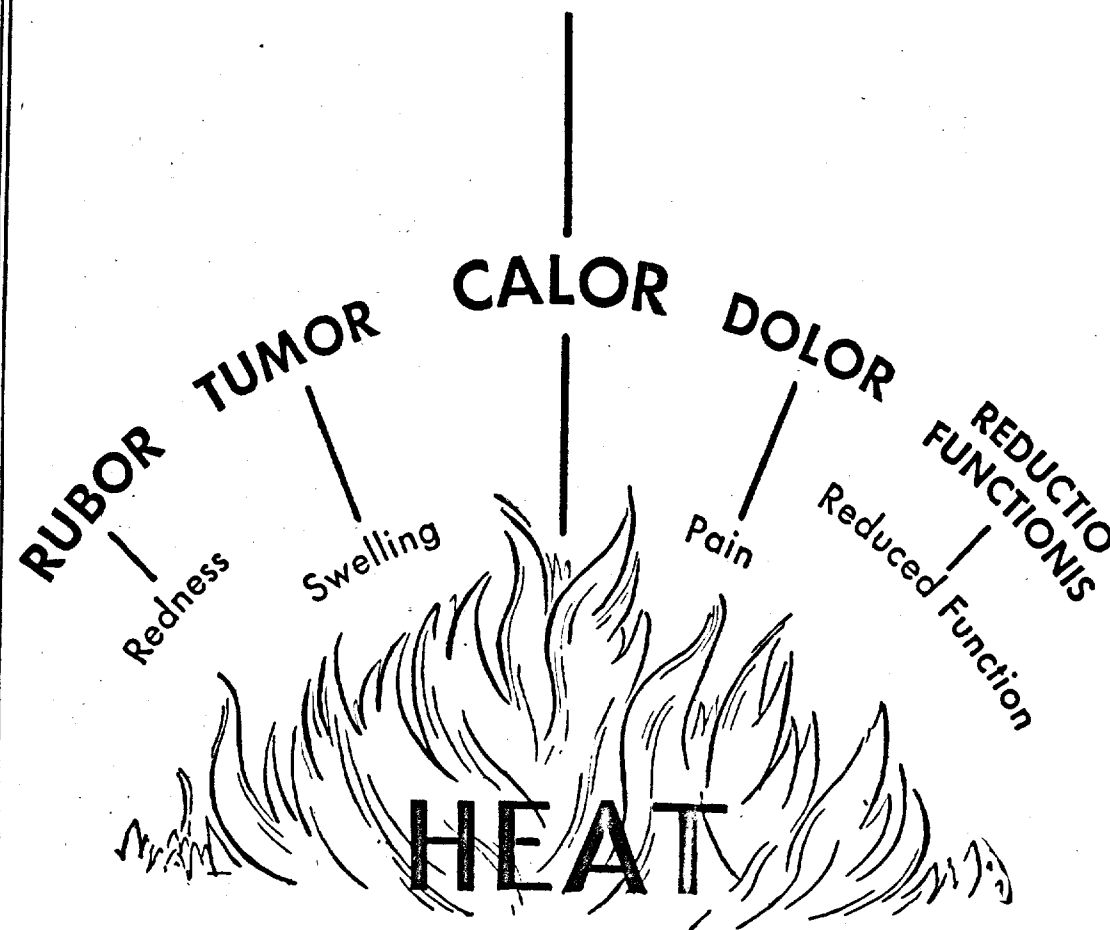
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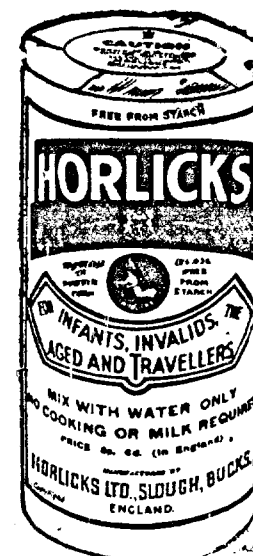
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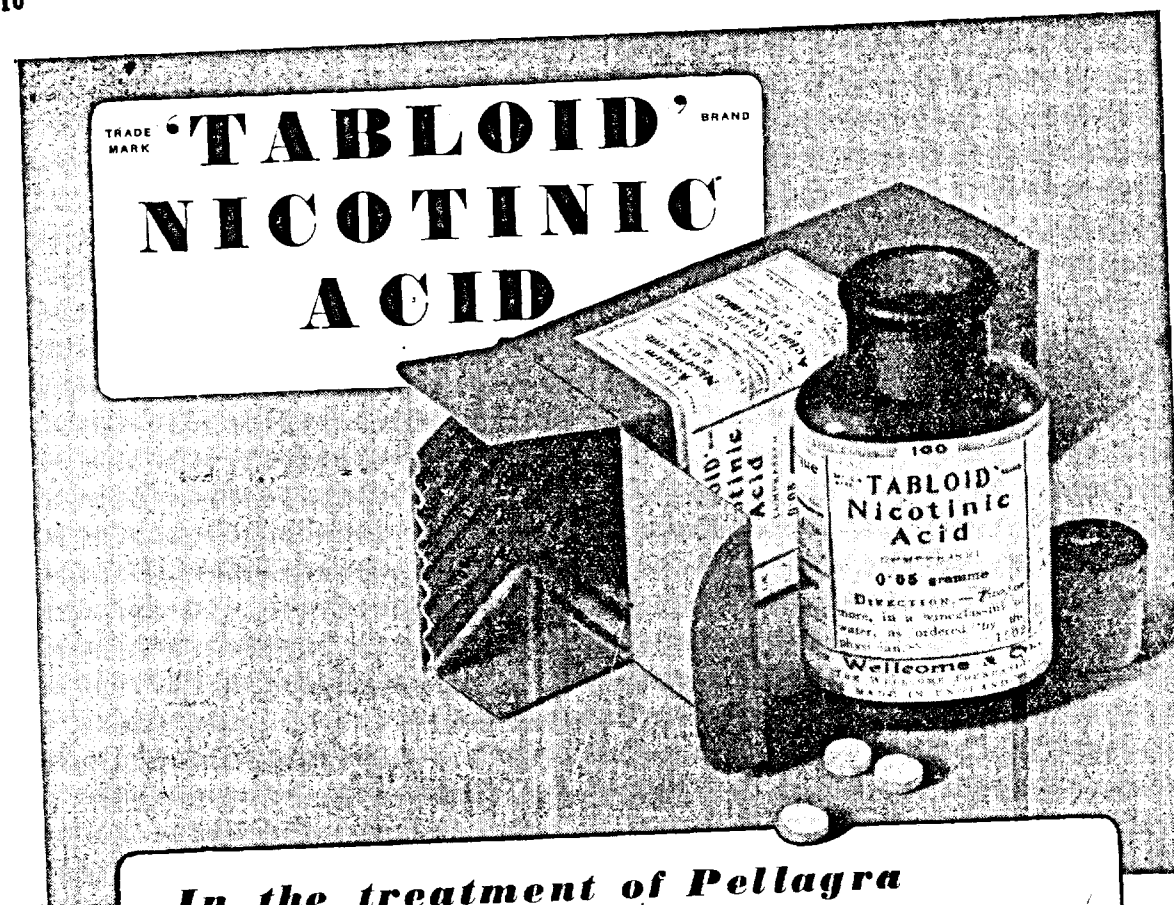
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Vol. 34 || MADRAS : Dec. 1940 || No. 12

Diabetes: Before and Now

MILESTONES IN DIABETIC RESEARCH

In our leading article in the February 1939 issue of the *Journal* we stated in broad outline that there have been four milestones in diabetic research: (1) The discovery that the disease was the result of a deranged carbohydrate metabolism (2) The attempts to trace the aetiology of this derangement to pancreatic lesions (3) The discovery of the islets of Langerhans and its internal secretion, the insulin, which controlled the metabolism of glucose in the body and (4) That diabetes mellitus was caused by the deficiency or absence of this internal secretion. At the same time we also stated that insulin was but one link in the chain of diabetic aetiology, and that strong experimental and clinical claims have been put forward by research workers involving the undoubted but undetermined influence of the hypophysis, the adrenalin cortex and the vitamins on the diabetic syndrome. And we

concluded by saying that the last word in diabetic research had not been said yet, nor insulin be taken as the last discovery in the treatment of diabetes mellitus.

MECHANISM OF DIABETES MELLITUS

While the position with regard to the specific aetiology of diabetes mellitus is almost the same as when we reviewed it last year, there have been in the interval a few outstanding contributions elucidating the mechanism of diabetes mellitus, notably by Himsworth in England, and B. C. Roy and H. N. Mukherjee in our country. The former classified diabetes into two types: the insulin sensitive and the insulin insensitive. According to him the first type is due to deficiency of insulin, tends to be severe, and in it the diminished ability to utilise carbohydrate is inadequately compensated. The second type is due not to lack of insulin but to insensitivity to insulin, is generally less severe, and in it the ability to utilise carbohydrate is usually more adequately compensated. These types are clinically represented, of course roughly, by the young, lean diabetic, and the middle aged plethoric diabetic. He concludes that high-blood sugar in diabetes is a controlled and compensatory mechanism with the specific object to facilitate the utilisation of glucose by the tissues; that glycemia is produced by the increased activity of the liver under the influence, perhaps, of the anterior pituitary gland; and that the diminished abilities of the tissues to utilise glucose is referable either to deficiency of insulin or to insensitivity to insulin, although it is possible that both factors may operate simultaneously. His exposition of the mechanism of diabetes may be shortly expressed thus: if the head of pressure of sugar in the blood is high, sugar utilisation and insulin action are facilitated; if it is low sugar utilization and insulin action are retarded. Roy and Mukherjee maintain, supported by experimental evidence, that hyperglycemia is an active body mechanism to combat acidosis which is the primary factor in the pathogenesis of diabetes. This acidosis, according to them, may result from ingestion of acid bodies, or from faulty method of living which causes retention of CO₂ and production of ketone bodies or from hyperalimentation specially with rich protein and fat foods for a long time. It is this continued acidosis which overstrains the delicate insulin secreting mechanism and brings about the condition of diabetes mellitus. They, however, recognise that the same factor which causes acidosis also depresses insulin action.

Index to Vol. 34 accompanies this Issue

METABOLISM IN DIABETES MELLITUS

The injury to the insulin-secreting mechanism affects the normal metabolism of sugar interfering with sugar production in the liver and sugar absorption in the tissues. Normally the level of sugar in the blood is maintained by a subtle mechanism which strikes a mean between two contending forces, viz., sugar production and sugar absorption. Thus defective storage and defective utilisation of sugar are the two cardinal features of diabetic metabolism. The causes of this metabolic disturbance are many and varied. Chronic sepsis and chemical, nervous and hormonal factors play their respective parts in a vicious circle and in an endless chain of cause and effect. The nature of this metabolic disturbance is however understandable in the light of modern knowledge about food metabolism in the body. Either from inefficient action of insulin or from lack of it, the normal conversion of carbohydrate, first as glycogen and then as glucose, and the normal utilization of glucose by the body tissues, particularly the muscles, are relatively affected resulting in the strange spectacle of sugar starvation of the tissues despite the high-blood sugar level. In other words, what ought to go for the maintenance of the tissues gets locked up in the blood, and partly excreted in the urine resulting in glycemia, glycosuria and ketosis.

KETOSIS, GLYCOSURIA AND GLYCEMIA

Ketosis represents the breakage of acid-base equilibrium of the blood plasma, brought about by incomplete combustion of fat and the unusual attempt of the body to convert fat, protein and other amino-acids into sugar in the face of a frank failure of normal carbohydrate metabolism. Thus the blood gets loaded with the products of abnormal metabolism. These products are called ketone bodies, and when they are in excess we get the dangerous condition of diabetic coma—a sort of auto-intoxication. Hyperglycemia and glycosuria are indeed compensatory phenomena, and prevent, as far as possible, the onset of explosive ketosis.

DIAGNOSIS OF DIABETES MELLITUS

It has now become a trite observation that a mere discovery of glycosuria does not necessarily constitute a diagnosis of diabetes. This is so because of the modern conception of the average threshold of the kidneys for sugar and the benign variations from this average. Many cases of glycosuria do not show abnormal blood sugar level, and some cases which show abnormal blood sugar level do not have glycosuria. The former is due to

low renal threshold, and the latter to high renal threshold. The normal renal threshold is usually found to be between 170 and 200 mg. per 100 c. cm. depending on whether the analysis is made on venous or capillary blood, and this figure represents the critical concentration of sugar in the blood which determines the presence or absence of glycosuria.

But if glycosuria is associated with ketonemia it is certainly pathognomonic of diabetes, and a preliminary diagnosis of diabetes is quite justifiable. This however should invariably be verified by the determination of the blood sugar level and glucose tolerance test. We think it is not necessary for the purpose of this leading article to go into the technical details of these tests or into their significance and interpretation.

TREATMENT OF DIABETES MELLITUS

The aim of treatment is to re-establish toleration to normal carbohydrate food, and normal carbohydrate metabolism. Before the discovery of insulin and its application in the treatment of diabetes, the disease was sought to be controlled by relative degrees of starvation and withholding of carbohydrate food. While this did some good to plethoric patients it did more harm to thin, lean and young patients. In fact it was soon found out that the total deprivation of carbohydrate interfered seriously with fat and protein metabolism resulting in acidosis and coma, although the total caloric value was maintained by extra fat and protein rations. In 1922, Banting and Best successfully prepared their first crude extract of insulin, and showed that it was possible to control diabetes both experimentally and clinically. The method of its preparation was quickly improved and its dosage was determined according to the need for carbohydrate utilisation over and above the limit of toleration.

Its administration also was so timed that carbohydrates ingested might be absorbed and metabolised properly. But its disadvantages soon became obvious. It could not be administered by mouth, because it is quickly destroyed by pepsin and trypsin. It has therefore to be administered subcutaneously. Its action is evanescent, and when the insulin injections are stopped hyperglycemia shows itself again. Thus its rapid absorption and rapid excretion have necessitated its repeated administration to control the blood-sugar, which means that the diabetic patient finds no specific cure for his ailment but that he should become a compulsory addict to too frequent insulin injections if he is to avoid the serious

medical and surgical complications of the disease. Hagedorn in 1935 and Scott in 1936 found that protamine and zinc compound of insulin overcomes this necessity for repeated injections of simple insulin by creating a small store-house at the site of injection and thus affording release of insulin in so slow and constant a stream as to facilitate optimum carbohydrate metabolism and utilisation of sugar by tissues. If hyperglycemia is high, this may be preceded by an injection of simple insulin. But in cases where the blood sugar level is not very high or where there is no excessive degree of ketosis, one injection of zinc protamine insulin before breakfast would suffice. The danger of insulin compounds lies in the insidious onset of hypoglycemia at an unpredictable time, and the possibility of its being overlooked both by the patient and his physician.

The relative value of diet regulation, insulin and insulin compounds in the control of diabetes is thus obvious. Not by a single line of treatment but only by a judicious combination of all available means of blood sugar control can success be attained. The present accepted lines of treatment may be briefly expressed. Wholesale starvation for a couple of days is definitely dangerous in juvenile diabetics while in the obese, middle aged diabetic a preliminary relative starvation may do some good. Simple insulin is needed if there is extreme intolerance to carbohydrate diet, and if there is excessive ketosis threatening coma. Insulin compound is the drug of choice at present, re-inforced by a small dose of simple insulin if immediate action is needed, in the control of diabetes, and while it has immeasurably improved the outlook of the diabetic patient it has however multiplied the problems of the physician in ascertaining the dose of daily injections and in keeping guard against its cumulative effect and insidious onset of hypoglycemia.

CONCLUSION

Such, in brief, is the account of DIABETES BEFORE AND NOW. There is no doubt that we have a better understanding of diabetes mellitus today. But there are still wide gaps in our knowledge which will perhaps be filled up in future. It is obvious that mere lack of insulin is not the only factor in its aetiology, nor do we know how exactly insulin helps glycogenolysis and utilisation of glucose by the tissues. It is also equally obvious that the insulin treatment of diabetes mellitus while it has enabled us to achieve control over acidosis and hyperglycemia has however made that

control a question of daily vigilance and perpetual dole of insulin. In other words, insulin gives us control but no permanent cure of diabetes mellitus. It is unfortunate that the nature of the disease is such as would not permit its permanent eradication.

Some of the other important points not covered by this leading article are discussed below under CURRENT COMMENTS.

CURRENT COMMENTS

Renal Glycosuria

Renal glycosuria is the result of a harmless functional peculiarity of the kidney to maintain the normal threshold for sugar. Normally the glucose from blood diffuses readily through the glomeruli, passes and filters through the tubules, and is reabsorbed there to avoid the waste of a valuable substance. Thus in a normal person there will be little trace of sugar in the urine while the blood sugar is maintained at a constant level ranging between 0.08 to 0.12%, and since sugar is a threshold substance no sugar is excreted in urine of a normal person even after a carbohydrate meals although blood sugar shows slight variations within its optimum limits. In renal glycosuria however, there is glycosuria despite the blood sugar concentration being normal. Such cases constitute what is called low renal threshold indicating neither disturbed carbohydrate metabolism nor renal disease. Renal threshold can be easily determined by a simultaneous comparison of glycemia and glycosuria by means of the standard glucose tolerance test. A high renal threshold is a condition in which blood sugar concentration is high with little or no glycosuria. Both low and high renal thresholds may co-exist along with diabetes mellitus, and this naturally leads to many difficulties in diagnosis and treatment. Little is known regarding the cause or causes that lead to the alterations in kidney threshold for sugar. This anomaly seems to be hereditary and fortunately quite innocuous. Lawrence, however, observes that the renal threshold is temporarily lowered in polyuria, pregnancy, after pituitary injections and severe toxemia; and raised temporarily in circulatory failure, diabetic coma and nephrosclerosis.

The Role of Hormones and Vitamin B₁ in Diabetes Mellitus

The role of the pancreatic, pituitary and thyroid hormones in the production of diabetic syndrome is yet imperfectly understood, and the current ideas are at best merely

an attempt to arrive at the truth. The crucial point is the understanding of the carbohydrate metabolism, and the factors which make this process normal or abnormal. Normally liver converts the ingested proteins and carbohydrates into glycogen and then into sugar which is thrown into the blood, and which is enabled to be utilized by the tissues for their maintenance and work by the action of pancreatic insulin. Thus the functions of insulin are to check excessive glycogenolysis in the liver, to convert the circulating glucose into muscle glycogen, and thus to keep up an optimum concentration of blood sugar. How insulin itself acts in this manner we do not know, but we do perceive a delicate balancing mechanism between anterior pituitary, adrenalin and pancreas in their action on liver and peripheral tissues, and that this mechanism may be upset by a partial or complete break in anyone of its constituent parts. The influence of these glands on carbohydrate metabolism may be diagrammatically represented thus:

CARBOHYDRATE METABOLISM

Pancreatic Insulin

Liver	Peripheral tissues
Inhibits glycogenolysis	Enables the tissue to utilize sugar
Antagonises glycogenolytic action of adrenalin and thus regulates sugar formation.	Stores it as muscle glycogen.

Anterior Pituitary

Liver	Peripheral tissues
Facilitates:	Retards:
(1) Formation of sugar from non-carbohydrate sources (fats and proteins)	(1) Insulin action on blood sugar
(2) Breakdown of glycogen into blood sugar	(2) Muscle glycogen formation
	(3) Oxidation.

Adrenalin

Liver	Peripheral tissues
Facilitates:	Converts:
(1) Conversion of stored glycogen into sugar to meet an abnormal need.	(1) Muscle glycogen into sugar.

(After Himsworth)

A close study of the above would reveal the complicated nature of the role which these hormones play in the normal and abnormal metabolism of carbohydrates. Apart from the fact that each gland by its own dysfunction may produce a diabetic condition, defective insulin secretion results in what may be called, for the sake of clarity, pancreatic diabetes. This, we think, is more common than any other type. We also know that hyperfunction of the pituitary, may, besides being responsible for conditions like acromegaly or Cushing's syndrome, produce hyperglycemia and ketosis.

There is no doubt of the secondary role which the anterior pituitary plays in the compensatory mechanism in diabetes, but there is yet little evidence of its being a primary factor in ordinary human diabetes. Similar is probably the position with regard to hyper-adrenalism and hyperthyroidism.

Peters has pointed out that vitamin B₁ also plays an important part in the utilisation of sugar since pyruvic acid, which is one of the breakdown products of sugar, can only yield the necessary final oxidation products through the agency of vitamin B₁.

Insulin Efficiency and Insulin Deficiency

In our leading article we have referred to Himsworth's classification of diabetes into two categories: one caused by insulin deficiency and the other by the inefficient action of insulin. He calls them insulin sensitive and insulin insensitive. The characteristic action of insulin lies in its ability to increase the rate in which the peripheral tissues remove sugar from the blood for their utilization. In the type of diabetes due to lack of insulin, artificial reinforcement of the same brings down the sugar concentration to normal and helps the tissues at the same time to utilize the blood sugar. But in the type of diabetes not due to lack but to inefficient action of insulin, the beneficent result of insulin treatment is not obvious, because, according to Himsworth, the action of insulin is impaired by a substance secreted by the anterior pituitary gland. He further observes that in the insulin-insensitive type, the islets of Langerhans have been found to be normal, and concludes, therefore, that there is some pituitary factor which prevents the normal functioning of insulin. His attempts to trace this anti-insulin factor in the body fluids of diabetics met with no success except in a case of acromegaly and two cases of Cushing's syndrome. From this, it follows that insulin-sensitive diabetes is more amenable to treatment by insulin than the other type where there is a pronounced degree of pituitary antagonism to the action of insulin, natural or therapeutic. We think, however, that the first type is more common than the second, and that till we arrive at a more accurate and comprehensive knowledge it is justifiable to believe that most cases of diabetes mellitus are due to insulin deficiency.

Hyperglycemia and Hypoglycemia

These two conditions must be clearly understood, because, although the symptoms of their manifestations are more or less alike,

yet their cause and treatment are entirely different. Hyperglycemia is a condition of blood sugar in a state of high concentration, and indicates the raising level of ketosis which when it reaches the saturation point develops into what is commonly called diabetic coma. Hypoglycemia, on the other hand, is a condition of blood sugar much below the normal concentration, usually due to ill-managed treatment of diabetes by insulin, although spontaneous hypoglycemia is not unknown. Particularly, after the introduction of protamine-zinc insulin in the treatment of diabetes, hypoglycemia may set in as insidiously as hyperglycemia with such symptoms as headache, restlessness, nausea, suppression of urine, coma and convulsion. Since the treatment of one is the very opposite of the other, differential diagnosis should be quickly made and immediate treatment instituted. In explosive hyper-glycemia, we have the acetone smell of the breath, dilated pupils and presence of sugar and ketone bodies in the urine, and high concentration of blood sugar. In hypoglycemic shock or coma we have none of these symptoms. There will be an history of insulin treatment, and blood-sugar much lower than normal. Insulin and alkaline transfusion are indicated in hyperglycemia. Withdrawal of insulin, plenty of glucose by mouth or intravenously, and adrenalin injection are indicated in hypoglycemia. Both the conditions are preventable, and if not prevented it is an evidence perhaps of mismanagement of the case.

Ketosis

Ketosis is a critical stage of diabetic condition and is due to extreme acidity of blood plasma as a result of incomplete products of fat and protein combustion circulating in the blood. This again is the result of the failure of carbohydrate metabolism. In the absence or deficiency of utilizable carbohydrate, nature attempts to make sugar out of fat and protein, more out of fat than of protein. But this only results in incomplete combustion of fat, and the plasma is littered with products of incomplete combustion, namely, aceto-acetic acid, B. oxybutyric acid and acetone. Usually the degree of the presence of acetone bodies in urine and the amount of CO₂ in the alveolar air are taken as a measure of the acidosis. Probably, the symptoms associated with ketosis are due not so much to acidosis and diminution of alkali reserve of the blood plasma and tissues but to ketonemia—expression of specific toxic action on the part of acetone bodies. The clinical counterpart of ketonemia is diabetic coma. It must be

remembered that in every case of diabetes mellitus there is a certain degree of ketosis and ketonemia, but it occasions explosive symptoms only when ketosis is of a severe degree. The essential principle in the treatment of ketosis is to supply the body with utilizable carbohydrates in the form of glucose which is necessary for proper katabolism of fat and sufficient insulin to help this process, preferably with alkalies by mouth or intravenously in severe cases.

Blood Sugar Tolerance Test

Blood sugar level in normal persons is remarkably constant, and this constancy indicates a delicate balancing mechanism behind it. The fasting level is between 0.08 and 0.12 per cent, and expressed in terms of milligrammes of glucose per 100 c. c. of blood it will be 80 and 120 m.g. per cent. After food, or ingestion of glucose, it raises to 170 mg. with the first hour but soon resumes its normal by the end of two hours. This is what is called the sugar tolerance curve. A fasting blood sugar above 120 mg. per cent indicates mild diabetes, and above 170 mg. per cent severe diabetes. The important point in the sugar tolerance test for diabetes is not the maximum height attained by blood sugar after the ingestion of glucose but the failure of the sugar to fall to its original level within two hours. Himsworth points out that the course of the blood sugar tolerance curve in diabetes is uninfluenced by the degree of glycosuria. He cites the case of a middle aged woman suffering from severe diabetic mellitus who though passing large quantities of sugar in the urine had a blood-sugar level fairly constant. In other words, it means that blood sugar level is uninfluenced by loss of sugar in the urine although large quantity of sugar may be depleted through this path. He believes that the outflow of sugar into the urine is actively compensated by an equivalent inflow of sugar into blood and this factor is responsible for the constancy of blood-sugar level.

The sugar tolerance test is done as follows: First the blood sugar level in fasting condition is ascertained. Then 50 grammes of glucose in 100 c. c. of water is given. Blood sugar estimations are carried out at half-hourly intervals and noted down in graph paper. J. P. Bose draws attention to the possible loss of sugar in blood samples as a result of effect of high-room temperature and unusual delay in the performance of the test. It seems essential therefore that samples of blood sent for determination of blood sugar should be examined as early as practicable.

Hypercholesteremia

J. P. Bose reports that the determination of the cholesterol content of the blood will be useful and give reliable data regarding the severity of a case of diabetes and its prognosis. He believes that the degree of hyperglycemia present is not always a reliable index, by itself, of the severity or otherwise of a case. He makes this observation after a study of four different groups of cases. Group I consisted of cases with normal cholesterol, although some of them had high fasting blood-sugar level. The response to treatment was very satisfactory and most of them improved on dietetic treatment alone. They were also free from all complications. Groups II to IV were arranged according to increasing cholesterol content of blood. It was found in these three groups that the severity of the diabetic condition and the complications present varied directly according to the degree of hypercholesteremia irrespective of whether the blood sugar was high or low.

Insulin and Insulin Compounds

Insulin is soluble and stable in acid solution, but is rapidly destroyed by alkalis. Hence it could not be orally administered. It is rapidly absorbed from the site of injection and excreted from the body probably after 6 or 7 hours. Thus arises the necessity for frequent insulin injections. There is no fixed dosage for insulin: the dose depends on the severity of the case, the presence of complications, and the diet prescribed. As a rule, it has been found that one unit of insulin can usefully metabolise one to two grammes of carbohydrates. On this basis, J. P. Bose reckons that the total number of insulin units required would be half of

Total glucose value of food + Total amount of sugar excreted in urine in 24 hrs.

2

If, however, the number of units required is more than 20, it should be divided and administered before tea, breakfast and dinner and this would help proper utilization of carbohydrates and thus keep the blood sugar level at or about its normal. Fortunately, insulin does not, unlike opium, tend to habit formation, and although prolonged insulin therapy is needed in a case of the diabetic, it is still possible that "the patient may improve so considerably that the dose of insulin may gradually be reduced until it may ultimately be stopped".

Insulin compound is insoluble because of its combination with a basic protein, called

protamine, and zinc. It has been found that an injection of insulin compound made up of both protamine and zinc is capable of controlling the blood sugar level for nearly 24 hours. And it does this by creating a ready store of insulin at the site of injection, and its insolubility helps in such a storage and slow absorption. The definite advantages of protamine-zinc insulin over soluble insulin are (1) it enables carbohydrates being consumed evenly between all the meals (2) a greater sense of well-being throughout the day (3) less likelihood of hyperglycemia and ketosis developing during night.

Mild cases of diabetes respond excellently to a single injection before breakfast. Severe cases may require a preliminary dose of soluble insulin before breakfast to control the post-prandial hyperglycemia, and followed by protamine zinc insulin 6 or 8 hours later. As regards the dosage, the number of insulin units required per day is to be arrived at in the same manner as for ordinary insulin.

The one great disadvantage of protamine zinc insulin is its cumulative action and a consequent possible onset of hypoglycemia. This is invariably due to mismanagement of the case.

It is necessary to note that the site of insulin injection should vary every time, since it has been found that if injections are given in the same spot, they not only injure the subcutaneous fat and muscles more or less permanently leading to what is termed as *insulin atrophies* but also effectively hinder proper absorption.

It may also be equally interesting to note, and J. P. Bose has recorded a few such cases, that some patients are highly sensitive to insulin and develop allergic symptoms. In such instances, a different brand of insulin may be tried or desensitization effected by minute doses of insulin.

Some cases are highly insulin-resistant. Probably they are not suffering from pancreatic diabetes.

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Metabolism in Diabetes Mellitus

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The great worker Minkowski and his school advocated the theory of under-utilization in diabetes. They held that in diabetes the tissues lose in a varying degree the power to oxidise sugar.

Von Noorden advocated another theory as regards the pathological physiology of the condition. This theory is quite opposite to that of Minkowski and is known as overproduction theory. He believed that the disease represented increased glycogenolysis with over-production of glucose from non-carbohydrate sources, i.e., from proteins and fat. Both these schools, however, maintain that the internal secretion of the pancreas-insulin corrects the abnormal tendency. On the one hand, insulin promotes proper utilization of sugar in the tissues and on the other, insulin inhibits the abnormal gluconeogenesis. None of these theories gives us a complete picture of the pathological physiology and the course of metabolism of the disease. Besides, they do not satisfactorily explain the various problems which the condition of diabetes presents. Before we consider these problems it would be better if we try to picture the course of metabolism that obtains in the normal condition, and then we would be able to understand the pathological variation that occurs in diabetes.

Carbohydrates are absorbed as the hexases, glucose, fructose and galactose. Absorption of these substances occur mainly in the small intestine. In the stomach it is slight or variable. The rate of absorption is constant for each sugar and independent of the amount in the intestine. The maximum amount of sugar that can be absorbed has been estimated to be 1.8 gms. per kilo per hour. Cori and Cori estimate that the glucose absorption rate in man can probably not exceed 0.85 gm. per kilo per hour.

The absorption process thereby constitutes a factor of safety preventing an inordinate rise of blood sugar. In normal condition the blood sugar level remains in the neighbourhood of 1% (i.e. 100 mgm. in 100 c.c.), but during absorption the blood-sugar level in the portal vein may go up to 300 mgms. in 100 c.c. The sugar thus circulating in the blood is free and diffusible and is carried to the liver. Brought thus to the liver, this organ, during digestion when it is supplied with much oxygen, picks out from the blood some of the glucose, levulose and galactose passing through it and converts these into a colloidal polysaccharide—glycogen (glycogenesis). This glycogen is laid down in such quantities in the

liver tissue that on a rich carbohydrate diet the liver may contain 12 to 15% of glycogen. The total quantity of glycogen, that can be stored up in the liver at one time, varies between 200 to 250 grams. The liver acts thus, in the first instance, as a store-house or reserve depot of carbohydrates in which surplus carbohydrate is stored until it is needed by other tissues. It functions, then, in this particular, like the adipose tissue which stores superfluous fat. Between meals, on the other hand, when no glucose is arriving from the intestine, the process is reversed, glycogen is hydrolised and glucose flows from liver cells into the blood (glycogenolysis).

The absorption of glucose by the liver cells and its conversion into glycogen and, further, the transformation of glycogen into glucose depends on various factors. The absorption of glucose by the liver cells is due to the concentration gradient. The tension of glucose inside the cell in resting condition may be taken to be that of 1% sugar, i. e., that of the blood-sugar level. When due to activity the cell-sugar is consumed, the tension of glucose becomes less and the sugar from the blood flows into the cell and brings about an equilibrium of tension.

The role of the liver cells, in this respect, is quite different from that of the other tissue cells of the body. The liver cells are not only consumers of glucose like other tissue cells but, in addition, they are to act as store-house of sugar for other tissue cells. Best finds the glucose content of the muscles to be 0.2 to 0.4% whereas that of the liver-cells 0.6 to 1.5%. During digestion the portal blood carries sugar at a much higher concentration (3% or even more) and thus a marked difference of tension of sugar between that in the cell and that in the outside blood is presented (got up).

To equalise the tension large amount of glucose enters the cell. When the sugar in the cells reaches a certain concentration, it is stored up in its polymerised form of glycogen. With the glycogenesis the tension of glucose inside the cells immediately falls and a further amount of sugar gets in. The process is repeated till the glycogen-content of the cells reaches its maximum. With further supply of sugar, the glycogen would partly be converted into fat and would help to accommodate more sugar in the cells. By this process two purposes are served. First, the maximum glycogen storage is ensured, and, secondly, the blood-sugar level which was very high to start with, in the portal vein, is brought down to the level of the general circulation. From the general circulation all tissues of the body are supplied with glucose. The muscles store it up in the form of glycogen. The glycogen content of the muscles as given by Best is 0.2 to 1.8% which in comparison with that in the liver (0.2-10.0%) is only a very small amount. But as the muscles form 50% of the body-weight, the total quantity of the sugar stored up in the muscles is quite considerable and even more than that in the liver.

Glycogen, though chiefly stored up in the liver and muscles, is also found in most of the tissues of the body. The relationship between the glycogen of the liver and that of the muscles is quite different. In general chemical properties, the liver-glycogen and the muscle-glycogen seem to be identical, and yet, the glycogen in the liver breaks down immediately following death into glucose, whereas that in muscle forms lactic acid. This essential physiological difference seems to depend on the process of break-down being controlled by entirely different types of enzyme in the two cases.

There is little doubt that in the normal living animal all the blood-sugar in the post absorptive state has its source in the liver. This store-house of glycogen is responsible for the constancy of the blood-sugar, any removal by tissues with resulting lowered concentration being immediately compensated by glucose poured out from the liver, formed by the hydrolysis of glycogen.

This conversion of glucose into glycogen and of glycogen into glucose depends on various factors. The ferment glycogenase has been held responsible for both the processes of glycogenesis and glycogenolysis. It is the change in the environment that decides which way the process would proceed. If the blood-sugar level is high, if the liver is well-supplied with oxygenated blood and with the internal secretion of pancreas the glucose is converted into glycogen. If, however, the content of glucose in the blood coming to the liver falls or if oxygen-supply is reduced the process is reversed.

When adrenalin is injected or when it is present in the blood in a state of emergency, the blood vessels in the liver are constricted, the blood-supply becomes limited, a condition of local asphyxia is produced and thus the glycogenase is presented with the required environment for the glycogenolysis. The glycogen is converted into glucose and the blood is flooded with sugar and this will take place even though the amount of glucose in the blood is already above the normal. The ultimate fate of glucose in the body is to be burnt up in the tissue cells. It is completely oxidised into CO_2 and H_2O and by its oxidation it supplies the energy and the body-heat. The tissues which consume most of the sugar are undoubtedly the muscles. In the muscles work is continuously going on, and more so during exercise. Even to maintain the tone of the muscles there is work and work means burning of a fuel, and sugar serves as the principal fuel.

Sugar is of prime necessity to the cell. A continuous fire and constant combustion is going on in the cell and its very existence as a living unit is but a manifestation of this fire. The moment this combustion stops, the cell is only a dead matter. A constant fire means a constant supply of fuel, and sugar is the fuel.

The main purpose of the whole metabolism seems to be to keep up a constant supply of sugar to the cells of the body and also to regulate the amount according to their activity.

The constancy of the blood-sugar level not only shows the importance of sugar as a fuel but also the nicety with which the supply is maintained and regulated with the varied activities of the tissue-cells. If, by any chance, the sugar-level in the blood goes down, disastrous consequences happen and life becomes impossible. On injection of a high dose of insulin, particularly in an animal which is being starved of carbohydrates, the blood-sugar level goes down, grave symptoms due to hypoglycaemia appear, and unless the condition be immediately relieved by introduction of sugar, the animal dies. But such a condition is impossible in normal state. One may cut off the carbohydrate supply entirely and carry on long continued exercise, burning off the sugar in the body, but still hypoglycaemia up to the level of causing alarming symptoms never appears. Nature realising the prime importance of sugar has provided for such eventualities. When the sugar supply from carbohydrate falls short, the system immediately manufactures sugar from other proximate principles, such as proteins and fats, and thus the blood-sugar level is constantly maintained. Even when the supply of proteins and fats are cut off, as in starvation, the body proteins and fats are broken down to yield sugar, and the blood-sugar level is maintained to prolong the life. Liver is not only a magazine of carbohydrate, but it is also a factory where amino-acids, lactic acid, dihydroxy-acetone and possibly glycerol and fatty acids are converted into glucose. By this synthetic power of liver not only is the body able to form glucose from protein and other foods, being able to live on proteins alone, but some of the partially oxidised metabolic products of other tissues, such as lactic acid, are saved and resynthesised into glucose to be used over again. All the amino-acids, however, are not sugar or glycogen formers. Those which play the role are glycine, alanine, aspartic, glutamic and hydroxy glutamic acids, cystine, arginine and proline. The quantity of these amino-acids present in 100 grams of protein is sufficient to form about 58 grams of glucose.

In normal condition, when the sugar-supply of the body is intact, the proteins do not contribute 58% glucose. The aminoacids are required intact for building body proteins, for the manufacture of hormones and other secretions. It is only when they are in excess and are not so required that they are broken up. The incombustible amino group (NH_2) is split from the amino-acid molecule and the ammonia so produced is combined with carbon-dioxide to form urea. The non-nitrogenous moiety—the fatty acid residue—is transformed into glucose. But in case of necessity, when the sugar-supply of the body is cut off, the proteins would yield its maximum quota of sugar even at the sacrifice of its normal rate of building up and repairing of body proteins and the manufacture of hormones and secretions.

This suggests that there is a mechanism in the body which regulates and controls the sugar-supply to the system. This mechanism depends on the hormones of the different endocrine glands. The

anterior pituitary with its so-called diabetogenic and ketogenic hormones helps in the breaking down of proteins and fats to yield sugar. These hormones seem to be constantly working except when its action is inhibited by the pancreatic hormone. The pancreas secretes its hormone only when there is an income of sugar from carbohydrates. With the ingestion of carbohydrates and sugars the blood sugar level goes up and this stimulates the pancreas to secrete insulin and so long the income is continued, the secretion of the pancreatic hormone is maintained. The insulin completely inhibits the anti-pituitary hormones and checks the proteins and fats from forming sugar enabling them to carry on their own role of growth and repair, etc., for the time being.

As the sugar formation from proteins is stopped by the insulin, the sugar-content in the cell becomes low, and a marked difference of tension of glucose inside the cell and that in the blood is presented.

As a result, glucose is rapidly absorbed from the blood and large amount of glycogen is stored up in the cells.

When the sugar income is stopped, the blood-sugar level goes down; the stimulant for the pancreas being removed the secretion of insulin comes to an end. Gradually the effect of insulin passes off, and the hormones of the anterior pituitary come to play. It is the balanced action of the hormones of the anterior pituitary and pancreas that regulates and controls the sugar-supply to the system and maintains the blood-sugar at a constant level. Other endocrine glands also influence the carbohydrate metabolism; adrenals in times of emergency mobilises sugar by increasing the glycogenolysis in the liver. Adrenalin also mobilises muscle glycogen (Cori) but here the immediate product is lactic acid and not glucose. A part of the lactic acid is carried by the blood to the liver where it is converted to glycogen which in turn furnishes the blood with glucose. Adrenalin does not accelerate sugar-formation from other substances in the liver. The main purpose of the metabolism of sugar is to give the maximum efficiency to the system in times of emergency.

Thyroxin has no immediate effect on blood-sugar, but when large doses are administered, changes in carbohydrate metabolism are observed. Patients suffering from Grave's disease exhibit slight hyperglycaemia and glycosuria, but a severe diabetes is not produced. Thyroxin by increasing cellular activity causes an augmentation of carbohydrate oxidation and it mobilises the sugar by glycogenolysis. The glycogen-content of the liver, skeletal muscle and heart becomes depleted. There is a slight increased rate of hepatic glucomogenesis.

It has been observed that when there is over-activity of the anterior lobe of the pituitary in man as in acromegaly, there is hyperglycaemia, glycosuria and lowered sugar-tolerance, all the symptoms of a true diabetes mellitus may be present in this condition. Evans and others observed that when animals (daspohound pups) were given continued injections of anterior lobe extracts, they not only

gained 100% in weight but also exhibited great thirst, polyuria and glycosuria. On the other hand, we find that when there is deficiency in the activity of the anterior lobe of the pituitary, such as in the conditions known as *Dystrophia adiposogenitalis* (Frohlick's syndrome) and in its prepubertal type (Baby Hercules) there is marked increase in sugar-tolerance and also there is great deposition of fat. It has been observed that hypophysectomy renders an animal hypersensitive to insulin, while implantations of anterior lobe or injections of an extract of it reduces the hypoglycemic action of the pancreatic hormone. It was also shown by Houssay that diabetic symptoms following pancreatectomy almost disappear after hypophysectomy but return when anterior lobe tissue is transplanted into the depancreatized and hypophysectomized animal.

Diabetes mellitus is a disease in which profound disturbances of metabolism occur owing, presumably, to the deficiency of pancreatic hormone elaborated by the cells of islets of Langerhans.

During the absorption of sugar from the ingested carbohydrates there is inadequate secretion of insulin. Consequently, there is sufficient check on the amino-acids from breaking down to sugar, even at the time when the sugar income of the body is going on.

One may suggest that the quantity of insulin secreted, being inadequate, is incapable of neutralising completely the activity of the anterior pituitary hormone and, as a result, sugar-formation from amino acids and even fats continue when they should have been completely spared. One can follow the march of symptoms of diabetes as they present themselves with the gradual increase in the deficiency of the pancreas.

When the deficiency is of a very mild grade, the only change in the metabolism would be that a part of the amino acids absorbed by the cell would be transformed into sugar. This sugar in the cell would naturally diminish the difference in tension between that of the blood sugar and that in the cell. Consequently, there would be slight interference in the absorption of sugar brought through the portal vein. But as the quantity of the newly-formed intracellular sugar is very small in relation to the total sugar-holding capacity of the cell, the defect is negligible. Besides, there would be plenty of amino acids that have been spared to carry on their role of growth and repair. The net result is that there is an increase in the sugar-income of the body. It is, as if, the patient has ingested more carbohydrates. When the defect persists for sometime, the patient gradually becomes stout. It has been a noted fact that patients generally put on fat before the onset of diabetes, so much so that certain observers think obesity as the cause of diabetes. Joslin refers to diabetes as the "fat man's folly" and to obesity as the "open door to diabetes". But here, as we see, obesity is really an early manifestation of the disease itself. But it should be noted that obesity may be due to various other causes and those that are really due

to diabetes would certainly show decreased sugar-tolerance.

With the progress of the disease the deficiency of the pancreatic hormone increases i. e., less insulin is secreted in relation to the amount of sugar absorbed. Due to the decrease of the inhibitory influence of insulin more amino acids are converted into sugar. The intracellular sugar retards the absorption of the ingested sugar. The liver cells fail to take up the sugar from the portal circulation with the same rapidity as in normal condition. The blood sugar level in the general circulation consequently rises. The hyperglycemia, thus produced, is only slight in such condition and usually does not reach the threshold level of 18%. The rise in the blood-sugar level acts protectively by providing additional stimulus to the pancreas which then secretes more insulin and attempts to compensate the defect. These patients do not as a rule show any symptoms of diabetes, but when they take an extra amount of carbohydrates, e. g., an additional dish of sweets, the compensatory mechanism becomes inadequate, the blood sugar level oversteps the threshold and passes sugar in the urine.

These are the cases which are generally classed as that of "alimentary glycosuria". One can very well see that it is a mistake, and rather an unfortunate one, as these potential diabetics instead of being taken in hand in time are allowed to become pucca cases of diabetes.

The term "alimentary glycosuria" means glycosuria in a normal healthy subject where the metabolism is quite intact and the sugar in his urine is simply due to his taking in such a large quantity of sugar that his liver, though normal, is incapable of manipulating it, allowing hyperglycemia to develop and consequent glycosuria.

This is only possible when one takes 200 grams or more of glucose in solution in empty stomach.

With a diet of mixed carbohydrates such a condition is not possible. One cannot possibly take in a very large quantity of carbohydrates at one sitting as his stomach would simply refuse it. And even, if he manages, there is very little chance of all being digested and transformed into sugar at the same time. The process would be gradual and so would be the absorption in the intestine. There is hardly any chance of all the sugar to be presented to the liver at the same moment. So, for all practical purposes, there cannot be any alimentary glycosuria after a diet of mixed carbohydrates.

As the disease progresses, more and more of the amino acids would be transformed into sugar, sometimes reaching the sugar-holding capacity of the cells. The cells being full, it is only at the height of absorption that there would be some difference in the tension of glucose inside the cell and that in the blood. It is only then that some of the ingested sugar would be able to get into the cell, otherwise most of the sugar would be refused admission. The blood-sugar level would rise in the general circulation. The hyperglycemic level would be more than the renal threshold. The renal tubules

after reabsorption of the glucose up to its limit of threshold would leave quantities of sugar in the tubules. This unabsorbed sugar in the tubules by exerting osmotic pressure would greatly retard the reabsorption of water, and, hence, there would not only be constant glycosuria but polyuria as well. Owing to the excessive loss of water from the body, great thirst is experienced. Constipation, which is so common in this condition, is due to loss of water from the intestine.

The glycogen-formation goes on almost in the normal manner, but as much of the ingested sugar is wasted particularly during the period when favourable environmental condition for the glycogenesis is presented, the amount of glycogen formed is decidedly less than normal. It has been observed that while the glycogen-content of the muscles of a depancreatized animal may be reduced below the normal level, appreciable amounts remain during the life of the animal. There is no diminution of heart-muscle glycogen and recently convincing evidence of an increase has been obtained. Liver glycogen in these animals falls to very low levels.

It is a well-known fact that the nitrogen-elimination depends on the protein intake. In diabetes, the amount of nitrogen eliminated in the urine is increased. This is generally explained by the large quantities of proteins in the diabetic dietary. But in cases where the protein ingest is restricted, it is found that the nitrogen elimination exceeds the amount taken in. The difference is due to the breaking down of body proteins. In the starving diabetic a constant ratio is attained in the urine between the sugar and the nitrogen excreted. The D:N ratio is about 3:6. The ratio being constant it is obvious that the sugar and the nitrogen are coming from a common source, which can only be tissue proteins. On the administration of the protein-meal both the nitrogen and the sugar excretions are increased but the D:N ratio still remains the same. This shows definitely that all the sugar-forming amino acids of the proteins are being transformed into sugar.

As the amino acids are mostly used up in supplying the fuel, tissue-repair and growth and the manufacture of the hormones and secretions are all badly neglected. The result is that the patient begins to lose weight and as his body-proteins begin to break down, he gradually becomes thinner and thinner.

The vulnerability of the diabetic patient to infection is proverbial. In no other condition we see such susceptibility to infection or such a lack of resistance on the part of the tissues. According to some it is believed to be due to the raised sugar-content of the tissues, but it has not been established. The real reason seems to be the unrepaired and so unhealthy wasted condition of the tissue-cells and the altered and less effective defensive mechanisms of the body brought on by the abnormal metabolism of the proteins.

When the store of glycogen gets depleted the liver sends a requisition to the fat depot. As an immediate stimulus for this transfer of fat from

the depot to the liver, the depletion of the glycogen store seems to be necessary. Rosenthal has observed that a more or less reciprocal relationship exists between the glycogen and the fat of the liver.

As a preliminary change, the ferment in the fat depot makes the fat diffusible. The diffusible fat gets into the liver cells where it is transformed into sugar.

During the transport there is a marked lipoemia. Though the liver is the chief centre for the conversion of fat into carbohydrate, it is quite probable that the other tissue-cells of the body are also capable of such transformation, particularly when there is a great demand for sugar. The ketogenic hormone of the anterior pituitary seems to play an important role in the metabolism. In diabetes large quantities of ketone bodies such as B-oxy-butyric and acetoacetic are formed. These substances pass into the blood causing a ketosis. It is believed that incomplete oxidation of fat, in the absence of utilisable sugar, is the cause of the production of these abnormal acids. We have seen that whatever be the source of sugar there is never any want of it and the oxidation of that sugar goes on almost in the normal way. It seems quite probable that the ketone bodies are formed during the transformation of the fat into sugar mainly by the liver cells but also other tissue cells. In diabetes, besides the fat, there is another source of the ketone bodies. Of the various amino acids that have been isolated from the proteins it has been seen that the phenyl alanine, tyrosin and leucine and possibly histidine can give rise to acetoacetic acid. The relative amount of ketone bodies that are formed in the body derivable from fats and proteins respectively can be seen from Woodyatt's formula for ketoniketogenic ratio.

The formula reads:—

$$\frac{90 \text{ per cent fat} + 46 \text{ per cent protein}}{100 \text{ per cent carbohydrates} + 58 \text{ per cent protein} + 10 \text{ p.c. fat.}}$$

From this it is evident that a large amount of Ketone bodies may be contributed by the proteins. When we consider the heavy break-down of proteins that occur in diabetes, we see how great a part is played by these bodies in the protection of ketosis in the condition.

When these acid bodies of abnormal metabolism are poured into the blood, they are neutralised by the plasma bicarbonate. The ratio $\text{H}_2\text{CO}_3/\text{BHCO}_3$ is thus disturbed and there is a tendency to acidoemia. Immediately augmented breathing sets in and additional amounts of CO_2 are eliminated and thus the ratio $\text{H}_2\text{CO}_3/\text{BHCO}_3$ is approximately restored.

To maintain the reaction of the blood other factors come into play. The kidney excretes a highly acid urine containing much (NaH_2PO_4) acid sodium phosphate. The ratio ($\text{NaH}_2\text{PO}_4/\text{Na}_2\text{HPO}_4$) in the blood is $1/4$ whereas in the

normal acid urine it is 9/1, but in these conditions the ratio may go up to 50/1. By this device a large amount of the body's fixed base is saved. Further, the renal epithelium manufactures NH_3 from the urea conveyed to it in the blood. In health, the daily excretion of ammonia amounts to, from 300-500 c.c. of $\text{N}/10 \text{ NH}_3$. In severe diabetes, it may be ten times this value. The abnormal organic acids are neutralised by this ammonia and excreted as salts. But when the quantity becomes still more large the urine may become sufficiently acid so that considerable portions are excreted uncombined with base. Some of the acetoacetic acid is also decomposed to form CO_2 which can be excreted in the lungs or is changed to a neutral compound, acetone, which is excreted both by the lungs and the kidneys without involving the loss of base. As the manufacture of these abnormal acids are being continued the depletion of the alkalies of the blood continues. Gradually the loss of the alkali reserve become appreciable and the CO_2 carrying capacity is brought down to a low level. Normal blood at 40 mm. CO_2 tension (alveolar air) has a combining power of CO_2 equal to 60 to 65 vols. per cent (60-65 vols.%). In diabetes, much lower values are obtained—sometimes going down as low as 10 vols.%. Due to the lowering of the CO_2 combining power of the blood the carbon-dioxide, formed in the tissues, are not properly eliminated and a condition of tissue asphyxia is produced. The respiratory centre is further stimulated producing hyperpnea and dyspnea. The depth of respiration is chiefly affected and becomes obvious when the alkali reserve is decreased to one-half its normal value. With the loss of salts by the kidney, Na and, to a lesser extent, K, Ca & Mg, decrease in the blood. Their excretion requires an increased elimination of water; hence, unless compensated by adequate intake of water, such polyuria leads to anhydraemia. As the acedosis becomes more severe dyspnea and typical "air hunger" develop. Coma marks the terminal stages.

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Laboratory Diagnosis of Diabetes Mellitus

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HISTORY

An ancient Egyptian record—PAPYRUS EBERS—is considered to be the earliest document on the subject of polyuria and the early authors considered this as the first mention of the disease diabetes itself. The name diabetes was given to this disease in the Grecio-Roman period and is derived from the Greek word *Diabatos*, suggesting a syphon. The scientists of those days were of opinion that flesh and limbs were melting down in the urine and were being exhausted as if by a syphon. The Chinese also observed the condition of polyuria very clearly and were the first to note that the urine was "so sweet that it attracted dogs" (Changki 229 A. D). Nearer home, in India, the disease was identified as MADHUMEHA as early as the 6th century. But though such a thing as sweetness of the urine in diabetes was known to many, yet to discover that it was due to a sugar and that it was glucose took some centuries. It took some more years again to correlate hyperglycemia with glycosuria. It was finally done by Claude Bernard (1813-1877). At the present day, scientific investigations have advanced to such a degree of accuracy that diabetes can be practically diagnosed by every physician, even before sugar appears in the urine. Yet with all the conveniences, many of the cases are undiagnosed in the early stages as there are no prominent symptoms which can arrest the attention of the patient himself to his ill-health. Majority of the patients come to know of their ailment quite accidentally by casual examina-

tions of their urine made for some purpose other than on grounds of health. Thus very often a patient comes to know of his condition when he goes up for medical examination for Insurance, for confirmation in Service etc. The result of the examination of the urine rudely brings the patient to understand the nature of his ailment and makes him sufficiently careful to watch and look for some of the symptoms which become quite clear to him thenceforward. In such cases periodical examination of the urine is of vital importance especially with regard to the diagnosis of diabetes. Examination of urine is a simple affair but the interpretation of the result of such an examination is fraught with difficulty. The mere presence of sugar in the urine does not warrant the conclusion that the patient is suffering from diabetes mellitus. Glycosuria with hyperglycemia should be looked for before one can venture on the diagnosis and the treatment of the disease. But this fact does not take away the importance of the first step of examination of urine.

URINE EXAMINATION

Fasting urine: When a patient is asked to bring a specimen of urine for examination, it is a common rule with practitioners to ask for a morning specimen. This is not based on any scientific principle. The morning specimen is asked for, probably because a patient can be expected to be in a fasting state early morning. But this is open to the fallacy that it is quite possible that a particular patient may not have voided his urine since his evening meals the day before and the urine passed next morning cannot therefore be considered to have been passed in a fasting state. So the importance is in the fasting state and not in the time of the day.

How to obtain urine for examination: Urine for examination can be obtained:

1. During the fasting state.
2. After food.
3. For the whole 24 hours.

The best method to get a specimen of urine in the fasting state is as follows: The first specimen of urine passed by the patient soon after rising from the bed should be discarded. A glass of water is given to the patient to wash out the urinary system and the urine obtained half an hour after this representing the washings is also discarded. A third specimen obtained after another half an hour is a good one for examination and represents fairly accurately the state of the urine in the fasting state.

(Continued from page 326)

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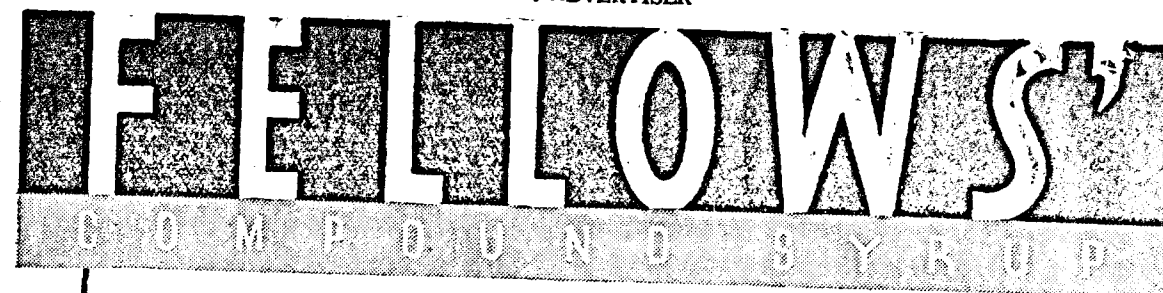
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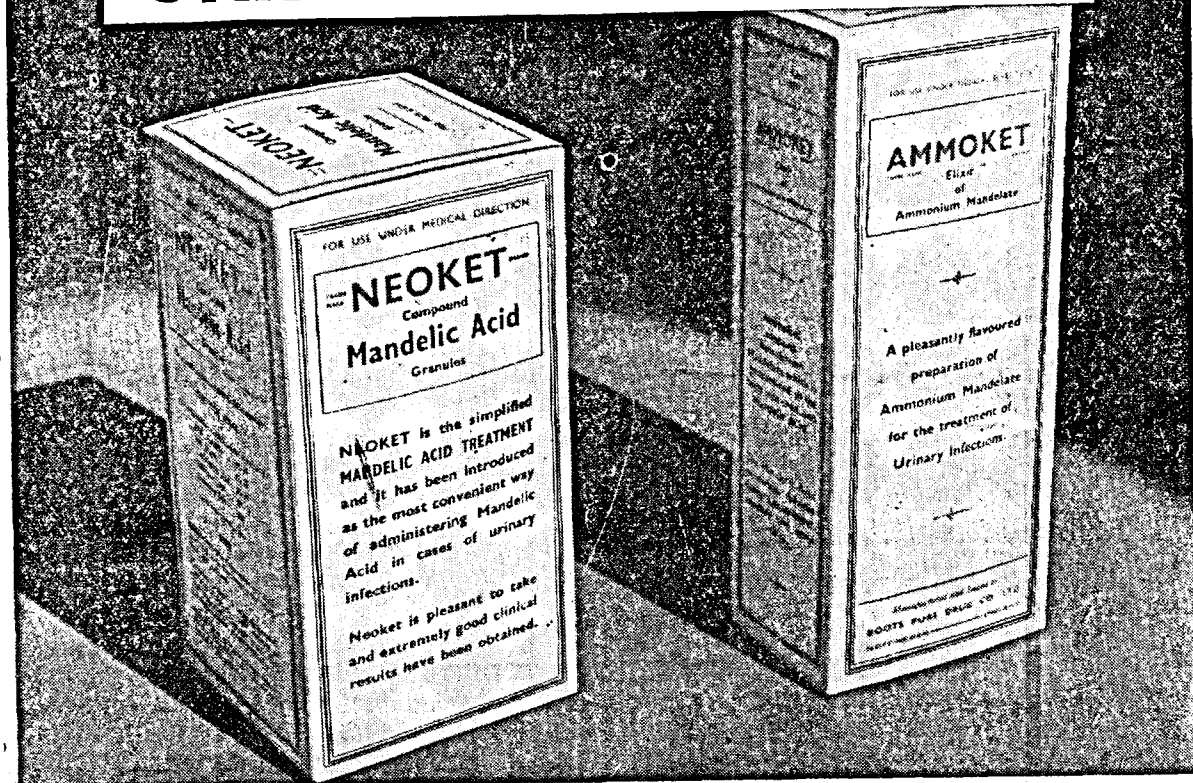
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Interpretation of Benedict's or Fehling's Test: Post-prandial urine can be obtained an hour or two after a meal and is the best for examination, and it is likely to contain all the normal and abnormal elements. The 24 hours specimen is obtained from a combined sample obtained from quantities passed from a fixed time in the morning of a particular day after the first specimen is voided to exactly the same time the next day. The two tests commonly used for the detection of sugar in the urine viz., Fehling's and Benedict's tests are well known and do not justify any elaborate description here. The chemical reaction on which the tests depend is the same in both the cases. The presence of sugar is detected by the conversion of the blue cupric oxide, which depending on various physical factors gives to the reacting mixture various colours from greenish blue to reddish brown. The reaction is expressed thus: $2\text{CuO} - \text{O} = \text{Cu}_2\text{O}$. Though most often we can conclude that this reduction is carried out by sugar present in the urine, yet, occasionally some other substances are also met with in the urine giving the same kind of reduction. Such substances are GLYCURONIC ACID, URIC ACID in excess, PHOSPHATES, HIPPURIC ACID etc. Benedict's solution is comparatively less affected by these than Fehling's.

The change of colour produced by the chemical reaction, when a specimen of urine is tested, is sufficiently clear, but occasionally, difficulty may be met with in the interpretation of result on account of a slight change in the colour. Sometimes there is also a delay in the formation of the precipitate. An opalescent mixture takes the place of the blue solution sometime after standing, though no change is observed while being heated. At other times instead of definite precipitate there is only a dirty greenish opalescence. Such anomalous results are no doubt puzzling and can be understood if we take into consideration the fact that though Fehling's and Benedict's solutions may be sensitive enough to detect one part of sugar in 12,5000 parts of water, yet are unable to do so if sugar is added to a normal urine and tested with these reagents. Thus very small quantities go undetected by these solutions. This is due to the presence of creatinine which has got the property of holding in solution the suboxide formed and thus preventing the precipitate. This is possible only with very small quantities. According to modern opinion even normal urine contains very small traces of sugar but they go undetected on account of the above mentioned reason.

As we are interested only in detecting the presence of sugar, over and above the normal traces the two solutions used for the tests are found to be best for our use. So when a doubtful change of colour occurs we are quite justified in assuming that a reduction has taken place on account of a small quantity of sugar which precipitates cuprous oxide and which again is kept in solution by creatinine and hence the modified colouration. This modification makes the precipitate appear as exceedingly nice particles floating in the liquid giving a dirty, milky or greenish appearance. Hence even the modified kind of precipitate can be taken to indicate the presence of sugar. Some investigators are of opinion that reductions are not definitely due to glucose. But recent opinion is that in the absence of definite indications to show the presence of other reducing agents one can easily conclude that a reduction, however mild it may be, is always due to glucose. But a clinician should have an eye open always for the possibility of other reducing agents being present along with glucose. To exclude some of these is fairly easy. For example, the precipitate due to phosphorous is coarse and flocculent and not crystalline and fine as glucose. Lactose which can produce a reduction can be expected in pregnancy or lactation. Galactose may be present in infants with digestive disorders. Glycuronic acid is usually found in combination with other compounds and is usually met with in patients taking coal-tar products or similar products by mouth. A careful study of the previous history, habits etc., of the patient can possibly help us to exclude many of the possibilities. Pentose is sometimes met with in the urine of a patient who has been taking much of grapes and other fruits. The best way to find out whether the reduction in the test is due to these is by discontinuing the administration of such things and testing the urine. There is one other very important point which I would like to mention in this connection, and that is that specimens of urine to be examined for sugar should never contain any preservatives like chloroform or formaldehyde as both reduce alkaline copper solutions. But fortunately the presence of these can be detected by their odours and hence mistakes are not likely though inconveniences and delay are possible.

Specific Gravity: Some practitioners attach much importance to the sp. gr. of the urine in diabetes. It is no doubt of value in cases where the reduction with Fehling's or Benedict's solution and the specific gravity

are studied together in comparison with specimens taken from the same patient at different intervals. Though generally a diabetic urine is of high specific gravity it is not uncommon to find a urine containing about 1% of sugar with a sp. gr. of 1008 or 1010. An increase of sugar by about 1% raises the sp. gr. by about 4. When the sp. gr. is high and over 25 and the reduction with Fehling's is very slight, it is better the urine is diluted and tested again, since the high concentration itself may produce a slight reduction.

Quantitative Estimation:—Quantitative estimation of sugar in the urine is dependent on the same principle as the qualitative test. The best and easy method is to do it with a stock Benedict's quantitative solution made according to the standard formula found in many of the text-books. The urine must be diluted to 1 in 10 parts. 25 c. c. of the Benedict's solution (quantitative) to which is added 10 gms. of sodium carbonate, is heated over a flame. When the solution is boiling the diluted urine is added drop by drop from a burette. When a chalky white precipitate is formed and the blue colour of the mixture disappears the dropping of the urine is stopped and the quantity of urine added is read in the burette. 25 c. c. of Benedict's quantitative solution prepared according to the approved formula will neutralize 50 gms. of glucose. From the quantity of urine added to neutralize 25 c. c. of the reagent we can easily calculate the quantity of sugar in 100 c. c. of urine. It is not enough for a practitioner to know whether there is sugar in the urine of a patient. Very often he has to obtain enough information from the examination of the urine of a patient to know whether any complications like acidosis and coma will set in and if so, when? Is it imminent or is there yet time for him to act? These questions can be answered to a certain extent by an examination of the urine for ketosis. The recognition of ketosis from urine is always by the well-known tests: Rothera's for acetone, and Jerhardt's for diacetic acid. The former is carried out by taking about 2" of urine in a test tube, saturating it with finely ground ammonium sulphate, adding a few drops of freshly prepared 10% solution of sodium nitroprusside and as much liq. ammonia fortis as the quantity of urine taken at first. If acetone is present a deep permanganate colouration develops. The depth of the colour and rapidity with which it appears indicates the amount of acetone present. The

test for diacetic acid is carried out by adding a few drops of 10% ferric chloride to some of the urine in a test tube, drop by drop, till no precipitate forms. The production of the portwine colour indicates the presence of diacetic acid. There is another easy test to know the existence of acidosis, that is Sellary's Test. Though it is not of value in nephritic cases, it is quite useful in ordinary cases and is very simple. Sodium bicarbonate administered by mouth in 5 to 10 gms. doses is usually sufficient to make the urine alkaline in a normal person. For one with acidosis a large quantity is required. This fact is made use of in the test. The suspected patient is given 5 gms. of sodium bicarb and urine passed half an hour after this is boiled, cooled, and tested with litmus for alkalinity. If the reaction is acid successive doses of sodium bicarb are given taking care to examine the urine as before till it is definitely alkaline. The quantity of sodium bicarb administered to make the urine alkaline gives an idea about the severity of the acidosis. Thus a mere examination of the urine done in a proper manner and interpreted well can give us a fund of information regarding the state of diabetic patient.

BLOOD SUGAR EXAMINATION

As mentioned before, glycosuria being the result of hyperglycemia the latter is the main point in a diabetic patient. In the diabetic the effect of ingestion of carbohydrates is different from that of a normal person. In a normal person, the passage of any sugar in the urine after the ingestion of very large amount of carbohydrates is prevented. In healthy persons with normal kidneys, even though the blood sugar level rises higher than usual after ingestion of large amounts of carbohydrates no excretion of sugar takes place. The normal blood sugar in the fasting state of person is between 0.08 and 0.12%. Till the concentration in the blood reaches 0.18% or thereabouts, no excretion of sugar takes place. This level, the highest to which concentration of sugar in the blood is allowed by the kidney, is said to be the threshold value. Over and above this (0.18%) it is excreted by the kidney and glycosuria sets in. It is very necessary therefore that in diabetes this hyperglycemia should also be taken into consideration when we study the glycosuria. In normal person with even very large amounts of carbohydrates it is impossible to get greater concentrations in the blood than the threshold value. When a patient with a disturbance in the renal threshold is excreting even the normal amount of

sugar present in the blood, then it is not a case of diabetes mellitus, but is purely a disturbance of kidney function and not of carbohydrate metabolism. In diabetes mellitus a patient with a normal renal threshold may suffer from glycosuria and the blood sugar is also raised to over 0.18% due to the disturbance in the storage mechanism. There are some cases of diabetes where the blood sugar concentration is just below the threshold value and hence no sugar is passed in the urine, but they still have to be considered as cases of diabetes as the blood sugar concentration is anyway higher than normal. So an estimation of the blood sugar with reference to a known carbohydrate at a particular time is quite necessary to form a correct opinion and to gauge the severity of the condition. A Sugar Tolerance Test is generally done to find the concentration of blood sugar before and after the administration of 50 gms. of glucose to the suspected patient. Such a test done gives a fairly accurate idea of the metabolism of carbohydrates of the patient. The test is done as follows:—

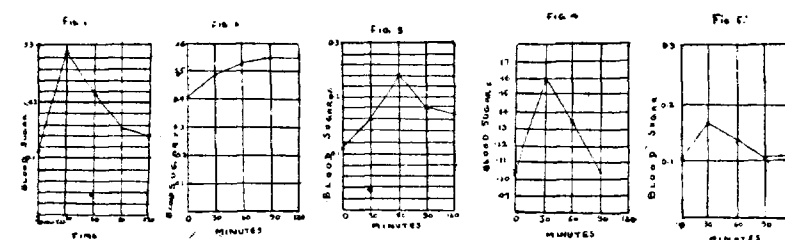
The patient comes to the laboratory in a fasting state. He is asked to pass water and a sample of blood is taken for sugar estimation. He then receives about 50 gms of glucose dissolved in 100 c. c. of water. Samples of blood are taken at intervals of half an hour for two hours while urine is passed at the end of each half hour. The nature of the blood sugar marked in the form of a curve taken in conjunction with the results of the urinary examinations afford enough information to determine whether the patient is a diabetic or not.

In a sugar tolerance test done for a normal person the blood sugar does not exceed 0.18% at the highest point of the curve and attains the original level of 0.1% in about 1½ to 2 hours. I am giving below the results of the Sugar Tolerance Test marked in the form of curves to give an idea about the degree of glycosuria in persons of varying ages.

Fig. 1 shows the curve in a middle aged man and shows markedly defective storage capacity for sugar which has to be regarded as suspicious of early diabetes. Fig. 2 is that of a man of 54 years with nearly about 4 to 5% sugar in the urine. The curve shows the case to be one of marked diabetes. Fig. 3 of a young man aged 25 years and the interpretation is that there is a marked disturbance in the storage capacity considering the young age of the patient and certainly indicates the diabetic condition. Fig. 4 shows the curve obtained from a patient with renal glycosuria. Note the peak of the curve which has not reached even 0.18%, the threshold value in an ordinary patient. But still this patient has been passing sugar in the urine. Fig. 5 shows the curve in an average normal person after administration of 50 gms of glucose and the Tolerance Test done as usual. It is given for comparison with the other curves.

There are some pathological conditions which may interfere with the test. In some cases of obesity a typical curve like that of diabetes is obtained and it has been observed to be so especially in elderly people. If the excess of fat is removed the glycosuria also disappears. Curves as that of diabetes may also be found in certain cases of the hyperactivity of the endocrinal glands. Patients suffering from boils and septic wounds are occasionally found to show evidence of hyperglycemia and glycosuria.

Barring these limitations, a Glucose Tolerance Test gives valuable information about the carbohydrate metabolism with special reference to the storage mechanism in the body and the kidney function in the excretion of sugar, and thus enables a physician to treat the patient on proper lines.



Clinical Diagnosis of Diabetes Mellitus

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THE PRIMARY NEED FOR A CLINICAL EXAMINATION OF THE DIABETIC

The characteristic disordered metabolism of diabetes mellitus has to manifest itself by some symptoms. The former is no doubt usually established long before the patient feels anything amiss. Very often the first intimation is the discovery of sugar in the urine in the course of an examination of urine during some other illness. But the detection of sugar in the urine alone does not in itself justify a diagnosis of diabetes mellitus. It is equally essential that a thorough physical examination should be made, even in patients who display obvious symptoms of the disease. If this is omitted the presence of secondary or associated infections like phthisis, oral sepsis, or any other focus of infection may be missed which may after all be aggravating what is really a mild diabetic condition. A careful consideration of the history and a thorough clinical examination are therefore essential pre-requisites to the subsequent investigation of both blood and urine. Each by itself is insufficient to establish the diagnosis.

EARLY SYMPTOMS

The first actual symptoms are usually the onset of unwonted lassitude and weariness, of thirst after meals, of passing too much urine (polyuria), of an increasing hunger and loss of weight in a person previously overweight as most diabetics are. These symptoms gradually increase in severity and others appear also. Two causes are at work in their production: (1) the failure to burn sugar and store it as glycogen and (2) the progressive poisoning by ketone bodies.

SYMPTOMS ARISING FROM GLYCOGENIC DYSFUNCTION

The failure to burn sugar and store it as glycogen leads to hyperglycaemia after meals. Thus the amount of sugar in the blood and tissues are increased. This is the cause of thirst. The tissues, especially the mucous membrane of the mouth, become dry (some patients describe it as feeling sticky or syrupy) and call for more fluid to dilute the excess of sugar. If the fluid drunk be milk or lemonade,

or another sweet substance, the concentration of sugar in the tissues becomes still higher, therefore thirst becomes worse and may be a terrible affliction. At the same time an excessive quantity of urine is voided, as the kidney excretes more water, partly in an attempt to get rid of the excess sugar and partly as a result of excessive drinking. The polyuria is proportional to the thirst and unless excess is drunk, it is only slight. These symptoms are rarely present until the blood sugar is over 0.25 per cent. In severe untreated cases the mouth is perpetually dry and the tongue becomes red, raw and sore. The excessive output of water leads to dehydration of the body, and this diminution of water contents produces a drawn appearance and often causes cramps in the legs at night. Constipation is an usual feature and is due to the dry condition of the faeces and the muscular weakness and sluggishness of the intestine. The skin also becomes dry and unhealthy, often itchy and is very liable to eczema, boils and carbuncles. Eczema and other cutaneous infections are liable to occur particularly round the genitals, where the skin is irritated by the sugary urine. This, in not a little number of cases, is often the first symptom to be noticed. Vast quantities of urine may be voided upto 10 litres (or 17 pints) in twenty-four hours containing as much as 500 grammes of sugar. The control of the urinary bladder is often affected and dribbling of urine results. This again is one of the first complaints which brings the patient to the doctor. The excessive loss of sugar sometimes produces great hunger. At first it begins as an increase of normal appetite, but becomes later ravenous. It is so experienced in the pit of the stomach, usually worst towards the end of a meal or felt as a constant empty ache encircling the body. When this is so, the pancreas may be found to be tender on pressure.

Failure to burn sugar as fuel more than even its storage as glycogen is the cause of gradual wasting, first seen in the body fat and then in the muscles. Muscular and general weakness gradually supervene, and there is marked aching and feeling of heaviness of the legs when going uphill or ascending stairs. The temperature also is frequently subnormal owing to the all-round metabolic depression. The patient finally becomes listless and depressed, and his temperature is greatly changed for the worse. This brings in its train lack of general resistance to other diseases. Tuberculosis, septic disease, boils and carbuncle find a ready prey in the untreated diabetic.

SYMPTOMS ARISING FROM PROGRESSIVE KETOSIS

The defective metabolism of fat and the accumulation of the poisonous acids (ketone bodies) give rise ultimately to the condition known as ketosis. This may lead to death by coma. Since acetone is excreted by the lungs the breath gives the characteristic smell of new mown hay. Acetone and di-acetic acid can also be detected in the urine by appropriate tests. The early symptoms of ketosis are mainly digestive: nausea, lack of appetite, vomiting, colic and abdominal pain. If a diabetic, who formerly had a healthy appetite, suddenly loses it the presence of a great degree of ketosis must be suspected. Giddiness, irritability and restlessness are other symptoms, but an unnatural drowsiness is much more common. Great breathlessness, apart from exertion, is a late but most serious symptom, the typical respiration being deep, heavy and gasping (air hunger). It is caused by the stimulation of the respiratory centre in the brain by the acetone bodies.

WHAT IS TRUE DIABETES

The common conception of the disease has undergone vital changes by the opportunity afforded by observation in children. Diabetes in a child is a pure diabetes, untainted by disease in the past and free from effects of the degeneration of old age. There are greater facilities in the case of children to observe the effects produced by the prolonged duration of the uncomplicated disease itself.

PRACTITIONER'S RESPONSIBILITY

Exaggerated importance is often attached to the need for frequent blood sugar estimations. Provided the diagnosis of diabetes mellitus has been established beyond doubt, it may be assumed that if the patient is passing large amount of sugar in the urine the blood sugar must necessarily be high. Except in diabetic coma or during insulin treatment there is seldom any necessity for frequent blood examinations unless to confirm when the urine is sugar free. Frequent examination for sugar and for quantity of sugar in urine, and for the presence or otherwise of ketone bodies, should have to be done by the practitioner himself while he is treating the diabetic by diet or in conjunction with insulin.

The urine should be tested at least four times daily with Benedict's solution and the result charted as follows:

Blue and clear	0
Green and turbid	+
Yellow and turbid	++
Red-brown and turbid	+++

Medical Emergencies and Treatment in Diabetes

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In this short paper surgical, ophthalmic, skin and other complications pertaining to diabetic mellitus have been omitted and only medical emergencies are dealt with.

The most serious dangers which threaten a diabetes patient are, coma, acute infections, severe nephritis and tuberculosis. Coma is due to the break down not only of the carbohydrate but also the fat and protein metabolism. When the fat does not undergo complete oxidation many intermediate products such as oxybutyric acid, acetone and diacetic acid are accumulated in the blood and tissues and excreted in the urine. Such a condition is known as 'acidosis' or 'ketosis.' Before these products appear in the urine we can find out their existence in blood and tissues by a few clinical signs which the patient exhibits. This condition may be called a pre-comatose condition where the unconsciousness is not complete, circulatory failure is absent, the patient can swallow and retain fluids and he responds to the directions given by the doctor. A few other clinical signs and symptoms like anorexia, abdominal pain,

Maintenance of such a chart will not only help in assessing the period of maximum amount of sugar during the day but also in assessing the degree of response to treatment. It is most important that in performing a Benedict's test strict attention should be paid to the details of the technique. It is particularly important that only eight drops of urine should be mixed with the re-agent and the mixture should be boiled for full two minutes.

In addition, tests for ketone bodies are of equal importance, because from the results some indications may be obtained as to whether there is any danger of diabetic coma or not.

The general practitioner will be well-advised to assume himself this minimum laboratory responsibility to arrive at correct diagnosis, to know the degree of severity of the case, to assess prognosis and to gain confidence in the treatment he is giving. After all, urine analysis is so simple and easy that the clinician should not abdicate this work to his laboratory colleague.

sometimes associated with diarrhoea and a dry skin are shown. It is at this stage, if efficient treatment is carried out, one can combat these complications very successfully. It is often necessary that the doctor in charge of a diabetic patient should not brush aside these symptoms which the patient informs as being minor but should treat them energetically.

Treatment of a case in pre-comatose condition.—When the diagnosis is established that the patient is suffering from ketosis an injection of 30 to 60 units of insulin and about 50 grams of glucose or cane sugar by mouth is all that is necessary to make the patient recover. When sugared water is retained large quantities of water, soda, tea, and sodium bicarbonate (2 to 3 drachms for a day) may be enough. One could expect the patient to recover from the pre-comatose condition within a day or two. The blood sugar in this pre-comatose condition may be found to be over 8% and if it is found higher it may be often necessary to increase the dosage of insulin from 50 to 100 units from the beginning. It is strange that though in a case of diabetic coma the blood sugar is invariably very high yet it has no definite relationship whatsoever between the degree of hyperglycaemia, acetone bodies in the urine and the degree of loss of consciousness in the patient. I have found diabetics without getting into coma having a blood sugar of over 15% and with just a trace of acetone bodies. But the converse, i. e., acetone bodies is found in large quantities though the blood sugar is less, is the rule. The determination of the total acidity of the urine and the ammonia co-efficient (the relation between the urinary ammonia and the total nitrogen) although gives valuable information are yet often found less valuable in the management of the cases. The determination of carbon-dioxide combining power (alkali reserve) and the estimation of carbon dioxide in the alveolar air are also valuable guides in predicting the severity of the case. Administration of glucose with the first dose of insulin in pre-comatose condition is a controversial point. I hold it is always wiser to allow hyperglycemia to go on than to produce hypoglycemia. The risk of hyperglycemia is not very great and cases of coma can be restored without glucose but never without insulin. The advisability of giving large doses of sodium bicarbonate to restore the normal alkaline reserve seems to be also controversial. I find the use of sodium bicarbonate is not essential for recovery.

Coma.—The most common cause which determines the onset of coma in a case of diabetes is an acute local or general infection of some kind which breaks down the patient's carbohydrate metabolism. A straight-forward case of diabetic coma usually presents no difficulties in diagnosis. But it is always good to keep in mind the possibilities of other diseases causing unconsciousness. A diabetic patient suffering from middle ear disease, as in many normal individuals, develops meningitis and becomes unconscious or he might suffer from acute uraemia due to anuria or the patient might have taken an over-dose of insulin. It is a common experience that old diabetics in India are somehow addicted to opium habit. On account of opium, the patient may become unconscious, the urine may give a positive reaction with ferric chloride giving us a false impression of diabetic coma. Therefore it is essential that the diabetic coma should be recognised before energetic treatment with insulin is carried. In pre-insulin days diabetic coma was always regarded with the greatest apprehension and fear and whatever lines of treatment are chalked out were an utter failure. After the advent of insulin there has been a complete change in the outlook and the majority of cases of coma have the best chance of recovery if the following line of treatment is carried out.

First, the patient is put to bed and treated on general lines for toxæmia and collapse. Digitalis, caffeine, and camphor are some of the useful drugs. If there is respiratory or circulatory failure coramine and adrenaline can be used.

Second, if the constipation is severe and if there is no collapse a soap and water enema should be given to ensure a thorough evacuation of the bowels.

Third, in cases of severe dehydration intravenous injections of normal saline is given to flush out the toxins. In cases with feeble pulse and severe collapse normal saline can be combined with glucose (10%) and given intravenously. Subcutaneous or rectal saline can be given if there is not much collapse. In cases of severe dehydration and collapse 7% of gum-acacia solution is added to the glucose-saline solution, and given intravenously. As soon as the patient can swallow water, tea or coffee can be given. Alcohol has no place in the treatment of collapse in diabetic coma.

Fourth, Alkalies in the form of 2 drachm doses of sodium bicarbonate has to be given every second hour until the urine becomes alkaline. They have to be stopped as soon

as the air hunger stops or the urine becomes alkaline. Injections of insulin (30 to 40 units) either subcutaneously or, if the case is very serious, intravenously can be given. The next dose of insulin has to be regulated very cautiously in accordance with the findings of urinary sugar, acetone bodies and blood sugar. As the condition improves the dose of insulin can be reduced.

Fifth, a careful examination of the sources of sepsis is made. Sepsis in the ear, nose, and throat should be dealt with, if found, under local anaesthesia. These probably might have been the immediate causes of coma and if not dealt with promptly the efficacy of insulin may be diminished. It is often my experience that cases of coma can be divided roughly into 2 groups:—1. The largest of the group, coma without anuria; and the other, the small group, coma with anuria. High blood urea occurs either at the beginning or later which always proves fatal. It is in these cases that it is very difficult to regulate the dosage of insulin, as urine is not available for examination even by a catheter. Blood sugar estimations are the only means by which one can have an intelligent guidance in carrying the treatment. In its absence, 50 grams of glucose and 25 units of insulin can be given empirically every third or fourth hour. The guidance for administration of large quantities of fluids is the observation of the tension of the eyeball. Under my care I had a case of an old diabetic for over 25 years and in a pre-comatose condition developing violent tremors of the hands, twitching of the lips and facial muscles. Administration of calcium gluconate in 10% solution of 10 c.c. intravenously controlled dramatically the tremors.

The relative value of alkalies in the treatment of acidosis, according to Joslin, is as follows:—

1. Cases treated without alkalies gave much more satisfactory results than the group of cases treated with alkalies.
2. When alkalinity of the blood is brought to normality coma was found to persist.
3. Alkalies upset the stomach which is not desirable in diabetic coma.
4. Large doses of alkalies in a few bad cases have led to convulsions.

Haldane has demonstrated that large doses of sodium bicarbonate given to normal individuals living on normal diet, the tolerance for glucose was diminished and acetone bodies appeared in the urine. In mild cases of acidosis sodium citrate is found to be much more effective in not only controlling vomiting but also making the urine alkaline. The use

of alkalies by intravenous method is to be condemned as they are often dangerous.

Cold and Influenza.—These increase the severity of the diabetes to a remarkable degree. In children particularly, I have seen a case developing acidosis and later coma. Any infection lessens the action of insulin, and hence these minor maladies are to be treated promptly.

Neuritis and Neuralgia are other complications in diabetes. Diabetic neuralgia may affect any nerve but the most commonest forms are brachial, sciatic and intercostal neuralgias. These are much more painful than the ordinary forms. They are often symmetrical and they do not yield to treatment readily. It is often found that they persist even after the blood sugar is brought to normal.

Anti-diabetic treatment should be followed by a judicious administration of diet and insulin. For the relief of pain, hot fomentation and antineuralgic drugs may be prescribed.

Diarrhoea.—This is generally caused by the patient taking an unaccustomed food which contains plenty of cellulose. The usual treatment is abstention from all vegetables for a little time. When the gastric upset is corrected the patient may be allowed to gradually return to the normal diet.

Pneumonia and other acute infections.—The onset of pneumonia in a diabetic is always serious. But after the introduction of insulin the condition is not so grave. Sufficient insulin should be given every 4 hours to lessen the blood sugar. By so doing it is possible to avert coma. The diet is given in moderation, as milk, eggs and gruel. M & B 693 is often found very useful in the treatment.

Tuberculosis.—This is one of the most dangerous complications—unfortunately a fairly common one in all neglected cases. It is insidious in its onset and hence elusive to correct diagnosis. The diabetics have often a low body temperature, sometimes one or two degrees below normal. So, with the early onset of tuberculosis the rise of one or two degrees of temperature may not be easily found out. Troublesome cough and loss of weight may be the only symptoms of a pre-tuberculosis condition. Hæmoptysis and pleuritic pain are often the manifestations of an advanced stage of tuberculosis in a diabetic patient. Therefore, it is always wise that a diabetic should be examined carefully for tuberculosis by modern methods.

Bright's disease is often associated with senile diabetics. Fortunately the diabetic diet is admirably suited for the cases of nephritis.

(Continued at foot of next page)

Surgery in the Diabetic

● A. Viswanathan, L. M. P., Madras

THE NEED FOR SURGICAL INTERFERENCE

The need for surgical interference may arise either as a result of diabetes mellitus itself or of associated surgical disease which call for urgent treatment. Thus we may meet with two types of cases: those in which surgery has to be undertaken owing to some complications of the diabetic state, and those in which surgery is considered necessary notwithstanding the diabetes. The first may again be subdivided into cases in which the diabetic condition is aggravated by other metabolic disturbances and those in which such local conditions such as sepsis and trauma are aggravating the disease.

Before Banting's discovery of insulin surgical interference in a diabetic was a much dreaded affair, and more often than not the survival of the patient was a result of chance and not of conscious design. In the post-Banting era, however, survival of the diabetic after a serious surgical operation can be designed in reasonable hope of success; because the diabetic conditions, such as glycemia, hyperglycemia and ketonemia, can all be controlled by a judicious combination of diet and insulin.

(Continued from previous page)

Albuminuria and hypertension are not contra-indications to the use of insulin.

Neurasthenia and sexual impotence:—Recently, in the author's experience, a case of diabetes of 24 years with all secondary sex characters showed a high blood sugar content and a high urinary sugar. Testicles were very small. The administration of very large doses of insulin did not appreciably reduce the blood sugar. Ketone bodies were also found in the urine. Whenever an attempt is made to reduce the blood sugar with insulin ketone bodies were found on the increase. After a few injections of a testicular extract the patient improved remarkably in his mental outlook. Correspondingly a lesser dosage of insulin was given to control the urinary sugar. This observation was in accordance with Dr. Venkatachalam Pillai and Ratnagiriswaran's findings that gonadal extracts reduce blood sugar.

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Although surgery in the diabetic is now a less dreaded affair, yet it has to be recognised that insulin by prolonging the life of the diabetic has exposed it to surgical peril greater than in a normal healthy individual chiefly because of the greater risk of infection and associated arterio-sclerosis in the former.

THE NATURE OF SURGICAL INTERFERENCE

It may range from a trivial to serious operation. A small injury may become septic with explosive suddenness and spread so rapidly as to necessitate amputation of a limb, or any part of a limb, particularly the toe may become so suddenly gangrenous, even without any external injury, as to need a high amputation. A carbuncle may be both simple and serious, depending on the virulence of the infecting organism, the resistance of the patient, and the extent and depth of the local necrosis. The nature of interference in a case of carbuncle therefore depends entirely on the successful control of the diabetic condition by diet and insulin, and the time taken to achieve such a control. If such a time is short enough not to allow an upper-hand to bacterial mischief, the only surgical interference needed would be magnesium poultices and probably an autogenous vaccine. If such a time is too long during which bacterial danger is greater than the diabetic danger, the nature of surgical interference would be incision or excision of the carbuncle according to the severity of the local necrosis besides the continuance of measures against hyperglycemia and ketonemia. It would be dogmatic to say, as some do, that every case of carbuncle can be treated without surgery, or that surgery alone is the earliest and the surest way of treating it successfully. Truth is always between two dogmas on a particular subject, and so it is in carbuncle. The need for surgery or otherwise should be always arrived at by the merits of the individual case taking all factors, such as the duration of hyperglycemia and ketonemia, the degree of control attainable by combined insulin and diet treatment, the age of the patient and the virulence of the infecting organisms, into consideration. All shibboleths are dangerous, particularly in the case of a diabetic where the unexpected may always happen.

A word of caution is necessary. All carbuncles are not diabetic although glycosuria, or even glycaemia, may be present. It is now well known that pyogenic infection, acute or chronic, local or general, may itself induce glycemia and glycosuria. In which case, the most sensible treatment would be to eradicate

the infection by surgical means combined with or without auto-vaccine therapy. But if infection is a supervention on an already existing diabetic condition, the most sensible treatment would be to bring the latter under control and deal with the former surgically if necessary.

The surgical affections incidental to the diabetic are abscesses, quickly infected traumas, carbuncle and gangrene and these are, as mentioned earlier, due to low resistance which in turn is due to imbalance of carbohydrate metabolism characteristic of diabetes mellitus.

But there are surgical affections to which the diabetics are as equally liable as the non-diabetics. They are strangulated hernia, acute appendicitis, gall-stones or, for that matter, any ordinary surgical affection which call for usual or urgent surgical treatment. Here the question is whether operation could be performed safely on the diabetic? And what is the choice of your anaesthesia? And what are the pre and the post-operative precautions which the surgeon has to take before he uses his knife, the cautery and the forceps?

PREPARATION OF THE PATIENT

Although every diabetic patient is a real surgical risk, that risk should, and could, yet be minimised by a careful preparation of the patient before and after the operation. If the operation is not urgent the condition of diabetes must be treated in the usual manner taking care not only to considerably reduce hyperglycemia and ketonemia but also to improve the general health of the patient by putting him on a balanced diet for sufficient length of time. If, however, the operation is urgent and cannot be postponed, the patient should be given sufficient glucose and insulin, preferably protamine zinc insulin to maintain a continuous supply, and then only should the operation be started.

Under no circumstance, however, the diabetic should be starved and purged according to the usual pre-operative procedure, because they will result in hypoglycemia and dehydration which are as dangerous as hyperglycemia and polyuria.

The post-operative management is of definite importance, and is aimed to avoid the too much dreaded dangers, viz., dehydration and ketosis. Glucose by mouth or by the vein, alkaline transfusion and regulated doses of insulin may all become necessary. The aim however should be to bring the patient as early as possible to normal diet and normal dose of insulin.

The time taken for a surgical operation in the diabetic should be shortened as far as possible by a dextrous rapidity of manoeuvres. It is equally imperative to minimise surgical trauma.

CHOICE OF ANAESTHETICS

This is the crux of the problem in surgical diabetes. It may be stated generally that any anaesthetic will injure the liver to some degree or other, and any such extra injury to it, already in a state of partial dysfunction due to disordered carbohydrate metabolism, has to be reduced to the minimum if it could not be avoided.

The anaesthetic of choice in the diabetic is either spinal or local anaesthesia without the usual adrenalin, since adrenalin antagonises the action of insulin. Spinal anaesthesia should be resorted to only for operations below the umbilicus.

Of the inhalation anaesthetics, chloroform has to be tabooed, because it impairs the functions of the liver more than any agent. Nitrous oxide and oxygen is the anaesthetic of choice among the inhalants.

Pre-medication with barbiturates lessens the risk of anaesthesia by enabling us to decrease the dose of anaesthetics, but it must be used with caution and in selected cases.

It is very difficult to recommend confidently the use of recently introduced anaesthetics such as avertin, ethylene and cyclopropane. The writer is of opinion that these should not be indulged in by the general practitioners. The decision of choice and the use of these anaesthetics must be left to the specialists in anaesthetics. So far as the writer is aware, the utility of these is based on a very limited experience.

There is one factor in surgical diabetes which is well worth remembering. Wounds and ulcers in the diabetic are not so painful to him as it is to the normal individual. Hence a relatively less dose of anaesthetic would be sufficient. The writer has done large excisions of carbuncle with a minimal dose of local anaesthesia with absolute freedom from pain to the patient. This is more so in those who are past middle age.

PREVENTION OF SURGICAL DIABETES

Leaving aside such surgical affections which are not directly or indirectly due to diabetes mellitus, surgical diabetes is better prevented than cured. Surgical diabetes is mostly, if

not always, the result of neglect in getting oneself treated, or of imperfect and injudicious treatment, and of want of care against accidents and infection. Therefore the methods of prevention are: (1) proper and early treatment and (2) exercise of great caution on the part of the diabetic against wounds and infection. Heavy exercise, irregular life, and intemperate habits are undoubted dangers to the life of a diabetic.

SOME PITFALLS IN DIABETIC SURGERY

They may be indicated briefly. The surgeon should as far as possible determine beforehand whether glycosuria is the result of surgical affection or whether the latter is due to the former. He should permit himself no naive satisfaction by unwarranted presumptions and hurried examinations. He must make himself sure of the ground he has chosen to tread.

When to operate the patient is a question, which if not determined with previous study of the case and necessary caution, is likely to land the surgeon and his patient in irreparable damage. It is always wise to consult the physician in these matters.

There is one condition in diabetes called *abdominal cramps* which is due to severe acidosis and which may show such symptoms as abdominal distention, rapid and small pulse, vomiting, dry tongue, pain, spasm, tenderness and rigidity of abdomen, nausea and vomiting. This has often been mistaken for some acute abdominal condition, and a diagnosis of acute appendicitis, perforation and peritonitis has been made wrongly. The surgeon should guard himself against this dangerous pitfall.

Antiseptics, strong or mild, should be avoided in surgical diabetes as they destroy the cells too rapidly in the diabetic patients. Only saline or mag. sulph or sodium sulph. dressings or irrigations should be resorted to. They do not traumatise the cells, help healing, and even if absorbed by the tissues are beneficial to the patients.

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Diabetes Mellitus

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Diabetes mellitus is a constitutional disease characterised by the passage of large quantities of urine containing sugar.

SYMPTOMS

1. The urine is abundant and may amount to even 20 pints a day; clear, pale, but of high specific gravity, 1030 to 1050.
2. Excessive thirst and a dry tongue, which may become raw and beefy. Pyorrhoea is quite common.
3. Loss of weight.
4. The appetite is normal or even excessive.
5. The skin is dry and harsh due to the dehydration of the subcutaneous tissue. Sweating is usually uncommon unless when tuberculosis co-exists. The skin is unhealthy and is very susceptible to infections such as boils, carbuncles etc. Eczema is common round the genitals, especially in women, due to irritation of the skin by the sugar in the urine.
6. The blood sugar is above the normal.
7. Obstinate pruritis, generalized or localized, specially in or about the private parts, may also lead to diagnosis of this disease.
8. Loss of sexual desire or even actual impotence may often be a symptom.
9. Complete cessation of the monthly periods in women at an age when it is not expected, leads to the diagnosis of diabetes.
10. In chronic cases of diabetes, myocarditis, arteriosclerosis and nephritis are common.
11. General symptoms such as lassitude, progressive weakness and ready fatigue are always present.

VARIETIES

There are two varieties.

- (a) A mild form in middle aged people where the symptoms are moderate and dietetic restriction removes the sugar from the urine.
- (b) The severe variety assumes acute and chronic forms. The acute form usually occurs in children or young adults; and occasionally after head injuries. The chronic form is met with in older people and is attributed to mental worry.

ÆTIOLOGY

- (i) *Nature of food*: Nature of food has to do a lot in the cause of diabetes. Over eating and indolent habit bring about strain on the liver and pancreas resulting in diabetes. So

long as the liver retains the power of converting glucose into glycogen or the combustion of sugar in tissues remains normal, the person does not suffer from diabetes. But, sooner or later, liver and pancreas give away with the result of occurrence of sugar in the urine.

(ii) *Heredity*: It is usually believed that diabetes is a hereditary disease. There are many families in which diabetes can be traced to run back for three or more generations.

(iii) *Obesity*: The association of diabetes with obesity is quite a common finding in a large proportion of cases.

(iv) *Age*: Majority of the cases occur between the age of 35 and 45; but no age is exempt.

(v) *Sex*: Males seem to suffer more from diabetes than females.

(vi) *Race*: Certain races are more prone to this disease than others. Jews seem especially liable but it is probably their sedentary habits and rich diet which are responsible. It is less common amongst the Chinese, the Japanese and the Negroes. In India, the disease is extremely common in Bengal, specially among the rich and the educated classes.

(vii) *Climate*: It is believed that tropical and subtropical climates lower carbohydrate tolerance and diabetes mellitus is more severe in hot regions than in the cooler and hilly cold places.

(viii) *Lack of physical exercise*: Exercise increases the self-production of insulin in mild cases of diabetes but is unable to do so in severe cases. Exercise helps oxidation of the carbohydrates in the muscles. With exercise and consequent burning of carbohydrates, the secretion of insulin is apparently augmented and this helps to lower the blood sugar. Strenuous exercise should, however, be avoided in severe cases of diabetes lest it induces acidosis.

(ix) *Social position and profession*: Diabetes is very common among well-to do educated persons, especially among landlords, lawyers, merchants, physician and clergymen.

(x) *Nervous origin*: Excessive mental strain, emotion and constant worry are responsible for diabetes. Sudden shock as death in family or sudden failure in business are causes of diabetes of an acute type.

(xi) *Insanity*: It is the common experience in the investigations of mental disorders to find the presence of glycosuria.

(xii) *Infection*: Infection of any kind may prove to be a direct cause of diabetes, probably owing to some kind of damaging

effect on the pancreas by the toxin, such as pyelitis, cystitis, urethritis and syphilis.

(xiii) *Trauma*: Injuries to the head and spine may bring about diabetes.

(xiv) *Internal secretions*: Hyper secretions of the thyroid, suprarenal and pituitary glands may produce hyperglycemia and glycosuria.

COMPLICATIONS OF DIABETES

The most serious complications are coma, gangrene, big carbuncles, severe nephritis and tuberculosis.

DIAGNOSIS

Always examine the urine of a patient suffering from boils or eczema of the genitals and apparently causeless wasting.

PROGNOSIS

Prognosis chiefly depends upon the complications. Insulin has made a huge difference in the prognosis. It has not only helped to make diabetic life comfortable, but has also prolonged it.

TREATMENT

Diet: Diet is more important than medicinal treatment. In all diabetics the power of assimilating sugar is more or less diminished, it is therefore important to limit the ingestion of carbohydrate food. The rule is to forbid it as far as possible and to advise a diet of meat, fish, eggs and green vegetables. Too great quantities of meat should be avoided.

Insulin is the sheet anchor in the treatment of diabetes, but it must not be forgotten that insulin has its uses and abuses too. The patient undergoing insulin treatment should be clearly made to understand that insulin is only a valuable adjunct to the dietetic treatment. To get the full benefit of insulin treatment, there must be a constant relationship between the dosage of insulin and the quality of the diet allowed. Under no circumstances should insulin be given in large doses. No doubt a large dosage of insulin with corresponding carbohydrate food increases the strength and weight of the patient but it is apt to cause further damage to the already damaged pancreas.

Indications for Insulin: 1. Insulin should be injected in all cases where there is prognosed hyperglycemia.

2. All cases of diabetes with complications such as coma, tuberculosis, infections, neuritis, carbuncles, cellulitis, gangrene etc. and

3. Diabetic children and persons below the age of 40 require insulin treatment.

Dosage of Insulin: There is no fixed dosages of insulin and the dose varies accord-

(Continued at foot of next page)

Diabetes in Children

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HEREDITY AND DIABETES

Very little mention of diabetes in childhood is found in the literature prior to 1922, as the duration of life of a diabetic child was so short and at best could be counted two to two and a half years under the best regimes. Osler in his PRINCIPLES AND PRACTICE OF MEDICINE as late as 1917 under *prognosis in diabetes mellitus* writes as follows:

"the younger the patient the less likely is recovery..... In children the disease may run a very rapid course, and death may occur within a few weeks, or, indeed a child may die in coma before the condition has been recognised..... Personally I have not known an instance of recovery in a child."

The discovery of Insulin in 1922 changed the prognosis so fundamentally that hopes could be reasonably entertained now, and the day is not far, as a result of further research, when eventual arrest of the disease may

(Continued from previous page)

ing to the severity of the case and the complications. In mild cases, one injection of 15 or 20 units of insulin before food is enough. If larger doses have to be given, it is best to give into two doses, the large doses be given before the evening meal. In diabetic coma, 50 units of insulin may be given at once, followed by large doses till the patient shows definite signs of improvement. In surgical complications, the quantity of insulin should be gradually increased.

DRUG TREATMENT

Antipyrin is used in certain cases of diabetes in which the production of sugar is very great. Salicylate of sodium also has been of service; its action is similar to that of antipyrin. Quinine acts in the same way as antipyrin and the salicylate of sodium, and has got the advantage of being a tonic. Opium reduces both polyuria and glycosuria. Codein is generally used in $\frac{1}{4}$ gr. thrice a day, but the disadvantage is that it forms a drug habit. Jambul possesses only a feeble antiglycogenic action.

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become possible. This welcome change in the prolongation of the life of the diabetic child is not only a factor which benefits suffering children alone, but also affords workers in the diabetic field to better evaluate the conception of the underlying causes of diabetes.

Diabetes in the child represents *true diabetes*, pure and simple, whereas adult diabetes has some background either of obesity, diseases of thyroid or some gall-bladder condition or any other intercurrent affection which may complicate or vitiate the true diabetic picture. The opportunity afforded by the observation of the incidence and progress of diabetes in childhood reveals the possibility of its being an inheritable disease. Recording and rechecking of the family history of the child-patients discloses the fact that 40 to 45 per cent of them have blood relatives with diabetes. Heredity, therefore, may be said to be one of the factors predisposing to diabetes.

THE PHYSIOLOGY OF DIABETES IN CHILDHOOD

Carbohydrate metabolism.—In the normal infant and young child it is characterised by a lower level of blood sugar, probably due to greater utilization of carbohydrate in the system, and consequently less amount of glycogen stored in the body. The diabetic child has a low level of fasting blood sugar. After a couple of years' duration of the disease the blood sugar is elevated in the fasting state as well. These cases respond well to insulin therapy.

Fat metabolism.—Fat metabolism assumes a greater importance in the child as against the adult, and excess of cholesterol in the blood rather than hyperglycemia or acidosis is the precursor or index of complications of juvenile diabetes.

Basal metabolism.—This bears a direct proportion to the blood sugar level—higher the blood sugar higher the B.M.R. Puberty is often established late in diabetic children.

Growth and development.—The rate of growth and development should be normal and any deviation indicates an unsatisfactory control of the disease. In a given case of failure of growth, though the ulterior cause may perhaps be found in the hypofunction of the pituitary with lack of growth hormone, mostly under-nutrition is the apparent cause in many cases. In the pre-insulin days under nutrition was essential to maintain life. Even the child of the insulin era fails to grow when reduction in diet rather than increase in insulin is the method of treatment employed to produce a sugar free urine. Defective

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30th Conference of The All-India Medical Licentiates' Association Change of Venue from Ahmedabad to BOMBAY Three Days' Session : 22-23-24, December 1940 President-Elect : Dr. A. D. Mukharji

Since Ahmedabad Reception Committee has finally informed, in spite of repeated appeals, that it is impossible for it to hold the Conference on the previously announced dates, 22nd, 23rd, and 24th December 1940, and since it cannot be postponed to next Easter, as desired by Ahmedabad, for the reason that there was no Conference during last year, I as the President of the Association, in its interests, has perforce to request the Bombay Branch to urgently arrange for and hold the Conference on the 22nd, 23rd and 24th instant, at Bombay. Bombay Branch has magnificently responded to this call of Duty. The Conference will therefore be held at Bombay, and all the delegates are requested to note this change of venue and reach Bombay by 21st evening. The time of their arrival may kindly be communicated to : Dr. U. B. NARAYAN RAO, 1, DAMODAR MANSIONS, NEAR OPERA TRAM TERMINUS, GIRGAUM ROAD, BOMBAY 4.

Dated Madras,
5th Decr. 1940.)

(Sd.) D. V. VENKAPPA,
PRESIDENT,
All-India Medical Licentiates' Assn.

14th Session of the Medical Council of India

[We have only one comment to make on Dr. B. C. Roy's presidential address, and this is in reference to Para 20 of the same. We will pass without any remark on his observations that the critics of the Council are mostly uninformed and that their criticisms are more destructive than constructive. He and his Council may disregard them, but, we believe, this attitude cannot continue for long. We however, thank him for having chosen to deal with at least one such criticism, viz., that the Council has failed to fulfil the established purpose of "SECURING EFFICIENCY AT HOME AND HONOUR ABROAD". We are glad that he has taken the public into his confidence and told them openly that, at any rate, "HONOUR ABROAD IS AT A DISCOUNT", while he however is sure that "EFFICIENCY AT HOME" was undoubtedly secured by the Council so far as the University Medical education was concerned. But may we ask: Was the University Medical Education before the Medical Act of 1933 so inefficient that the Council should take credit for its present efficiency? We are not loth to admit that the Council has effected uniformity in the standard of University Medical Education, and probably made it a trifle more efficient than what it was before 1933. But that the Council should take credit for having secured "EFFICIENCY AT HOME" when the larger field of Medical School Education has been deliberately left out of the purview of the Act is something which baffles our understanding. Probably we are one of those critics who are uninformed.]

—Ed., I. M. J.]

The Indian Medical Council held its 14th session on 26th Oct. '40, under the presidency of Dr. B. C. Roy. In the afternoon session, after full two hours' debate, Dr. K. S. Ray's resolution for setting up a uniform standard for medical study in the medical schools was adopted by an overwhelming majority.

Another resolution recommended that provision should be made in the Indian Medical Council Act, 1933, for an All-India Medical Register. The register would include (i) all persons who hold qualifications borne on schedules to the Indian Medical Council Act, 1933, and (ii) all persons who are practising the modern scientific system of medicine and who possess qualifications granted by examining bodies in British India other than the Universities on December 31, 1947, and whose names are on the register maintained by the Provincial Councils.

The recommendations of the Executive Committee to move the Central Government to take early steps to implement the proposals and also to ask the Provincial Governments to abolish the

medical schools for Licentiates in their respective provinces or raise them to university standard, so that there would in future be only one uniform minimum standard of medical education for the whole of British India, were also adopted.

The Council also elected Dr. B. N. Prasad of Patna, Dr. Kuttumbiah of Andhra and Dr. Jivraj Mehta of Bombay to the Drugs Technical Advisory Board in accordance with the provision of the Drugs Act.

Another resolution of Major-General G. G. Jolly recommended to the Council that pending amendments to the present Act, the Council should have an All-India register and frame its own rules governing registration which would be applicable to the whole of India and the Government be moved to take necessary steps by enactment or otherwise to ensure that so far as qualifications granted by licensing bodies outside British India are concerned, the Provincial Medical Councils will recognize and register only such qualifications as are included in Schedule II of the Indian Medical Council Act.

DR. B. C. ROY'S ADDRESS

Members of the Council,

We welcome to our deliberations Dr. Lazarus, who has been nominated by the Central Government in place of Dr. Ruth Young, so also Dr. Satakopan who has been re-elected to this Council as the representative of the Registered Graduates of Madras.

2. Since we last met your Executive Committee have considered the Inspectors' Reports on the courses of Instruction and Examinations in the subjects of Anatomy, Physiology and Pharmacology of the various British Indian Universities together with the observations of the Universities thereon and the General Reports drawn up by the Inspectors. The Committee's observations on these Reports with their recommendation are being placed before you.

3. The Executive Committee decided that the detailed inspection of the courses of Instruction and Examinations in the subjects of Pathology, Forensic Medicine and Hygiene should be made; they have appointed Inspectors to carry out these on behalf of the Council.

4. The Executive Committee have directed that information collected regarding the teaching of Hygiene and Public Health handed over to the Inspector they have appointed to inspect the courses of Instruction and Examination in the subject of Hygiene in order that he might find out to what extent the various Institutions in India have developed Health Museums. He will also draw the attention of the authorities to the importance of the preventive aspect of medicine throughout the medical curriculum to which reference has been made in the Council's Recommendation No. II (3) on Professional Education.

5. At the instance of the Executive Committee the Universities were asked whether they had any rules, convention or system prescribing minimum qualifications for the appointment of teachers and examiners. The replies received are being placed before the Executive Committee for their consideration.

6. The Executive Committee considered further communication from the Central Government in connection with the non-recognition of the L. M. S. qualification of the Madras University by the Federated Malay States; they drew the attention of the Government to the resolution of the Executive Committee of the 24th October, 1938. Our reciprocity with the Federated Malay States is through the General Medical Council which recognises the L. M. S. of the Madras University only if its holders began professional study before January 1, 1892. This degree ceased to be granted since 1935. No steps can be taken, therefore, for its retrospective recognition in view of the arrangement with the Federated Malay States.

7. Under the direction of our Executive Committee the draft requirements in the Basic scientific subjects formulated by the Board Experts appointed by the Committee were forwarded to the various British Indian Universities included on the First Schedule to the Indian Medical Council Act, 1933, for their views. So far a reply has been received from the University of Madras and the matter is receiving the attention of the other Universities.

8. As directed at the last session a Draft of the rules to be issued under the Medical Diplomas Act, 1939, was forwarded to the Central Government. The rules, as framed by the Government, are being placed before you.

9. Information has been received that the Central Government are consulting the Provincial Governments regarding the Council's proposal for the amendment of the Indian Medical Council Act, so as to vest in the Council power to appoint visitors in addition to Inspectors. Meanwhile, your Executive Committee have drawn up the Instrument of Instructions to be issued to the Inspectors or visitors as and when necessary for their guidance. The Committee have also considered it desirable that the President should send a personal letter to the Inspectors or Visitors, as the case may be, explaining the purpose and object of these Instructions.

10. As a reply to the enquiry made by the Executive Committee, the Bombay University have intimated that under the revised rules for the M. B. B. S. examination, which come into force from the year 1943, the question papers on Forensic Medicine and Hygiene at the 3rd Professional Examination would be set separately, each paper being of two hours' duration and carrying 100 marks.

11. You will recall that the Andhra University and the Government of Madras objected to the inclusion of a date, from which their degrees were to be recognised by the Council, and asked for its deletion from the Schedule. Legal opinion was taken which showed that the action taken by this Council was in accordance with the provisions of the Act. The Executive Committee decided that no further action in the matter was called for.

As regards the communication from the General Medical Council about the non-recognition of the M. B. B. S. degree of the Andhra University, all I can state at this stage is that the Executive Committee in accordance with the previous Resolution of the Council have already decided to carry out an inspection of the courses of Instruction and facilities for teaching at the Vizagapatam Medical College affiliated to the Andhra University and have appointed Inspectors for that purpose. The Inspection will be made early in February next year as suggested by the University.

The Central Government have forwarded a copy of a communication received from the Andhra University requesting the inclusion of M. D. & M. S. degree in the First Schedule to the Indian Medical

Council Act. Your Executive Committee have postponed consideration of the matter till after the inspection of the Andhra University in February, 1941.

12. You may be aware that the Agra Medical School has been raised to the Collegiate standard. The Agra University requested for a preliminary inspection by this Council of the First M. B. B. S. examination of the re-organised Agra Medical College. The Executive Committee asked for legal opinion as to whether the Council have power to inspect the courses of Instruction and pre-clinical examinations of Medical Schools, which at present do not grant a medical qualification covered by the First Schedule to the Indian Medical Council Act but intend to do so in future. The reply from the Central Government being in the affirmative the Executive Committee have decided to carry out the inspection during April next as proposed by the University.

13. This Council's recommendations on Professional Education have come into operation with effect from the commencement of the Academic Year, 1940. An enquiry was accordingly made to ascertain how far these recommendations have been given effect to by the various British Indian Universities and in particular whether each student during his period of clinical clerkship or surgical dressership in the wards has had continuously in his sole charge as clerk or dresser not less than five beds. The replies received in this connection are being placed before your Executive Committee.

14. As desired by you, efforts were made to secure a more suitable and commodious house for the Council in a central place, but no suitable building has so far been found. I suggested that some firm or financier be approached who could build a small suitable house or flat for the Council on a reasonable rent on 10 years' lease in the first instance. In order to make the offer attractive and to provide that the building be constructed in a central place, the Government were requested to allot a plot of land in the neighbourhood of the Secretariat buildings. The Government have, however, expressed their inability to do so as all such land is reserved for Government buildings. Meanwhile, the lease of the present building has been renewed for one year with effect from the 1st October, 1940.

15. One of the items in the Agenda before you is the election of three persons to the Drugs Technical Advisory Board, two of whom shall be from among teachers of Medicine or Therapeutics on the staff of a University or College in British India providing a course of study qualifying for admission to the examination for a degree which is a recognised qualification under the Indian Medical Council Act, 1933, and one shall be a registered medical practitioner not being a servant of the Crown. No definite rules were prescribed for holding the Election. As it was found that collecting, printing and circulating the large number of names to the members would involve a heavy cost, the Executive Committee directed that no list be supplied to members but that the members be

informed that copies will be laid on the office table for their use for the purposes of this election.

16. Under the direction of your Executive Committee the General Medical Council was again addressed on 1st May, 1940, for a final reply to our communication of the 27th February, 1939, regarding the removal of the defects relating to Professional Examinations of the various Qualifying Bodies in the United Kingdom and it was requested that the reply should reach this office before this meeting. In spite of two subsequent reminders no reply has so far been received. The matter is receiving the attention of the Executive Committee.

17. Final reply is still awaited from Japan regarding direct mutual recognition of medical degrees.

The Central Government have forwarded a communication from the Government of Burma enquiring whether the Council have decided to recommend the removal of the Burma degree from the Second Schedule. Meanwhile, the Burma Medical Council has informed us that a draft Bill revising the Burma Medical Act, 1915, on the lines of the Indian Medical Council Act, 1933, has been drawn up and accepted by that Council. The Bill is under the consideration of the Burma Government. Both these communications are receiving the attention of your Executive Committee.

18. As you are already aware, a communication was received from the South African Medical Council regarding the necessity for mutual understanding in respect of the erasure of names of medical practitioners from the Registers so that the decision of the Registering Authority in one country would be accepted in all other reciprocating countries. The Executive Committee have duly considered the matter and their recommendations are being placed before you.

19. At the last session I reported to you about the position that had arisen out of the attitude taken by the New South Wales Government in refusing to complete the scheme of direct reciprocity with us. The matter has been considered fully by your Executive Committee and they have sent up their recommendation for the withdrawal of recognition of the qualification granted by the New South Wales Medical Board which is being placed before you.

20. You must have noticed that in the press and on the platform people have, from time to time, questioned the utility of this Council. I may state at once that most of these critics are uninformed, as they must be, and their criticisms are mostly destructive rather than constructive. We may, as we have done in the past, choose to disregard them. But I feel it desirable that we should consider some of them seriously and place our point of view before the public. It is said that this Council has failed to fulfil the purpose for which it was established, namely, "Securing efficiency at home and honour abroad". Those who have had the opportunity of studying closely the activities of this Council for the last six years

will admit that we have progressed a great deal towards securing greater efficiency at home and establishing "a uniform minimum standard of higher qualification" throughout the whole of British India. To this end, we have laid down, in broad outline, the syllabus and courses of study for medical students to be adopted by all Licensing Bodies included in Schedule I of the Act. When we started drafting these, we were not unmindful of the fact that Universities in India enjoy statutory powers to regulate the courses of study for their students and consequently our proposals are in the nature of recommendations to the Universities and Licensing Bodies, so as to leave them plenty of room for adjustment according to their local needs. Let me here acknowledge our debt of gratitude to these Universities; they, as experts, gave their ungrudging help to us, directly by advising us regarding the details of the course of study and indirectly by accepting, in the main, our recommendations and by working up towards the standard of efficiency these courses of study were intended to achieve. It is my conviction that the Inspections conducted by the Council have been useful in maintaining a uniformity of standard amongst the various teaching bodies in India and in raising the efficiency of these institutions. I may claim, therefore, that the Council had done valuable work in securing "efficiency at home".

21. But can we declare with the same satisfaction that we have been able, by our efforts, to secure "honour" abroad? We cannot say that we are satisfied with the present state of affairs in this regard although this Council is not primarily responsible for it. To my mind, I have no doubt whatever that with increased "efficiency" in teaching, our graduates will soon succeed, by engaging in advanced studies and deeper research, in achieving glory for this country and "honour" abroad. But I also recognise that meanwhile our graduates desire to resort to foreign countries for higher education or for earning their livelihood; and they expect us to secure for them facilities for either, by negotiating with these countries for mutual recognition of degrees as is provided for in the Act. As I indicated in my last address it was, for this purpose, that in 1936-37 this Council approached 27 countries asking them to enter into schemes of reciprocity with us. Many of them did not reply, four of them agreed in the first instance to reciprocate directly with us, though two of them subsequently withdrew, and fourteen countries intimated that they would recognise our degrees only so long as they continue to be recognised by the General Medical Council. This scheme is what I called "Indirect Reciprocity". It has been suggested in some quarters that this unwillingness on the part of foreign countries to enter into schemes of "direct reciprocity" with us, is an indication that our "honour abroad" is at a discount. This Council, however, accepted this scheme of indirect reciprocity in 1937, as we then felt that some form of "reciprocity" was better than none; on our recommendations the qualifications granted by these fourteen countries

were included in Schedule II of the Act. On the last occasion I expressed my doubts regarding the utility of continuing this form of reciprocity. Dr. P. B. Mukerjee moved a motion at the last meeting bearing on the same issue. This motion was referred to the Executive Committee and their considered opinion on this motion and on the problem generally is being placed before you for your consideration.

22. At the last session, I pleaded for an amendment of the Indian Medical Council Act, if this Council were to continue functioning with increasing usefulness. Such an amendment should provide for an All-India Register, which would necessarily imply that this council will have the authority to frame rules for purposes of recognition of medical qualifications and their registration and which would be applicable to the whole of India. The present system is very unsatisfactory. Section 12 of the Act directs that all qualifications recognised by this council will be enrolled on the Provincial Registers. But this section does not debar the autonomous Provincial Councils from recognising and registering even those qualifications which are not included in our schedules. When this Council approaches the "authority of any state or country outside British India, which is entrusted by the Law of such state or country for the maintenance of a Register of Practitioners" it must seem curious to them that we possess no Register and that the present Registering Authorities in the Provinces have their own statutory power in this regard. The Executive Committee has placed before you a resolution for the creation of a general Reciprocity Board for purposes of facilitating reciprocity between India and the various countries comprising the British Commonwealth of Nations. Such a Board can only function effectively if this Council can speak and act for the whole of India. But there is another reason why this All-India Register is a necessity. This council now is concerned with providing for efficient teaching; to my mind, it should also be its duty to insist on the observance of the fundamental ethical principles concerning medical profession and practice, applicable for the whole of India, so that the status of the profession might be raised to such a high standard that it would form an object of admiration of and emulation for other countries. Our code of medical ethics should not be a mere replica of its prototype in England but should in a large measure, be adapted to the peculiar customs, genius and culture of the country. This can only be possible, if we have our own Register, inclusion into or exclusion from which would be the visible sign of the application of such ethical principles.

23. There is one another subject I desire to mention before I finish. A question has often been raised, in some quarters, as to the place it is proposed to be assigned to the present Licentiates in any scheme of reorganisation of the medical profession and in any proposed amendment of the Act. As I indicated in my last address, time has come when we should lay down one uniform

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A Memorandum by the All-India Medical Licentiates' Association to the Executive Committee of the Indian Medical Council on the need for amending the Indian Medical Council Act, 1933, and on the nature and extent of such amendments.

Dr. B. C. Roy and the members of the Executive Committee of the Indian Medical Council,

THE PURPOSE OF THIS DEPUTATION

(1) It is not our intention to discuss now the pros and cons of the Indian Medical Council Act of 1933 or to criticise the working of the Indian Medical Council during the last seven years of its existence. It is sufficient for the purpose of this deputation if we simply bring to your attention, what is common knowledge to the profession as a whole, that the Act has got many grave defects. Of these, a few are so grave that they cannot, if possible, be allowed to remain for long unremedied, because they are not only unjust and invidious but also against the very basic conception of a Medical Council as it should be and as, in fact, it is in other countries. Any more negligence to rectify these defects will continue to undermine insidiously the organic unity of the Indian Medical Profession and to affect the prestige and usefulness of the Indian Medical Council itself both at home and abroad. The purpose of this deputation is, therefore, to lay bare briefly these fundamental defects and to request the Indian Medical Council to bring them to the notice of the Government of India with a view to effect suitable amending legislation to the existing Act.

TWO FUNDAMENTAL DEFECTS

(2) We would but mention two fundamental defects, which, in our opinion, call for an early

(Continued from previous page)

minimum standard of medical education for the whole of India. The Amended Indian Medical Act should provide that this minimum standard must be reached by every medical student before human life can be entrusted to him. Such a standard should be made uniformly applicable to the whole of India. Naturally, considering from this point of view, the word "higher" in the preamble of the present Act should disappear. In the West, they have abolished two standards of medical education and have laid down such a minimum uniform standard. I recognise that this Council cannot do much except to record its opinion in this matter and to recommend action to be taken by the Governor-General in Council. The Central Government will then, we trust, approach the Provincial Governments requesting them to abolish the School-type of education. Some Governments have already done so. If this Council accepts Dr. K. S. Ray's proposition as modified by the Executive Committee we shall place before the Government a concrete proposal for taking such action. May your deliberations secure, now and in future years, increased efficiency at home and honour abroad.

rectification. The first is the restricted scope of the Act and the second is the non-provision of a Medical Register. The latter is a corollary to the former. That this is so will be obvious to one who has closely followed the developments after the failure of Dr. U. Rama Rao's and Dr. Lohakare's Medical Council Bill in the Central Legislatures in 1926 and the subsequent Draft Medical Council Bill by the Government of India in 1929. Between 1929 and 1933 strange things happened owing to the interference of the General Medical Council of the United Kingdom from behind the scene; and the Government of India quietly dropped, almost unobserved, their original idea of establishing a uniform minimum standard of medical education and qualification within British India and replaced it by that of "securing a uniform minimum standard of higher qualification in Medicine for all provinces". That is probably because the General Medical Council wanted no more than a formal statutory Indian Medical Council with which it could deal directly instead of with a larger number of statutory Provincial Medical Councils. This is evident from one of the addresses to the General Medical Council by its President at the time when the Indian Medical Council Bill was on the Legislative anvil. But the idea, of a "British Indian Medical Register" was then still hanging in the balance. The Select Committee to which the Bill was referred on 1st September 1933, dropped the idea of the Medical Register altogether, and we think rightly under the circumstances. In doing so it remarked:

"Most of the opposition to this Bill has arisen from the proposal that a British Indian Medical Register should be confined to graduates in medicine, to the exclusion of the large and important class of the licentiates. We considered whether the Register should be enlarged so as to include the licentiates and whether it should be divided into two parts, one for graduates and one for licentiates; but we are of opinion that this, apart from other objections, would entail a troublesome and expensive duplication of the Provincial Medical Registers and unnecessary and invidious double appeals in disciplinary matters and that the benefits to be gained would be negligible. We, therefore, have decided to recommend a different course and we propose that the All-India Register should be dropped entirely. We have accordingly deleted from the Bill all matters relating to the British Indian Medical Register. The scope of the Bill is now confined to the establishment of an Indian Medical Council, whose functions shall be broadly speaking, two only, namely:—

(1) the securing of a uniform minimum standard of higher qualifications in medicine for all the provinces, and
(2) the arrangement of schemes of reciprocity with medical authorities of other countries.

(3) Thus has the present Act become basically defective in two elements that usually go to make up the soul and the body of a statutory medical council; and because the present Indian Medical Council has neither of it, it did not bring, as

expected by its sponsors, either efficiency at home or honour abroad.

(4) It has not brought efficiency at home for the simple reason that it left the larger field of medical school education to be continued in a state of inefficiency by tacitly guaranteeing non-interference. Neither has it brought honour from abroad, as you yourselves know how foreign countries, and even some Empire countries, refused reciprocity unless it was sought through the General Medical Council.

(5) Our submission to you, therefore, is that the Indian Medical Council should recommend to the Government of India to change the present preamble and scope of the Act into one of establishing and maintaining a uniform minimum standard of medical education and qualification in Medicine for all the provinces, and, then, to provide for a Medical Register inclusive of the existing licentiates.

(6) We submit, our country really needs an efficient medical education. This can be attained only by giving up the existing dual standards, one for the Provincial Medical Council and other for the Indian Medical Council, and introducing instead one standard of minimum medical education and qualification for all the provinces.

(7) Even so early as 1907, the first Conference of the All-India Medical Licentiates' Association, held at Bombay, urged the Government to affiliate all the Provincial Medical Schools to the Provincial Universities. We mention this fact just to show and prove to you that it is not as an after-effect of the invidious and unjust exclusion of the licentiates from the scope of the Act that they have commenced to demand and urged for the establishment of a uniform minimum standard of medical education and qualification but out of actual experience and realisation that what our country and our profession needed, and still in need of, was, and is, one standard of medical education and one homogenous body of medical men.

HOW TO EFFECT THESE CHANGES

(8) We will now proceed to give you our idea as to how to effect these changes proposed by us. It is not at all the intention or the policy of the Association to seek such terms as are only favourable to the licentiates and to claim a statutory provision for the same as an exception. On the other hand it is the deliberate policy of our Association to influence the authorities to establish, in all provinces, a high but uniform minimum standard of medical education and qualification and to put it under the guidance and control of a central statutory Medical Council. Therefore it is, we again suggest, that the present standard laid down by the Indian Medical Council be declared as the future uniform minimum standard for the whole of British India. This would by no means prejudice the position of the medical graduates from the point of view of foreign reciprocity while it would at the same time enable a provision to be made for the recognition of the

existing licentiate qualification in the first schedule of the Act with a note that similar qualifications acquired after, say, 1947 or 1948 would not be so recognised, and for the institution of a "British Indian Medical Register" on that basis. This, in our view, is a very just, equitable and satisfactory arrangement to all concerned.

SOME OBJECTIONS ANSWERED

(9) The first possible objection raised may be: Where is the need for changing the present scope of the Act, since, if the standard of medical school education comes up to the standard of, and recognised as such by, the Indian Medical Council, the licentiate qualification would be automatically lifted from the present ban?

(10) This objection pre-supposes that the Provincial Governments would integrate the medical school education, of which they are in charge, to the standard laid down by the Indian Medical Council. This is a false hope. The long and chequered history of medical school education proves it. There is no statutory binding on the part of the Provincial Governments to adopt the standard of the Indian Medical Council, particularly so because the present standard of the Indian Medical Council is allegedly for "a uniform minimum standard of higher qualifications in medicine" and not for a uniform minimum standard of medical qualification. And it is our firm belief that unless such a statutory compulsion is enforced on them, they would rather try to keep on to the existing medical school standard, sometimes on the ground of need for cheap medical relief, at other times on the plea of financial exigencies, and occasionally even on the basis that the people want such medical schools. In truth, such were the excuses put forward by the various representatives of the Provincial Governments at the last Government of India Conference on Medical School Education held during November, 1938. The fact, however, that the Madras and the U. P. Governments converted their medical schools into University Medical Colleges does not disprove this contention. They have done so out of strong conviction which may not be so enthusiastically shared by other Provincial Governments. Hence the need for a statutory declaration of a uniform minimum standard of medical education and qualification for all the provinces, which, unfortunately, has not been, to-date, yet laid down either by the earlier Provincial Medical Acts or by the later Indian Medical Act. It is really amazing to find that the preamble of each and every Provincial Medical Act mentions not the expediency to establish a uniform minimum standard of medical education but only the expediency "to provide for the registration of certain medical practitioners" in respective provinces. The words differ in some of the Provincial Medical Acts, but in substance they mean the same thing. This would show conclusively that at no time in the history of our medical education has any constituted authorities defined or laid down the uniform standard of minimum medical

education and qualification. You would concede, we hope, that this has been the cause of multiple types of medical education and qualifications, of disunity and divisions in the rank and file of the profession, and of discomfiture in our attempts to obtain foreign reciprocity for Indian medical qualifications.

(11) The advantages, therefore, of making the present standard of the Indian Medical Council as the minimum standard for the future are obvious. They may be recounted briefly as follows:—

(1) It would enable the existing Council to effectively fulfil the usual statutory functions of such a Council, just as it is in the case of the General Medical Council.

(2) It would enable it to provide for a Register without any contention.

(3) It would statutorily compel the Provincial Governments to adopt such a standard for the medical schools in their charge, and thus bring about a uniform standard of medical education and qualification in all the provinces at an earlier date than otherwise seem possible.

(4) By virtue of the introduction of one minimum standard of medical education and qualification, the existing licentiates could be brought within the scope of the Act, and its Medical Register without in any way injuring others' interests.

The second possible objection is that medical school education is within the purview of the provincial legislatures and therefore the Central legislature cannot legislate on this matter. We do not think this is insuperable provided the Government of India is sincerely inclined to help in the achievement of this objective. Central legislation can be undertaken with the consent of the Provincial Legislature under section 100 (2) of the Government of India Act, 1935, on the same lines as they adopted in respect of the recently passed Drugs Act.

THE FUTILITY OF A MERE REGISTER WITHOUT LAYING DOWN THE MINIMUM UNIFORM STANDARD OF MEDICAL EDUCATION

(12) We have been closely following your recent annual addresses to the Indian Medical Council and your other public utterances, and we understand you have been laying more stress on the need for an All-India Medical Register as if such a provision would place the Council on a better footing for the purpose of negotiating foreign reciprocities. You all know that the Provincial Medical Councils have been provided with statutory Medical Registers but they met with little success during the last 27 years in the matter of favourably settling foreign reciprocity. We think, therefore, that the real difficulty and impediment is not wholly in the absence of a Register, but mostly in the Act itself which has virtually, if not apparently, made the Council of little consequence without the patronage of the General Medical Council. Section 14 of the Act has been wholly nullified by Section 120 of the Government of India Act, 1935 and has made the question of reciprocity depend on the consent of the General

Medical Council. This may be instanced by the difficulty which the Council has been experiencing in the matter of retrospective recognition of the Calcutta University medical qualifications. Therefore, apart from political considerations, to attain the normal status of a Medical Council requires that in the Act itself should be embodied two of its essential functions, as in the General Medical Council, viz., (1) to establish a uniform minimum standard of medical education and qualification, and (2) to provide for a Register of such qualified practitioners. One without the other is, we submit, both meaningless and futile.

(13) Mere provision of a Register, even if it includes the present licentiates, without the consequential amendment of the existing preamble of the Act defining and laying down the uniform minimum standard of medical education and qualification, and of section 3 (C) of the Act enabling the licentiates in the Provincial Medical Registers to stand for election to the Indian Medical Council will not serve, as it appears to us, the needs of the situation from whatever angle they may be viewed. It will be no more than a mere shadowy achievement.

CONCLUSION

(14) May we therefore request the Indian Medical Council to pass a resolution recommending to the Government of India to legislate amendment to the existing Act in such a way as to provide not only an All-India Medical Register but also, as an inevitable pre-requisite for the same, to change the preamble into one of establishing a uniform minimum standard of medical education and qualification on the very basis of the present standard of the Indian Medical Council, and to secure through Section 3 (C) of the Act representation to the licentiates in the Council.

(15) Only an amendment of this kind, and to this scope and extent at least, will be the first real step towards consolidation and integration of the different standards of medical school education in our country, towards the creation of a homogenous medical profession in effective service to our people, and towards raising the status, prestige and usefulness of the Indian Medical Council itself.

(16) Any other minor recommendation would be tinkering with the problem without solving it once for all; and a mere Medical Register without defining and enforcing a minimum standard of medical education and qualification would be more like a Medical Directory than a statutory Medical Register.

(17) We thank you for your receiving us in deputations. And we hope we have made our position clear and stated our case in a just and equitable manner. We further believe that what we have asked for and requested you to help in its accomplishment is based on the oft expressed opinion of the organised medical profession in our country as well as that of the enlightened public.

After the Memorandum was read, the following discussion took place:*

Dr. B. C. Roy: Please turn to para 2. You say there that "between 1929 and 1933 strange things happened owing to the interference of the G. M. C." What are these "strange things"?

Dr. A. Viswanathan: The giving up of the basic statutory duty of regulation and control of a uniform standard of minimum medical education and qualification; the consequent abandonment of a Medical Register; the resultant neglect of medical school education; and the non-recognition and the non-inclusion of the licentiates within the schedule of recognised qualifications; all these are indeed "strange things"; brought about by the yielding of the Government of India to the advice of the G. M. C. contrary to the spirit of a Medical Act of this kind. The result was this 'strange' I. M. C. Act. May I ask you, sir: Is it not a fact that of all the Medical Acts in different countries, ours strangely excludes functions which are part and parcel of a Statutory Medical Council?

Dr. B. C. Roy: Please turn to para 4. You say there that the I. M. C. has not brought efficiency at home for the simple reason that "it has left the larger field of medical education to be continued in a state of inefficiency....." you, therefore, admit that medical school education is inefficient. If so, how can you ask for a representation for the Licentiates in a Council which is intended to set up a high academic standard of medical education?

Dr. K. B. L. Varma: If the L. S. S. A. qualification is recognised by the G. M. C., there is no reason why the L. M. P. qualification should not be recognised by the I. M. C. particularly where the licentiates themselves ask for the establishment of a uniform standard of medical education, and when, after sometime and in consequence of levelling up of standards, the L. M. P. diploma will cease to be given. Even as it is, the licentiates could and should be recognised by the Council in the same manner as the G. M. C. did when the British Medical Act came into operation.

Dr. Manohar Lal Kapur: "Inefficiency" is a relative word, and it is not used in the absolute sense. The "efficiency" of a licentiate is common knowledge to all.

Dr. A. D. Mukherji: What is meant is that the I. M. C. has not brought more efficiency in the existing standard of medical school education as a

result of the limitations of the Act, and that therefore it can by no means be sustained that Act has brought "efficiency at home."

Dr. A. Viswanathan: Recognition of and representation of the licentiates in the Council are but interim arrangements, while the Association has certainly no objection to, and in fact is really anxious for, raising of the standard of medical school education.

Dr. B. C. Roy: Please turn to para 10. You say there that "by virtue of the introduction of one minimum standard of medical education and qualification the existing licentiates could be brought within the scope of the Act etc....." How can you say "by virtue of"?

Dr. A. Viswanathan: I hope, sir, you are aware that it is the normal and natural procedure to recognise such qualified people during the period of transition from a lower to a higher standard of medical education. Such a recognition is usually a statutory pre-requisite to the statutory integration of educational standards. I can quote instances to support my statement.

Dr. B. C. Roy: Please turn to para 11. You say that section 14 of the Act has been wholly nullified by Section 120 of the Government of India Act. This cannot be because section 14 of the I. M. C. Act deals with reciprocity in general while section 120 of the Government of India Act deals only with reciprocity between our country and Great Britain.

Dr. A. Viswanathan: No doubt this is technically true, but you know, as much as we do, what it really amounts to.

Col. Jolly: Suppose your request for the establishment of uniform minimum standard is acceded to, but not your request for the recognition of the existing licentiates, what would be your attitude towards such an amendment of the I. M. C. Act?

Dr. A. Viswanathan: I do not think the licentiates would worry about this. I may state on behalf of the Association its general policy which it has been pursuing for the last 34 years. It is this. It has always placed the interests of the country and the interests of the profession before the immediate interests of the licentiates. We have continued to remain in this position for a long time, and we shall be content, if there is no honest way of escape, to remain as we are for a few more years.

[* The above is only a free summary of the discussion Ed. I. M. J.]

The Proposed All-India Medical Register

• Dr. V. Ramakamath, Vice-President, Madras Medical Council

The proceedings of the 14th Session of the Indian Medical Council are more than of usual interest.

The Council has decided to establish a General Reciprocity Board for negotiating reciprocal recognition of Medical Degrees granted in British Dominions and other foreign countries and to request the Central Government to amend the Indian Medical Council Act, 1933, so as to provide for an All-India Medical Register in which will be registered all practitioners recognised at present by the Medical Council of India and practitioners holding qualifications granted by Examining Bodies in India other than the Universities and who will be in the Provincial Registers on December 31st, 1947. In the meanwhile the Medical Council of India has suggested to the Central Government to call upon the Provincial Governments to establish a minimum uniform standard of medical education in the existing Medical Schools.

In regard to critics who questioned the utility of Indian Medical Council Act, 1933, Dr. Roy is of opinion that their criticisms are mostly destructive rather than constructive when they said that the Council failed to secure "efficiency at home and honour abroad." At the same time, Dr. Roy admits that "Our honour abroad is rather at a discount" as a large number of countries with whom the Medical Council of India has been negotiating for reciprocity did not care to reply and some that replied said that they would recognise the degrees if such degrees were recognised by the General Medical Council, Great Britain, which recognition Dr. Roy styles as, "indirect reciprocity". I ask Dr. Roy with all humility at my command whether he is justified under the circumstances in condemning the critics who questioned about the utility of the Council.

In regard to the publication of All-India Register contemplated by the Council I wish to remind Dr. Roy that this question had been discussed threadbare and given up by the Central Government when the Indian Medical Council Act was on the anvil of the Central Legislature. If a Register is now to be instituted, it must therefore include the licentiates and they should be automatically given representation in the Council, taking into consideration the prestige, status, and honour of the Indian Medical profession as a whole. Where is the efficiency in the medical profession which Dr. Roy claims as the bulwark of the Indian Medical Council when this Council minds only about a third of the members of the medical

profession? Can Dr. Roy or his comrades in the Indian Medical Council assure the medical profession and the public that it would be possible for the Central Government to force the Provincial Governments to transform all the existing Medical Schools into institutions to be recognised by the All-India Medical Council when it is so painfully known that the same Council would not recognise the Andhra Medical Degree in spite of the fact that the Council found both the teachers and the taught in the Andhra Medical College not wanting? Dr. Roy should also remember that the Government of the Province from where he hails and where are found the largest number of Medical Schools would not consider the desirability of closing them in spite of a large number of these schools being found most inefficient by the Medical Council of Bengal and also by the medical profession in Bengal. There is thus no justification for excluding the largest group of qualified and registered practitioners from the contemplated All-India Medical Register. For, Dr. Roy must know more than I that the purpose of a Medical Register is not so much to display the qualification of those found in the Medical Register but to enable persons requiring medical aid to distinguish qualified from unqualified practitioners as could be seen from the preamble of the British Medical Act which runs thus:—

"Whereas it is expedient that persons requiring medical aid should be enabled to distinguish qualified from unqualified practitioners: be it therefore enacted by the Queen's most Excellent Majesty by and with the advice and consent of the Lords, Spiritual and Temporal, and Commons in this present Parliament assembled, and by the authority of the same, as follows:—

All the Provincial Councils maintain Medical Registers to enable the public to find out who is qualified and who is not. Regarding efficiency in the profession the same Councils have statutory powers to control medical education imparted by the Universities or by other Examining Bodies in addition to the control over the former by the Universities themselves. It may not be out of place to mention that the Madras University has been demanding much higher qualifications from the teaching staff of the medical colleges than those demanded by the All-India Medical Council.

Under the circumstances the Medical Council of India with Dr. Roy as its head will do well to seriously consider whether there is a need for such a Council to exist at all and if it should exist whether it should not coordinate its functions with those of the existing Provincial Councils instead of its working independently of them. It costs this poor country over a lakh of rupees every year to run the show of the Indian Medical Council which has no funds of its own whereas the Provincial Councils cost little or nothing to the Government as their running expenditure is met from the fees paid by the practitioners for registration.

MEDICAL COUNCILS

Bombay Medical Council

The following summary of the proceedings of the Meeting of the Bombay Medical Council held on the 16th September 1940 is published for general information:—

Government Notifications regarding the nomination of Dr. George Coelho, M.B.B.S., B.Hy., M.R.C.P., and the election of Dr. S. B. Gadgil, L.M.S., L.R.C.P., M.R.C.S., F.R.C.S., as members of the Council were read by the Registrar. The Chairman, Col. Bhatia, then introduced Dr. Coelho and Dr. Gadgil to the meeting.

Dr. Mrs. K. Tarabai, L.C.P.S., was unanimously elected a member on the Bombay Nurses, Midwives and Health Visitors Council in place of the late Dr. Mrs. Cecilia D'Monte, L.M.S.

Dr. U. B. Narayanarao, L.C.P.S., was unanimously elected a representative of the Council on the Provincial Branch of the British Empire Leprosy Relief Association in place of the late Dr. Mrs. Cecilia D'Monte, L.M.S.

The Reports of the Visitors on the 3rd M.B.B.S. Examination of the Bombay University held in December 1939, appointed as per resolution of the Council, were decided to be recorded and it was resolved that a copy of the Reports be forwarded to the Bombay University drawing their attention to certain observations made in the report of Dr. Amesur and the observations contained in the Joint Report of Dr. Bharucha and Dr. Jivraj Mehta.

Papers relating to the memorandum from the Surgeon General with the Government of Bombay regarding Bill to prohibit conferment of unauthorised medical degrees were recorded.

The recommendations of the Executive Committee on the Draft Bill to provide for the registration of Dental Practitioners in the Province of Bombay, forwarded by Government for the opinion and suggestions of the Council thereto, were accepted with certain modifications therein, and it was resolved that the opinions and suggestions on the provisions of the Draft Bill as finally approved by the Council be communicated to Government.

The Council passed a resolution by a majority expressing the opinion that in the absence of a common Indian language, medical education of the western type should be imparted through English as is done at present, for the time being. An amendment to the original resolution seeking postponement of the consideration of the question for the next meeting of the Council was declared lost.

It was decided to restore to the Bombay Medical Register the name of Dr. D. S. Pathre, M.B.B.S., removed under section 9 of the Bombay Medical Act, 1912, on 8th November 1938, accepting the recommendation of the Executive Committee in that behalf.

Government Resolution regarding amendment to rules 61 and 64 of the Rules of the Council was recorded.

The notice of motion regarding amendment of section 3 of the schedule to the Bombay Medical Act, 1912, was referred to a Sub-Committee for report to the Council.

The notice of motions regarding amendments in the Rules of the Council relating to election was referred to a Sub-Committee for report to the Council.

Notice of motions relating to certain amendments in the Bombay Medical Act, 1912, and other subjects was referred to the Executive Committee for report to the Council.

Bengal Medical Council

Abstract of the Minutes of the Bengal Council of Medical Registration, dated 16th February 1940.

1. The Council had at their meeting of 8th August 1939 adopted a resolution that it was desirable that the country of qualification particularly when the qualification had been obtained from a foreign country, should be mentioned along with the abbreviation used for medical degrees, so that the public might not be misled: and expressed hope that a convention would be gradually established by registered medical practitioners if they followed this practice.

The question whether this should be included in the Penal and Ethical Rules was considered at the meeting of 16th February 1940: and it was decided that though desirable this could not and need not be included in these Rules.

2. On a reference made to the Council on the question as to the propriety or otherwise of supplying personal history of patients in a Hospital such as one for Tuberculosis, to Life Insurance Companies, the Council observed that Rule 19 in Part III of the Council's Penal and Ethical Rules was clear enough on the subject and that it would not be proper for the authorities of such hospital to supply personal history of patients treated therein to such companies. The Rule is as below:

"A medical practitioner is under an obligation to his patient to preserve his secrets and in legal matters should, except with the patients' consent, answer questions only at the express direction of the Judge or Magistrate presiding in a court of law. A medical practitioner is not bound to answer questions put to him by policemen except as provided in section 44 of the Criminal Procedure Code."

3. This Council noticed with regret that an amendment of the Bengal Medical Act on the lines suggested by the Council from time to time, had not yet been taken up by the Government. They also desired to impress upon Government the necessity of improving the standard of Licentiate'ship course, by raising the preliminary qualification to I. Sc., and extending the period from 4 years to 5 years.

4. The Council also resolved that the departments of the Government of India such as the Posts & Telegraphs, Income-Tax, etc. should be requested to follow the Government of Bengal circular No. 2536 dated the 8th September 1921, which lays down that when a medical certificate has been given to a non-gazetted officer by a registered practitioner, no further examination by the Civil Surgeon or a higher medical officer should be required unless the genuineness or veracity of the certificate was doubted.

Abstract of the Minutes of the Bengal Council of Medical Registration, dated the 13th August 1940.

1. The council repeated their recommendation that with a view to restrain quack practice, all dispensaries should be licensed and no dispensary should dispense any preparation which is not signed by a practitioner possessing registrable qualification.

2. The Council repeated their recommendation for suitable measures forbidding the use of the prefix "Dr." as a medical qualification except by persons possessing registrable qualifications.

3. On the question of propriety of registered practitioners being Directors of firms manufacturing medicines, the Council adopted the following resolution:

"That this Council would not object to the mere association of medical practitioners with firms of manufacturing medicines, whether as directors or shareholders, but the names of such medical practitioners should not appear in any publication (such as catalogue of prices or advertisements) circulated to the general public by the firms or its agents.

4. The Council amended their Ethical Rule 8 as follows:

"When a medical practitioner is asked to see and report on a case of illness or injury of a person who is under the care of another registered practitioner or about whom the letter has already given a medical certificate, the former should in all cases give the latter an opportunity to be present at the time of examination, if he so liked; and for this purpose he should direct the patient to inform the latter practitioner in sufficient time.

The medical practitioner when so seeing or reporting on a case, shall not interfere with the treatment nor make any disparaging remarks about the first diagnosis and treatment, even though he might differ."

5. Certain cases of medical certificates being given by practitioners without sufficient care and caution, the practitioners were warned by the Council.

6. On the question of registered practitioners teaching medical subjects in Ayurvedic institutions, the following resolution was adopted qualifying section 3, Part I of the Ethical Rules:—

"This section does not apply so as to restrict the teaching of pre-clinical subjects such as Anatomy and Physiology, by registered practitioners, in institutions recognised by the Ayurvedic State Medical Faculty of Bengal."

7. The Council decided to add a chapter at the end of the Annual Medical List giving a list for the general information of all practitioners showing references to various provisions in the law which impose certain legal obligations on them and penalties for default. A copy of the list is enclosed. [This will be published in the next issue of the Journal. Ed.]

Annual Report of the Finances of the All-India Medical Licentiate's Association for the period from the 1st October 1939 to 30th September 1940, by the Hony. Treasurer of the Association.

This report covers the period of the Association Financial Year from 1st October 1939 to 30th September 1940.

MEMBERSHIP

The total number on roll of the members of the Association on the 1st October 1939 was 2688, while on the 30th September 1940, it was 2548 i.e., a fall in the number of members by 140. During the period under review 428 new members joined the Association, whereas 568 left it; most of whom did so by refusing to accept the V. P. P's issued to them and thereby caused a further additional drain on the funds of the

Association in the shape of postage and registration charges on V. P. P's. This unnecessary drain would have been avoided if the retiring members had kindly taken the trouble of intimating the Honorary Treasurer beforehand.

A comparative table of membership of the Association is given below:—

1st Oct. 1938	1st Oct. 1939	1st Oct. 1940
2716	2688	2548

It shows a gradual yearly fall in the number of members. It may be due to general economic depression and partly to lesser enthusiasm. If each and every existing member believes in the force and strength of large membership and makes it his duty to enlist at least one new member every year, the strength of the Association can be multiplied very easily in a short time.

SUBSCRIPTION

The total subscription realized during the period under review is Rs. 11275-5-0. A comparative table showing the income by subscription is given below:—

1937-38	1938-39	1939-40
Rs. 12054-8-0	Rs. 11711-13-6	Rs. 11275-5-0

It shows that there has been a slight fall in the subscription which can be made up only if more new members come in.

INCOME AND EXPENDITURE

During this period, the total income of the Association from all sources is Rs. 26369-0-9 and £ 157-17-10. There was a sterling War Loan of £ 157-17-10. in the possession of the Association, but this was not entered in the Account Books of the Association. It came to the knowledge of the Hony. Treasurer during this period and was therefore taken on the receipt side, in order to tally the accounts. The total expenditure of the Association during the period under review was Rs. 19968-6-0. Thus there was a saving of Rs. 6400-10-9 during this period.

In spite of very hard times and dislocation of business due to the war, it is very creditable that our Association has shown a surplus. A comparative table showing the income and expenditure of the Association is given below:

	1937-38	1938-39	1939-40
Income	Rs. 29449-5-10	Rs. 26153-12-5	Rs. 26369-0-9
Expenditure	20881-4-1	Rs. 27076-9-0	Rs. 19968-6-0

Note: Rs. 734-0-3 on account of the Omera Gold Medal Account, Contribution by the late R. B. Dr. Sarjo Pershad and 13 Shares of the Bacterio Clinical Laboratory, Calcutta, were already in the possession of the Association but were not entered in its account books; hence they had to be entered in the income side in 1938-39. Therefore the total net income (excluding the above items) during 1938-39 was Rs. 26163-12-5.

BALANCES

The total cash balance of the Association on 30th September 1940, was 70388-14-11 and £ 157-17-10. Out of this the Association possesses Government Securities to the face value of Rs. 40800-0-0 and £ 157-17-10. All the rest of the amount is in Banks and Cash Certificates etc. except Rs. 793-12-1 which is standing against Dr. S. P. Sen Gupta, Ex-Manager of the *Indian Medical Journal* and is being shown as balance not yet recovered.

A comparative statement of the balances of the Association is given below:—

30th Sept. 1938	30th Sept. 1939	30th Sept. 1940
Rs. 64170-0-9	Rs. 63988-4-2	Rs. 70388-14-11
		£ 157-17-10

The present balance includes Rs. 283-6-0 donated by the late Dr. R. B. Sarjo Prasad and Rs. 43-0-0 collected on account of the Sarjo Prasad Memorial Fund.

The surplus of income over expenditure and the balance in hand clearly show that the finances of the Association are very sound. It is a matter of great satisfaction that the Association has been able, during the period under report, to maintain its financial prosperity notwithstanding the fact that the present War conditions have somewhat affected the subscription and advertisement income of our journal. Not a little credit for this goes to those who have the finances of the Association in their keeping and who exercised strict control and economy of expenditure whenever possible without sacrificing efficiency.

An outstanding feature of this period under review is that all the Reserve Funds of the Association are deposited in the Imperial Bank of India, Delhi. All the Government Securities are also deposited there with the exception of two which are in the safe custody of Assistant Account General, Posts & Telegraphs, and whose safe custody receipts are with the President of the Association. The date of one Government Security for Rs. 5000 had expired, and it was repurchased through the Post Office, Madras by the President.

Fresh Government Securities (3½% G. P. Notes), to the face value of Rs. 6000 were purchased during the year. The details of the balance are given in the consolidated account of the Association for the month of September 1940. (Vide page 167 of this supplement)

Monthly consolidated accounts showing actual receipts and payments are published in the *Indian Medical Journal* regularly. An annual consolidated Statement showing the details of the actual income and expenditure is published on page 166 of this supplement.

BILLS

During this period the total number of bills received from the Finance Committee for payment or adjustment was 325. All payments were made promptly without any delay. Total amount paid to Branches on account of Contingent and Propaganda Bills amounted to Rs. 2050-9-6 and Rs. 796-5-0 respectively.

ADVANCE WITH BRANCHES

Rs. 279-1-3 is standing against the various Provincial and District Branch Secretaries under the sanction of the Finance Committee. A certificate is obtained from them each year showing the amount standing against them.

OFFICE CLERK

The Hony. Treasurer's office clerk did his work very well, regularly and honestly, kept the accounts up to date and gave perfect satisfaction all round.

ACKNOWLEDGMENTS

The Hony. Treasurer wishes to acknowledge and place on record that he received full help and perfect co-operation from all his co-workers and colleagues but for which it would not have been possible for him to successfully do all the work of the Association in these critical days. He thanks them all for their selfless labours.

All those who cherish the well-being of the Association in their heart must make united and consolidated efforts to increase the number of members of the Association, so that with the increasing number, it may gather strength and be fit enough to achieve its objects.

(Sd.) KHAN CHAND WORHA,

Hony. Treasurer, A. I. M. L. A.

List of the properties of the All-India Medical Licentiate's Association as on 31-9-1940

Articles	Approximate value
GENERAL SECRETARY'S OFFICE	
Same as last year, vide <i>I. M. J.</i> Supplement, Decr., 39, page 201	Rs. A. P. 387 3 0
OFFICE OF THE INDIAN MEDICAL JOURNAL	
Same as last year, vide <i>I. M. J.</i> Supplement, Decr., 1939, page 201, with the following additions:—	1250 0 0
2 Wooden boxes for keeping office records, 3 ft. x 1½ ft. x 2 ft.	15 1 0
2 Almirahs, with double glass doors lock cases, 5 compartments, 5½ ft. x 3 ft. x 1½ ft.	33 0 0
OFFICE OF THE TREASURER	
1 Punching Machine, 4 locks, 5 trunks, 1 seal brass, 24 rubber stamps, 2 paper weights iron, 1 stamp stand, 1 dictionary, 1 Book-keeping by Fieldhouse, 1 table lamp, 1 Pin cushion, 1 waste paper basket, 1 glass ink pot, 1 blotter, 3 chairs, 1 cup board (almirah), 2 tables, 1 rack, 1 tea-poy, 2 paper racks, 1 stationery rack, and 1 table cloth flannel.	—
OFFICE OF THE FINANCE SECRETARY	
Same as last year, vide <i>I. M. J.</i> , Jan. '39 Supplement, page 11	—
Hissar Branch	
1 trunk, 28" x 18"	3 4 0
2 Rubber stamps	—
Dibrugarh Branch	
Same as last year, vide <i>I. M. J.</i> , Dec. '39 Supplement, page 201	4 0 0
Gauhati Branch	
Same as last year, vide <i>I. M. J.</i> , Dec. '39 Supplement, page 201	9 14 0
Madras Branch	
1 Almirah, 4' x 2' x 1'	0 8 0
Tinsukia Branch	
2 Rubber stamps	—
1 Tin Box 20"	0 12 0
Madras Prov. Branch	
1 Punching Machine	—
Total Rs.	1703 10 0

CONSOLIDATED STATEMENT OF ACCOUNTS OF THE ALL-INDIA MEDICAL LICENTIATES' ASSOCIATION
FOR THE FINANCIAL YEAR 1939-1940

Receipts			Payments		
	Rs.	A. P.		Rs.	A. P.
To Subscription	11275	5 0	By Printing <i>I. M. J.</i>	5496	2 9
„ Advertisement	11887	2 1	„ Establishment	5076	0 0
„ Interest	3191	2 11	„ Office Rent	720	0 0
„ Miscellaneous Receipts	15	6 9	„ Stationery	184	2 0
„ *War Loan	£ 157	17 10	„ Travelling allowance	1508	7 6
Total	26369	0 9	„ Postage etc.	2408	4 9
& £ 157	17 10		„ Miscellaneous	738	15 9
Opening Balance on 1st Oct. 1939	63988	4 2	„ Other Printing	210	10 9
Grand Total	90357	4 11	„ Propaganda work	796	5 0
& £ 157	17 10		„ Expenses for Branches	2050	9 6
			„ Furniture	62	14 3
			„ Audit fees	60	0 0
			„ Payment to Contributors	125	15 0
			„ Advt. Commission	66	6 0
			„ Subscription to foreign Journals	163	0 9
			„ Scientific Section	1	7 0
			„ Expenditure for Omera Gold Medal Account	101	12 0
			„ Other contingencies	55	8 0
			„ Expenditure for Standing Committee Meeting	141	13 0
			Total	19968	6 0
			Closing Balance on 30-9-40	70388	14 11
			& £ 157	17 10	
			Grand Total	90357	4 11
			& £ 157	17 10	
Note.* This was already in the possession of the Association, but was not included in the Treasurer's Books. Hence taken into the income side.					
Total (net) income	26369	0 9			
Net Expenditure	19968	6 0			
Profit	6400	10 9			

“MALARIDOL”

(REGD.)

An effective treatment for Malaria and all kinds of intermittent fevers

Composition:—*Tinospora Cordifolia*, *Pterocarpus Marsu Pium*, *Carum Nigrum*, *Sarsae Radix*, *Chiratta*, *Pyrus Cydonia*, *Anathi Fructus*, *Cardamomi Semina*, *Alstonia Scholaris* and other indigenous drugs.

Therapeutics:—A preparation for combating MALARIA OF ANY SEVERE TYPE AND ALL KINDS OF FEVERS. By virtue of its composition “MALARIDOL” has been found to control even the most obstinate forms of chronic Malaria.

PACKINGS:—AVAILABLE IN BOTTLES OF 63 TABLETS AND 27 TABLETS RESPECTIVELY AND PACKETS OF 9 TABLETS ALSO

JAMBU & COMPANY,
(Agents for Pharmaceutical Products)
Thyagarayanagar, MADRAS.

Branches and Delegates are requested to note the change of Venue of the 30th All-India Medical Licentiates' Conference from Ahmedabad to BOMBAY.

The Conference will be held on
22nd & 23rd Dec., 1940.

Consolidated Accounts of the All-India Medical Licentiates' Association

RECEIPTS	JULY 1940	AUGUST 1940	SEPTEMBER 1940
	Rs. A. P.	Rs. A. P.	Rs. A. P.
To Subscription (old and new)	710 4 0	557 1 0	793 12 0
„ Advertisement	1013 14 0	763 15 10	629 10 0
„ Interest	592 2 3	727 0 2	112 14 0
„ Miscellaneous	1 0 0	2 10 0	
„ Loan from General Secretary, Karnal		0 13 6	
Total	2217 4 3	2051 8 6	1536 4 1
Balance of last month	70382 8 2	71212 13 11	70741 2 11
&	£ 157 17 10	£ 157 17 10	£ 157 17 10
Grand Total	72649 12 5	73264 6 5	72277 6 11
&	£ 157 17 10	£ 157 17 10	£ 157 17 10
DETAIL OF BALANCES			
3½% War loan (Imperial Bank, Indore)	£ 157 17 10	£ 157 17 10	£ 157 17 10
13 Shares of the Bactro-Clinical Laboratory	85 0 0	85 0 0	85 0 0
Omera Gold Medal Account	387 6 11	387 6 11	387 6 11
New Loans	8200 0 0	8200 0 0	8200 0 0
5 Year Postal Cash Certificates	4010 10 0	4010 10 0	4010 10 0
G. P. Notes	10891 9 0	16291 9 0	16291 9 0
Govt. Bond for 1943	5600 0 0	5600 0 0	5600 0 0
Govt. Bond for S. S.	5000 0 0	9409 2 1	9409 2 1
Imperial Bank of India, Delhi	4095 10 6	2365 14 6	8189 10 6
Suspense Account (for cash certificates)		2467 8 0	
With Dr. S. P. Sengupta, Ex-manager	793 12 1	793 12 1	793 12 1
Postal Savings Bank with President	7779 13 6	3370 11 5	2 11 5
With Branches	562 4 9	304 1 3	279 1 3
With Editor			
In hand	65 1 8	61 13 11	94 14 11
In Bank	1838 0 0	1721 14 10	1396 8 10
In Stamps	47 1 0	40 10 3	38 11 6
With General Secretary	16 12 6		166 2 9
With Treasurer			
P. O. S. Bank	12276 7 6	12021 7 6	11914 7 6
Pb. N. Bank	9452 2 6	3513 0 8	3513 13 6
In hand	111 2 0	92 8 6	15 4 6
In stamps			
Cheque post-dated		4 0 0	
Grand Total	71212 13 11	70741 2 11	70388 14 11
&	£ 157 17 10	£ 157 17 10	£ 157 17 10
EXPENDITURE			
By Printing <i>I. M. J.</i>	415 0 0	441 11 0	192 0 0
„ Establishment	390 0 0	400 0 0	788 0 0
„ Office rent	60 0 0	60 0 0	70 0 0
„ Stationery	14 1 0	12 11 6	14 8 3
„ Postage	158 0 9	209 12 9	166 0 3
„ Travelling Allowance	57 12 0	829 14 6	399 13 6
„ Miscellaneous	53 2 3	63 7 3	57 14 0
„ Propaganda work	191 12 0	84 8 0	89 12 0
„ Expenses of branches	72 2 6	207 5 6	93 14 6
„ Advt. Commission	5 0 0		
„ Omera Gold Medal			
„ Payments to contributors	20 0 0	45 12 0	
„ Furniture			
„ Other Contingencies			
„ Expenses of Standing Committee held at Bombay		26 4 0	4 0 0
„ Loan returned to General Secretary		141 13 0	11 12 0
Total	1436 14 6	2523 3 6	1888 8 0
Balance in hand	71212 13 11	70741 2 11	70388 14 11
&	£ 157 17 10	£ 157 17 10	£ 157 17 10
Grand Total	72649 12 5	73264 6 5	72277 6 11
&	£ 157 17 10	£ 157 17 10	£ 157 17 10

NO. 75—IP (3)/40

Government of India

Department of Commerce

Office of the Superintendent of Insurance

New Delhi, The 14th August, 1940.

From M. V. Nayudu, Esq., M. A., B. L., F. I. A.,
Asstt. Superintendent of Insurance.To The Honorary Secretary,
The All-India Medical Licentiates'
Association Family Fund, Bombay.

Sir, I have to forward herewith under sub-section (3) of section 87 of the Insurance Act, 1938, a copy of the report embodying the results of an enquiry held on the 15th & 16th July, '40, by the Inspector appointed under sub-section (1) of that section, into the manner in which the business of your society is conducted. (2) I have to request that the receipt of the report may be acknowledged at an early date.

Report of Inspection Under Section 87 (1) of
the Insurance Act, 1938

Dr. U. B. Narayanarao, the Honorary Secretary, was present.

1. The society insures money to be paid on death or on the members' becoming invalid. One rupee is collected from every living member when a claim occurs, and nine-tenth of the collections are paid to the claimants.

The society has so far issued 867 policies, of these 345 have lapsed, 193 have matured: one member has resigned and 3 sympathetic calls have been issued. On the date of inspection 325 policies were in force.

The policy form does not clearly reproduce Rule 16 of the Society as regards payment on a member's becoming invalid.

2. The name of the Fund has not been altered in accordance with the provisions of section 67 of the Insurance Act.

3. The Society's scheme is one on dividing principle. The permission of the Superintendent of Insurance under section 69 (2) has been obtained on 24th January 1940 to continue the business for a period of six months.

The society did not issue any call after 15-4-39, but moneys were received after that date in respect of the earlier calls issued, and claims were also paid in November 1939.

The actuary's report on the society's scheme as at 30-9-38 together with his reorganization proposals were submitted to the Extraordinary General Body meeting held on June 1940. The meeting resolved to wind up the business for the purpose of distribution of the assets amongst the members. The Superintendent of Insurance is stated to have been informed of this, and his instructions in the matter are awaited.

4. The deposit required to be made under section 73 has not been paid by the society.

5. The society's rules and policy forms have not been changed in accordance with section 74 (2) of the Insurance Act, 1938.

6. The society's office was shifted to the present address on the 1st July, 1940. There was not signboard hung outside the office. The society has contravened the provisions of section 77 of the Act. (1)

7. The society, it was stated, did not employ any agents. Hence, agents' register was not maintained. The other statutory books were maintained. The books were not written up-to-date. (2)

8. The accounts as at 30-9-39 were stated to have been sent to the Registrar. The accounts for the year ending 30-9-39 are not yet submitted. The society has not therefore observed the time limit laid down in section 82 of the Act. The society's financial year does not end on 31st December. The attention of the society is invited to the provisions of Section 82 of the Act. (3)

Remarks and explanation by the Secretary:—(1) The board is since put up. (2) This is according to a resolution of the Working Committee. (3) The audited report is published now along with this report. (4) This is bound to occur and has been so far the last so many years. (5) Dr. Shah, the Manager, has since refused to sign the cheque.—Secy., Family Fund.

(a) Revenue Account from 1-10-38 to 13-7-40

Death Calls.	General Fund.	
Paid and outstanding.	as on 1-10-38	14783-15-1
2903-6-7	Calls due	3631-2-7
Establishment. 553-14-0	Penalty calls,	
Stationery,	Interest &	
Printing & other)	admission fees	666-14-6
Expenses.)	1557-14-9	
Less on furniture		
& Depreciation. 62-3-0		
General Fund		
on 13-7-40.	13404-9-10	
	18482-0-2	18482-0-2

The expenses have exceeded the limit laid down in Rule 13 of the society's Rules. (4)

(b) Balance Sheet as on 13-7-40

General Fund.	Furniture	
13404-9-10	Postal cash	63-0-0
Death Calls	Certificates	8812-8-0
admitted	Fixed deposit in	
1271-14-7	Indore Premier	
but not paid.	Co-operative	
Death Calls	Bank Ltd.	4083-14-0
received in	Calls due from	
advance.	members	340-9-0
280-0-0	Post Office	
Deposit from	Savings	
members.	953-0-0	
Sundry Creditors	175-0-0	
Suspense	8-0-0	
	Bank account	313-2-1
	Imperial Bank	2478-13-7
	Savings Account	
	Cash on hand	1-1-9
	16093-0-5	16093-0-5

LIABILITIES.

Death Calls admitted but not paid

Many of the claims have not been fully settled. A large part of this figure represents small balances remaining to be paid on account of the old claims. The Fund should settle these at an early date.

Sundry creditors

Rs. 100 has been advanced by the Secretary to the society to meet expenses. It was explained that the Secretary had to do this because the Bank account could not immediately be drawn upon, all the persons in whose name it was to be operated not being in Bombay at the time. The remaining Rs. 75/- are owed by the society to its auditors. (5)

Assets	Rs. A. P.
Furnitures. As on 1-10-38.	1093-0-0
Sold during 1938-39	21-0-0
Loss on sale.	55-3-0
	76-3-0
	32-0-0
Additions since 1-10-38	38-0-0
	70-0-0
Depreciation written off.	7-0-0
	63-0-0

Postal Cash Certificates and Fixed Deposit in the Indore Premier Co-operative Bank Limited.

These could not be produced for inspection. It was stated that these assets were kept with the two directors in Indore.

Post Office and Imperial Bank Savings Accounts

I have checked the amounts with the pass-book.

Cash on Hand

I have checked the cash balance and found it correct. 9. The Society got its scheme examined by its actuary. The actuary's report dated 30-9-39 was sent to the Superintendent of Insurance on 12-6-40. The report of the actuary in as on 30-9-38.

10. The lapses are heavy. The members, it seems, do not desire to continue the society.

(Sd.)
Ms. 26-7-40. Inspector.

ALL-INDIA MEDICAL LICENTIATES' ASSOCIATION FAMILY FUND
BALANCE SHEET AS AT 30TH SEPTEMBER, 1939

FUNDS AND LIABILITIES	Rs. A. P.	ASSETS	Rs. A. P.
General Fund : ...	14488 7 1	Furniture : As per last Balance Sheet ...	108 3
Death Calls :		Less : Sold during the year	21-0-0
Admitted but not paid 1715-0-7		Loss on sale	55-3-0
Payable on realisation of calls in arrears	451-1-0		76 3 0
	2166 1 7	Add : Additions during the year	32 0 0
Received in advance	263 8 0		38 0 0
	2429 9 7	Less : Depreciation written off on above at 10 per cent	70 0 0
Deposits from members : ...	959 0 0		63 0 0
Sundry Creditors : (For expenses)	125 0 0	Death calls in arrears :	451 1 0
Suspense :		Investments and accrued interest :	
Admission Fees ...	13 0 0	2 Post Office 5 years Cash Certificates of Rs. 5000 each, at cost. (Due on 13-6-1944) ...	8812 0
Cost of Certificates ...	1 0 0	Interest accrued on above from 13-6-1939 to 30-9-1939 ...	Nil
	14 0 0	Fixed Deposit with the Indore Premier Co-operative Bank, Ltd., including interest. (Due on 10-10-1940) ...	4083 14 0
Total	18016 0 8	Cash and other balances : Savings Deposit Accounts :—	12896 6 0
		Imperial Bank of India, Bombay	4136 6 1
		Post Office Savings Bank Indore	313 2 1
		In hand	156 1 6
			4605 9 8
		Total	18016 0 8

Bombay, 12th September, 1940.

REPORT OF THE AUDITORS

We have audited the Balance Sheet of the All India Medical Licentiate's Association Family Fund as at 30th September, 1939, above set forth and have obtained all the information and explanations we have required. The above Balance Sheet exhibits a true and correct view of the state of the affairs of the Fund according to the best our of information and the explanations given to us and as shown by the books of the Fund.

A. FERGUSON & CO., Chartered Accountants, Registered Accountants.

REVENUE ACCOUNTS FOR THE YEAR ENDED 30TH SEPTEMBER 1939

To Balance at the beginning of the year :	Rs. A. P.	By Death calls paid during the year :	Rs. A. P.
General Fund ...	14783 15 1	(a) Calls maturing for the current year	437 13 9
Death Calls claimed and levied on members :—		Less : Unpaid carried down.	256 15 9
Unpaid ...	1307 13 7		180 14 0
Payable on realisation (of this Rs. 837-4-0 were realised during the year ...)	1052 5 0	(b) Previous Calls :—	
.. Calls made during the year :	2360 2 7	Unpaid last year	1307-13-7
Cash Receipts	435-0-0	per contra	1307-13-7
Outstanding	236-0-0	Maturing during the year	707-10-3
Less : 1/10 of the total call receipts during the year appropriated to general expenses	126 12 0		2015-7-10
.. 1/10 of Call Receipts as above	126 12 0	Less : Unpaid carried down	1458-0-10
.. Penalty Calls.		.. Office Establishment	312 3 0
(Amount recovered from 2 members over 35 years of age at the time of admission)	85 0 0	.. Audit Fees	50 0 0
.. Interest	490 12 6	.. Printing & Stationery	123 6 0
.. Admission Fees	61 0 0	.. Postage, Money Order Commission	97 2 6
.. Cost of Certificates	3 14 0	.. Miscellaneous Expenses	87 11 0
	767 6 6	.. Travelling Expenses	330 5 0
		.. Depreciation on Furniture.	7 0 0
		.. Loss on sale of Furniture...	55 3 0
		.. Balance at the end of the year :	
		General Fund (contra) 14783-15-1	
		Less : Excess of Expenditure over Income.	295-8-0
		Death Calls.	14488 7 1
		Payable on realisation of previous calls	215-1-0
		Of calls made during the year	236-0-0
			451-1-0
		Unpaid. For previous years 1458-0-10	
		.. this year 256-15-9	1715-0-7
			2166 1 7
			16654 8 8
		Total	18455 12 2
		Total	18455 12 2

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growth will obviously result from gross dietary indiscretions. The child who excretes 5 per cent sugar in a twenty-four hour volume of urine loses 100 grams of sugar, or 400 calories. One pound of human body is equivalent to 1500 calories. At this rate if a pound of weight could be lost every three days, the growth in stature has to be necessarily retarded. To prevent defective growth the ordinary food requirements of childhood do not seem to be sufficient. According to Joslin, under five years of age 1400 calories, six to ten years of age 1600 calories, and ten to fifteen years of age 1800 calories on the whole are found to be adequate for the diabetic children.

THE PATHOLOGY OF DIABETES IN CHILDHOOD

This is characterized by the absence of pathology of the pancreas and substitution of fat for carbohydrate metabolism. This is shown in the liver, spleen, kidney, muscle and skin. Histological staining and chemical analysis reveal glucose in place of glycogen or glycogen deposited in abnormal fashion or fat stored where storage of glycogen is normal.

SYMPTOMS

Sudden or rapid onset in children is often the rule as contrasted with the indefinite onset found in adults. Except for this rapidity of onset juvenile diabetic patient differs little from those of the adult. Loss of weight, strength, hunger, loss of appetite, polyuria and pruritis are common. Pain in the leg muscles, cramps and change of disposition too may be present. The differential diagnosis of diabetes in childhood may be exceedingly difficult. With a classical case and with obvious symptoms it is smooth sailing, but in the case of an early or latent diabetes it is very difficult. This is complicated by the fact that routine urine analysis reveals some traces of sugar at one time or other of even healthy children. The final diagnosis rests invariably upon the level of the blood sugar and even here one uniform standard has not been established. But the majority view is that a diagnosis of diabetes could be made if fasting blood sugar is 0.14 per cent, and the post parandial blood sugar is 0.17 per cent, venous, or 0.2 per cent capillary blood.

THE TREATMENT OF DIABETES IN CHILDHOOD

This reduces itself to the adjustment of three essentials—diet, insulin and exercise. By means of diet control, carbohydrate is restricted; by

means of insulin burning and storing of it is effected; and exercise enables it to be consumed.

(1) *Diet*.—The dietary problem resolves into the choice of division and the determination of the number of calories necessary for growth. 3 : 1 carbohydrate-fat ratio is found adequate.

(2) *Sugar-free urine*.—A five day plan of therapy is given below as recommended by Joslin.

TECHNIQUE OF DESUGARIZATION OF DIABETIC CHILDREN

Day	Age under 5 years				Age 10 years				Age 15 years			
	C	P	F	Units of insulin	C	P	F	Units of insulin	C	P	F	Units of insulin
1st	100	60	60	3+3+3	120	60	60	5+5+5	140	60	60	10+10+10
2nd	110	...	...	...	130	...	...	...	150	...	...	...
3rd	120	...	...	...	140	...	...	...	160	...	...	...
4th	130	...	...	...	150	...	...	...	170	...	...	...
5th	140	70	70	...	160	70	80	...	180	80	90	...

Specimens of urine are collected for two hourly qualitative and twenty-four hourly quantitative tests. Most important are the specimen voided at 11-30 A.M., 4-30 P.M. and 9-30 A.M. and if they are sugar free, or nearly so, additional carbohydrate from 5 to 10 grams is given. If these tests are unsatisfactory, the insulin dosage is increased the following day to anticipate the poor test. The time of injection of insulin is important. One hour before breakfast, half hour before evening meals and at retiring are the three most suitable periods for injection of insulin.

COMPLICATIONS IN DIABETES IN CHILDHOOD

In the management of juvenile diabetes more than treatment of the disease, treatment of sudden emergencies occupies equal or more important place as emergencies occur more frequently in diabetic children than in diabetic adults. Of these coma easily leads the list and the management of the same is discussed below in detail.

Coma.—During the pre-insulin era this was the major cause of death in juvenile diabetes especially amongst those far away from hospital facilities. Now the outlook is radically changed, coma is now preventable, and if treated reasonably early curable by insulin. The signs and symptoms of diabetic coma are the same in the child as in the adult. The onset may be more rapid in the child. When the onset has been insidious, there is usually an indefinite period of malaise, pains in the

head, legs, abdomen and back. The aggravation of symptoms of untreated diabetes may develop. Nausea and vomiting are grave warning signals. Air hunger is the most characteristic sign. Drowsiness and unconsciousness come on slowly. Patient is flushed and the skin is dry. There is the characteristic odour of acetone in breath. The pulse is rapid. The heart sounds are weak, often with a systolic murmur. The blood pressure is sub-normal. The lungs may show harsh breath sounds. The abdomen may be tender and frequently distended. The liver is enlarged and the eyeballs are soft. The pupils react sluggishly to light.

In the treatment of diabetic coma:—(1) *Insulin* is the sheet anchor. One should be quite sure of the diagnosis. When the coma is diabetic the measure of improvements is not to be judged by the units of insulin given but by clinical condition and also by frequent examination of urine for sugar. Insulin is to be stopped when the Benedict's test shows blue or light green. Till then insulin occupies the most conspicuous place in the treatment of this complication.

(2) *Fluids*.—Dehydration is one of the striking clinical features of diabetic coma. The skin is dry and inelastic. The mucous membrane of the mouth and tongue are parched, the eyeballs are soft and the subcutaneous tissues are depleted of fluid. Normal saline 1000 c.c. (subcutaneous) may be given to start with and may need repetition during the first few hours. The solution should be slightly above body temperature and should be given very slowly.

(3) *Gastric lavage*.—Unless the patient is in *extremis* one's efforts in the removal of gastric contents will not go in vain as the stomach may be filled with quantities of fluid, food remains, etc.

(4) *Circulatory — stimulants*.—are rarely needed in children but in cases of great collapse, adrenalin 3 to 1 c.c. or coramine may be given subcutaneously.

(5) *Food*.—Usually glucose is not added to the subcutaneous or intravenous infusions used at the outset owing to the existing high sugar content of the blood. When giving insulin and other accessory treatments during the first 24 hours carbohydrate not exceeding 100 grams in the shape of orange juice may be taken.

(6) Cleansing enema of the bowel is helpful and authorities on the subject are of opinion that alkalis especially in quantities necessary for effectiveness in a chemical sense is not only unnecessary but may be harmful. The

Diet in the Treatment of Diabetes Mellitus*

● S. C. Dutta, L. M. P., Itakhooli,
T. E., Khero, Assam

METABOLISM IN DIABETES

In order to formulate diet for diabetics a thorough knowledge about the physiology of blood in relation to carbohydrate metabolism is necessary. The normal blood-sugar level varies from 0.08 to 0.17 per cent. Probably this concentration of sugar in blood is necessary for the normal working of the vital organs of the body. When the level rises above 0.17 %, sugar is excreted in the urine. Hence this percentage is termed the 'renal threshold' or the 'leak point' of kidney. When the tolerance of kidney is reduced due to some reason or other, sugar may be excreted in urine even though blood-sugar percentage is less than this. This fact has an important bearing on the diagnosis of diabetes mellitus which should not be confused with renal glycosuria. Therefore, blood-sugar estimation is necessary for accurate diagnosis.

A normal individual should assimilate about 200 gms. of glucose without sugar appearing in urine. The clinical test used for ascertaining the power of the body to deal with carbohydrates is the sugar-tolerance test.

* A paper read at the Provincial Conference, Assam A. I. M. L. Asson. on 28th October, 1940.

treatment following coma for the first few days may be difficult and needs a plan, and once again Joslin is handy.

PLAN OF TREATMENT FOR FIVE DAYS AFTER COMA

Day	Carbo: gram	Proteins: gram	Fats: gram	Insulin
1	100	40	40	Insulin every 3 hours
2	110	50	50	" " " " 4
3	120	60	60	" 4 times a day
4	130	70	70	" " " " "
5	140	80	80	" " " " "

Dangers during the first 24 hours of treatment are (1) over-dose of insulin which is not of very great consequence (2) anuria, complete or partial, may develop which needs fifty per cent glucose or 10 per cent saline intravenously to be given immediately.

The responsibility of the physician in the management of a juvenile diabetic is indeed great. In his expert handling depends the patient's future health, happiness and well-being.

The body uses sugar with the help of insulin in three ways:—

(1) By burning it up for the supply of heat and energy.

(2) By storing it up as glycogen in liver and muscles for immediate use when necessary.

(3) By transforming some part of it into fat, in which state the energy-store is less easily available.

In diabetes mellitus, there is deficiency of secretion of insulin from the pancreas due to the degeneration of some of the cells of the islets of Langerhans. The carbohydrates absorbed from the intestine in the blood cannot, therefore, be utilised and there is rise of blood-sugar above renal threshold with excess of sugar passing out in urine. As a result, polyuria, polyphagia and polydipsia occur and these are considered to be the diagnostic trio of this disease.

The most important effect of deficient carbohydrate metabolism is imperfect burning of fat in the body, because "fats burn in the fuel of carbohydrates". Normally fats burn to CO₂ and water but when carbohydrate-metabolism is deficient, the fats cannot be fully oxidised and end in an intermediate stage, forming some poisonous products viz., beta-hydroxy-butyric and aceto-acetic acids in such quantities that they are excreted in urine. These acids upset the alkali-reserve of blood and lead to acidosis or ketosis which ends in coma and death. So, fatal end in diabetes is due to the production of these fatty acids and not necessarily due to hyperglycaemia.

Proteins are easily digested and assimilated by the diabetic, but in the process of digestion some of these are converted into carbohydrate and some into fatty acids. Hence excessive proteins are also harmful in diabetes.

POINTS TO CONSIDER IN FORMULATING DIET FOR A DIABETIC

Severity of disease, age, weight, nature of occupation and constitution are the factors influencing the diet of a diabetic patient. A knowledge of the body weight is necessary for the preliminary estimation of the basal-calorie requirement.

The severity of disease is probably the main thing to consider when estimating the amount of carbohydrates to start with. If carbohydrates are more than the patient can assimilate, excessive amounts of sugar will be

excreted and if much less, ketone bodies will appear in urine.

The age should be considered because a young patient requires more nutrition than an old one. If the patient is pursuing an active occupation, he requires more nourishment for the proper supply of energy.

If the patient is fatty and over-weight he should get less amount of fat. Starvation diet in such a patient is very liable to cause ketosis; because when the carbohydrates are wanting in the diet the liver-glycogen is first used and then the fat storage is encroached upon, which burns into ketone bodies.

The diet should satisfy the patient as regards his appetite and taste. It should not be monotonous and should be sufficient to keep him fit for pursuing his ordinary duties of life.

Finally, there should be a careful adjustment between the three principal food-factors, particularly when the case is treated with insulin. A mistake in the balance may kill the patient by ketosis or hypo-glycaemia. Hence it is essential that the diabetic diet should be calculated with the help of metabolic data.

CHANGING CONCEPTS OF DIABETIC DIET

Before the discovery of insulin, diabetics were treated by severe restriction of carbohydrates and liberal allowance of fats in the diet. By this method the patients' life could be saved for the time being, but it was an invalid's life ending sometimes in ketosis and coma. After insulin this severe dietary restriction has become unnecessary. Severe restriction of carbohydrates increases the chance of ketosis, which is immediately dangerous and liberal supply of fats adds further to this danger. Hence the present idea is to give a liberal diet sufficient for the energy-requirement of the body, serving this purpose more by carbohydrates than by fats. The minimum amount of carbohydrates recommended in 'basal diet' is 100 gms. which may be increased in uncomplicated cases getting insulin, upto 200 gms. considering of course, the appetite of the patient and conditions of blood and its sugar level.

The fats should not exceed 100 gms. and if carbohydrates be nearing the maximum, fats may be still further lowered down to 50 gms. in fatty subjects.

The protein-requirement in all cases is rather a fixed one. As a rule, it should be 5 gms per lb. of body-weight of the patient. This may vary under special circumstances e. g., in ketosis.

SYSTEMS OF DIETARY IN DIFFERENT STAGES OF DIABETES

Very mild cases of diabetes, without causing any constitutional trouble, occur generally in elderly patients and can be controlled by simple restriction of diet by avoiding sugar and other articles rich in carbohydrates. If the urine is not sugar-free, the case requires better care and further restriction of dietary and accurate estimation of urine-sugar and blood-sugar are necessary.

Moderately severe cases may be treated in two ways: (1) A preliminary starvation stage followed by rapid increase of diet (Ladder Diet). If this dietetic measure alone fails to keep the urine sugar-free then the case should be treated with (2) A diet just sufficient to satisfy the basal-calorie requirement of the body (Basal Diet) together with injections of insulin.

Ladder Diet—Grahams ladder diet consists mainly in the following steps for an adult of average weight.

- (1) First day—Complete fast.
- (2) Second day—Carbohydrate 12 gms.
Protein 36 „
Fat 48 „
- (3) The diet is gradually increased till on the tenth day it reaches.
Carbohydrates 72 gms.
Protein 71.4 „
Fat 97.5 „
- (4) If urine is sugar-free on the tenth day, increase carbohydrates by 10 gms. every three days till 100 gms. are reached.

In mild cases on this diet, the urine becomes sugar-free on the second or third day; but if sugar persists, the cases should be treated with 'maintenance diet' and insulin.

Ladder Diet is contraindicated if ketone-bodies are present in urine because of its high fat and low carbohydrate content.

Basal Diet and Maintenance Diet.—The basal calorie requirements of the body may be estimated approximately by multiplying the patient's ideal weight in pounds by 11.3. The total calories are now distributed in carbohydrate, protein and fat, for which the following rules are observed.

The amount of carbohydrate given is 100 gms. The amount of protein given is calculated at the rate of .5 gms. per pound of body weight. After protein and carbohydrate are calculated, the remaining calories go to fat.

Suppose the patient's weight is 118 lbs. His basal requirements is 1333.4 calories (118 × 11.3). The carbohydrate to be given to him is 100 gms. That approximately gives 400 calories (100 × 4).

The protein portion to be given to him is 59 gms. (.5 gm. × 118). That gives approximately 236 calories (59 × 4). Both these together give 636 calories. The remaining 697.4 calories (1333.4—636) should go on to fat. This

means 77.5 gms. ($\frac{697.4}{9}$) should go to fat.

Therefore, an adult of 118 lbs. of weight should get 100 gms. of carbohydrate, 59 gms. of protein and 77.5 gms. of fat as his 'basal diet' having a calorie value of 1333.4.

The carbohydrate portion is then gradually raised with the tolerance of the patient so long as sugar is not passed in urine and with the increase of dosage of insulin, till the diet is sufficient to supply the energy requirement of his body for the pursuance of ordinary duties of life. This is considered to be the 'maintenance diet' for the patient. Dr. Bose's formula for calculating a basal diet based on the older concept of less carbohydrate and more fat, is described below.

$$\begin{aligned} \text{Total calorie requirement} &= \text{weight of the patient in kilos.} \\ &\quad (2.2 \text{ lbs.}) \times 25 \\ \text{Carbohydrate requirement} &= \frac{\text{Total calorie requirement}}{30} \\ \text{in gms.} &= \frac{55}{30} \\ \text{Protein requirement} &= \frac{\text{Total calorie requirement}}{33} \\ \text{in gms.} &= \frac{55}{33} \\ \text{Fat requirements in gms.} &= \frac{\text{Total calorie requirement}}{12} \end{aligned}$$

Calculating on the above formula, a patient weighing 118 lbs. should get the following diet.

$$\begin{aligned} \text{Basal calories} &= \frac{118}{2.2} \times 25 = 1337.5 \\ \text{Carbohydrate} &= \frac{1337.5}{30} \text{ gms.} = 44.6 \text{ gms.} \\ \text{Protein} &= \frac{1337.5}{33} \text{ gms.} = 40.5 \text{ gms.} \\ \text{Fat} &= \frac{1337.5}{12} \text{ gms.} = 111.5 \end{aligned}$$

It is claimed that, in average mild diabetes on this diet, the urine becomes sugar-free in four days and blood-sugar comes down to normal in a week.

INSULIN TREATMENT AND DIET

Generally the treatment with insulin begins with small dosage. In average moderately severe cases, a daily injection of 10 units is given. If after two days the urine shows sugar, an additional 5 units in the evening is indicated. The insulin is then increased by 2 units every other day till urine is sugar-free. In severe cases and if ketone bodies are present in urine, increase in dosage may be more rapid. In cases treated with insulin, the patient should get 100 gms. of carbo-

hydrate at the outset, the greater part of which is given in the meal which is given about $\frac{1}{2}$ hour after the injection. Young patients following manual labour should get more carbohydrate to prevent hunger and loss of weight. Insulin acts better if the patients are a bit under weight. Therefore, if the patient is over-weight, fat may be reduced in the diet to as low as 50 gms. but the carbohydrate should be gradually increased and in uncomplicated cases, may go up to 200 gms. While the carbohydrate is being raised, the dose of insulin should be increased by 1 unit for every additional 4 gms. of carbohydrate, though later on the dose may be reduced to the original level or below that. Every individual case should be treated according to its own circumstances. The dose of insulin should be adjusted carefully to the carbohydrate given by examination of urine every three hours after each injection and if necessary by blood-sugar estimations.

DIET IN JUVENILE DIABETICS

Diabetes in children is of graver prognosis and should be treated with insulin immediately the diagnosis is established. The treatment begins generally with 3-5 units of insulin two or three times daily. But the difficulty is with the estimation of correct dietary. The basal calorie requirement of children is relatively high because allowance should be made for their growth. The diet should also contain sufficient proteins, vitamins and minerals. In such cases it has been found that the carbohydrate-fat ratio should be 2:1.

The following diet is recommended for diabetes in children, getting insulin treatment.

Age	Calorie requirement	Carbohydrate	Protein	Fat
5 Years	1430	140 gms.	60 gms.	70 gms.
10 Years	1650	160 gms.	70 gms.	80 gms.
15 Years	2100	200 gms.	90 gms.	100 gms.

CONVERSION OF FOOD VALUE INTO ARTICLES OF DIET

This is the most difficult task in the treatment of diabetes. In Western countries standard food-tables and diet-menus provide easy reference for the purpose; but these are not suitable for Indian patients. Moreover, standard food-tables suitable for India are difficult to prepare owing to the widely varying dietary habits in different parts. Therefore the attending physician should always consult food-value charts for the prescription of individual diets. The following information

on food-values of some selected articles may make the problem easier.

The following vegetables contain little or no carbohydrate and are recommended for the diabetics:

Lettuce, mustard, cabbage, french bean, spinach, cauliflower, radish, cucumber, papaya, tomato.

The following articles of diet contain little or no carbohydrate:—Saccharine, tea, coffee, lemon, pepper, bran-biscuits.

5 grams of carbohydrate are contained in the weights mentioned against the corresponding articles in their edible parts below:

VEGETABLES		FRUITS	
Potato	1 oz.	Banana	1 oz.
Green peas	1½ oz.	Grapes	1½ oz.
Broad bean	1 oz.	Mango	1 oz.
Onion	2 oz.	Oranges	2½ oz.
Garlik	½ oz.		
STARCHY FOODS		OTHERS	
Rice	½ oz.	Milk	3½ oz.
Atta	½ oz.	Dal	½ oz.
Barley	½ oz.		
Sago	½ oz.		
Arrowroot	½ oz.		
Oatmeal	½ oz.		

1 oz. of dal (average) cooked with 1/3 oz. of butter or ½ oz. of mustard oil, yields about 7 gms. of protein, 7 gms. of fat and 16 gms. of carbohydrate.

100 gms. of carbohydrate are contained in about 2 chattaks of rice or 2½ chattaks of atta.

The following articles contain about 7 gms. protein and 7 gms. of fat in weights mentioned; and any one item may be substituted for another without causing any serious error.

Beef	1 oz.	With addition of $\frac{1}{2}$ oz. of butter.	
Mutton	1 oz.	Chicken	1 oz.
Egg	1 oz.	Goat	1 oz.
		Pigeon	1 oz.
		Fish (lean)	1 oz.

The following articles contain about 5 gms. of fat in weights mentioned.

Clarified butter	1/5 oz.
Mustard oil	1/6 oz.
Olive oil	1/6 oz.
Cod liver oil	1/6 oz.
One gm. of carbohydrate yields	4.1 calories
One gm. of protein yields	4.1 „
One gm. of fat yields	9.3 „

PATIENT'S PERSONAL KNOWLEDGE ABOUT HIS DISEASE AND DIET

All intelligent diabetics should be instructed about the main metabolic disturbances going

Diabetes and Pregnancy

● **Dr. Sudhir Bose, B.Sc., M.B., F.R.C.S.E., M.R.C.O.G., Teacher of Midwifery, Campbell Medical School & Hospital, Calcutta**

on in the system and the principles of dietary to be followed. He should know how to test his urine and take insulin injections. He should be warned about the signs of danger and the occasion to send for the physician immediately.

The following facts are important and should be impressed on every diabetic.

(1) Excess of sugar in urine is not immediately dangerous but it leads to many complications and should be controlled. Sugar in urine may be tested and a rough percentage estimation done by Benedict's test.

(2) Diabetic coma may develop rapidly when there are ketone bodies in urine, which may be detected by ferric choloide test. Ketosis may be controlled by cutting off fat and increasing carbohydrate in the diet, at the same time giving big doses of insulin.

(3) Hypoglycaemia may be caused by excess of insulin, by delaying meal after the injection or by excessive exertion after the injection. The warning symptoms are restlessness, nausea, perspiration, head-ache and mental disturbance. It may be controlled by taking sufficient sugar orally or by taking intravenous glucose.

ACKNOWLEDGEMENT

My thanks are due to my Chief, Dr. K. P. Hare, for his valuable help in the preparation of this paper and kind permission to publish it.

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It was long known that glycosuria may be associated with pregnancy and puerperium. It was considered to be a normal physiological process, as sugar was found to exist in the form of lactose in most of the cases. Mathews Duncan in 1882, first collected 22 authentic cases of diabetes mellitus associated with pregnancy.

Diabetes affects the reproductive organs unfavourably. But the same despondent outlook of pre-insulin era of the time of Mathews Duncan does not prevail nowadays. With the advance of insulin therapy, along with the progress of modern bio-chemistry, more pregnant diabetics are coming under observation and a larger number of patients, of mild or average severity, reach the full-term and are confined, without any untoward result. This, however, has imposed a bigger responsibility on the modern obstetrician and he has to work in co-operation with the physician and the biochemist.

Though ante-natal clinics are getting more popular in Bengal every day, the response is not as universal as one may think due to the indifference and the social bias that still prevail among the general mass of Indian population. Sporadic cases of diabetes and pregnancy are sometimes met with, but most of them seek hospital aid too late when the disease is far advanced and hence complete case records are difficult to obtain. The following short history of a case observed during two successive pregnancies is of interest.

Mrs. S. B., Hindu female, —30 years—married 5 years. Suffered from irregularity of menstruation after an attack of typhoid fever 12 years ago. She found that she was putting on fat after that. Both her father and grandfather had diabetes mellitus.

She developed pruritus vulvae, a year after marriage, which did not respond to any local applications or treatment with Prognon B. oleosum.

When she was about four months pregnant, urine on examination showed 3% sugar and fasting blood-sugar was 2%. The regulation of diet, insulin treatment and ultra-violet radiation soon brought the disease under control—urine sugar a trace and blood-sugar 13%. During the last trimester she showed an apparent improvement. She never developed any sign of toxæmia except a trace of albumen, in the last week of pregnancy. She was allowed to go to full-term and during the last week of pregnancy external examinations revealed that the baby was small in size. She went in labour, a week after the expected date of confinement; but with the onset of labour pains the foetal heart sound could not be heard. Labour ended in a difficult craniotomy and the baby weighed about 5½ lbs. The puerperium was uneventful and blood-sugar was found to rest at 13%.

She conceived again one year after and the same regime of diet and insulin was followed. Except a slight anaemia and general weakness she showed no untoward symptom. Repeated examinations revealed that blood-sugar ranged from 13 to 17% and urine sugar from 1 to 2%.

Two weeks prior to the expected date, she was getting frequent uterine contractions, more of the nature of cramps.

As this was getting worse, induction of labour was started with castor oil, quinine and pituitrin. This failed and high rupture of membranes was performed two days after. The labour pains soon started but was not effective. After twenty-four hours she developed high temperature with rigor. Midforceps were applied as there were signs of maternal and foetal distress. A baby weighing about 8 lbs. was delivered badly asphyxiated. The cervix was lacerated and there was an extensive tear of left vaginal wall. The mother had a stormy puerperium. High temperature continued for about three weeks and she developed parametritis and whiteles. She was confined to bed for about 8 weeks. Blood sugar went upto 3%. She could not nurse the baby.

The baby is now six months old, healthy and putting on weight. Mother is keeping a good health and attending to her daily duties. Blood sugar is steady at 13%.

There is a functional disturbance of ovulation if the disease is chronic. There is reduction in the activity of pituitary, thyroid and the reproductive glands. Atrophic and sclerotic changes are noted in the ovaries and uterus. There is a lack of oestrin, disappearance of graafian follicles and deficiency of genital developments. Patients suffer from sterility, menstrual disturbances and obesity. The last was noted in the case illustrated. Menstruation may be absent in about half the number of cases. Only 1 to 5% of patients suffering from diabetes become pregnant.

More cases of primary amenorrhoea are coming under observation, where with efficient insulin treatment, regular menstrual cycle has been established and pregnancy occurred when treatment was continued. Organic changes occur more frequently when the disease is chronic and a long-standing one, and do not depend so much on the severity of the disease alone. The damage to the organs is supposed to be permanent, when there is no recovery of the reproductive function even with assiduous treatment.

Inheritance of diabetes has not yet been settled, though many cases show a predisposing factor due to heredity. The latter is illustrated in the case noted. According to Joslin, the disease tends to be transmitted as a recessive mendelian trait.

Pregnancy exerts an unfavourable effect on diabetes. As in the case cited, diabetes may appear, for the first time during pregnancy. If it is already present, it undergoes an exacerbation during pregnancy. Diabetes, appearing first during pregnancy, generally manifests itself at about the fourth or fifth month and very seldom later, or during the puerperium. The onset is generally insidious, but coma may be the first symptom in some acute cases due to increased ketosis. Pruritus vulvae was the first sign in the illustrated case.

Opinion is divided amongst the physiologists regarding the prognosis of diabetes with the advance of pregnancy. Many state that there is an apparent improvement of the diabetic condition, especially in the later months, as long as the child is living and placental circulation intact. This is shown in the case of Mrs. S. B. Death of the fetus *in utero* may result in the immediate breakdown in the control of the disease. This is ascribed to the work taken up by the foetal pancreas which shows hypertrophy and evidence of a greater physiological activity. But some

state facts to the contrary. So whether this improvement is due to the over-activity of the foetal pancreas or due to the greater utilisation of carbohydrate in the fetus in such a state has yet to be proved.

Vomiting of early pregnancy augments the danger of diabetic acidosis, requiring an intake of a greater amount of carbohydrate. There is a liability to the onset of toxæmia in the later months of pregnancy. Joslin suggests that this is due to the upset of carbohydrate metabolism. There is a striking association of diabetes with the vascular type of toxæmia. High blood pressure is recorded in most of the cases.

The estimation of maternal mortality among pregnant diabetics varies between 25 to 50% with different observers. Williams found it to be 27% during pregnancy.

Miscarriage or premature labour occurs in about 30% of cases. Labour is more often difficult due to the large size of the fetus. This, as illustrated in the case cited, necessitates a more frequent manipulative interference, which predisposes to puerperal sepsis. Herrick quotes the morbidity figure as high as 41%. Sepsis may terminate in coma.

An excess of sugar in liquor amnii, indicated by sweet taste and sticky consistence, was long known to early observers. It is found to rise about 10 or 12 times the normal amount of 0.054%. Hydramnios is a frequent occurrence. Ludwig and Chapiet found it as common as 27%. The excessive liquor amnii is the result of both foetal diuresis and osmosis from surrounding tissues. This helps to prove that amniotic fluid is partly secreted by foetal kidneys.

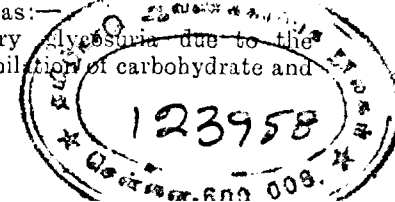
The size of the fetus is often enormous. The average weight may rise up to 10-12 lbs. This is the result of the increased supply of sugar to the fetus. Underweight is also noted in some of the cases. In the illustrated case, the first baby weighed 5½ lbs. and second 8 lbs.

Dubreuil and Anderodias first described the characteristic hypertrophy and hyperplasia of the islets of Langerhans in the foetal pancreas. This results in an excess production of insulin by the fetus which, as referred to previously, produces an apparent improvement in the diabetic mother in the later months of pregnancy.

The percentage of stillbirth, abortions and the various congenital malformations of the child is still high. The death-rate of the fetus is about 50%. The fetus often dies *in utero*, due to the onset of acidosis and sometimes as a result of hydramnios. The placenta does not show any characteristic change. There is also a susceptibility of the child dying within a few days after birth. In the case of Mrs. S. B., during the first pregnancy, the child died suddenly *in utero*, as soon as the labour pains started.

Various types of glycosuria, apart from true diabetes mellitus, may exist during pregnancy. Lambie classifies them as:—

(a) Early alimentary glycosuria due to the lower limit of the assimilation of carbohydrate and



usually met in the third or fourth month of pregnancy.

(b) Renal glycosuria, due to the lowered renal threshold, first appears in the sixth or seventh month and disappears towards the end of pregnancy or in the puerperium.

(c) Lactosuria may be present a few days before confinement, but is marked when the baby is weaned or when there is more secretion of milk than required by the baby.

(d) Late alimentary glycosuria—or sometimes called "transient diabetes of pregnancy"—usually appears about the fifth or sixth month of pregnancy and due to the lowered limit of the assimilation rate for carbohydrate. Another type of glycosuria, transient in its behaviour, due to the diminished intake of carbohydrate is also noticed in the later months of pregnancy.

When sugar first appears in the urine, it should first be decided that it is not due to any deficient intake of carbohydrate. It should disappear if the patient is allowed a liberal carbohydrate diet viz., 150-200 gms. of glucose for 2 or 3 days.

If sugar in urine does not exceed 2 to 3% and the total output *per diem* does not exceed 10 gms. then it is not a case of diabetes mellitus. If diet does not influence glycosuria, it is not of diabetic origin. The fasting blood sugar, sugar tolerance test and the height or prolongation of the blood sugar curve will help in diagnosing diabetes mellitus from other types of glycosuria, as mentioned.

The essentials in the treatment of a pregnant diabetic lie in strict supervision, proper control of diet and the administration of insulin in requisite dosage.

She should take adequate rest, avoid chill and guard against sepsis. During treatment blood sugar should be estimated from time to time. Urine must be daily tested for sugar, acetone and albumen.

With slight modifications, the diet should follow the same principles as that in an ordinary diabetic. The slightly higher calorie and protein requirements by the pregnant diabetic and the disturbed ketogenic: anti-ketogenic balance demand the necessary change in the dietary. The calorie requirements are about 4% above that required by a non-pregnant diabetic, and 35 calories per kilogram should be sufficient.

The general principles of insulin therapy is the same as in the case of a diabetic, when she is not pregnant. Insulin causes an efficient storage and combustion of the large amount of carbohydrate that is required to prevent ketosis. This would, otherwise, produce an injurious effect on the pancreas. It diminishes the tendency to acidosis with its attendant dangers to both mother and child. Insulin does not produce any injurious effect on the foetus probably by preventing the excessive developments of islet tissue. It controls

the development of hydramnios, death of the foetus and its excessive gain in weight.

During labour and twenty-four hours following it, if the usual dose of insulin is administered, the amount of easily assimilable carbohydrate, in the form of glucose, orange juice, etc., should be sufficiently increased to avoid acidosis. It should be noted that hypoglycæmic reactions frequently appear during the early puerperium when the insulin requirement should be diminished.

Whether a diabetic mother should nurse the baby is a debatable question. Lambie, like many others, is of opinion that nursing of the baby is better avoided, so that there may not be an extra-strain on the mother, nor induce her to take more food. But Peckham states on the contrary, and furnishes evidence that suckling produces better results in a diabetic mother.

Diabetic should be advised to avoid pregnancy. The outlook is more hopeful if it appears for the first time during pregnancy. If a patient suffering from chronic diabetes becomes pregnant, she should be under careful supervision, better in a hospital or in a well-equipped nursing-home, for a few months at a time. If she shows improvement with insulin treatment, regulation of diet and there is no continuance of the loss of tolerance or any severe sign of toxæmia, the pregnancy may be allowed to proceed to term. Otherwise, abortion should be induced. Pregnancy should be terminated in ill-nourished patients or in grave cases of acidosis. Premature induction of labour may be required, if the child shows evidence of excessive increase of weight and if hyperglycæmia continues even at the later months of pregnancy.

Chloroform or ether should not be administered owing to their toxic action on the liver. All precautions should be taken to prevent sepsis.

Successful results of Cæsarean section under gas and oxygen are recorded from time to time. But Bland has shown the mortality-rate of the operation to be not less than 6.5%, when it is performed for the condition. The survival rate of the child is not so promising, as one may expect. So diabetes alone should not be an indication for Cæsarean section.

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Management of Diabetic Emergencies

[A summary of discussion between the staff members and students of the Cornell University Medical College and the New York Hospital. Reproduced from the *J. A. M. A.*, Aug. 10, 1940, with due acknowledgments.]

GENERAL TREATMENT

Dr. Eugene F. Du Bois: The management of the ordinary, uncomplicated case of diabetes is not difficult these days; the management of emergencies will always remain difficult. Dr. Tolstoi will take up the clinical aspects.

Dr. Edward Tolstoi: As the result of recent experiments performed in collaboration with Dr. Weber and Mr. Toscani, we have been led to adopt a somewhat less orthodox point of view in the treatment of the diabetic patient. We found that our experimental subjects were free from the symptoms of diabetes in spite of glycosuria when they were receiving a diet of 75 Gm. of protein, 60 Gm. of fat and 200 Gm. of carbohydrate, and protamine zinc insulin in daily doses of 50 units. Two rather unusual patients whom we observed in the metabolism ward for periods of fifty to sixty days maintained their weight. I want to emphasize this point. They also had no thirst or polyuria; they had no ketonuria and they were in nitrogenous equilibrium in spite of the fact that they excreted as much as 150 Gm. of sugar some days, and in spite of the fact that there was an ever present postprandial hyperglycemia. From such experiments we postulated that for patients treated with protamine zinc insulin the guiding features of satisfactory treatment should be (1) the maintenance of weight, (2) freedom from symptoms and (3) absence of ketonuria. Furthermore, we were not concerned about the hyperglycemia or the amount of dextrose excreted. It is our belief that if the diabetic patient is made to utilize a quantity of carbohydrate essential for his particular metabolic needs the excess may be excreted without damaging results.

We have had further opportunity to pursue these studies in ambulatory cases in the outpatient department, and our results there have been gratifying. We paid little attention to the glycosuria; we were guided by the principles mentioned, and the majority of our patients have done exceedingly well. They have been liberated from multiple injections of insulin and now receive one injection of insulin a day, putting their syringes away for the remainder of the time as they would their tooth-brushes.

Dr. Du Bois: Are you referring to insulin now or to protamine zinc insulin?

Dr. Tolstoi: All reference will be to protamine zinc insulin unless I specifically state otherwise.

Simultaneously there have been others in the same field who have adopted the point of view that the patient may excrete sugar with safety. The quantity of sugar excreted in twenty-four hours has been a question of controversy. Some say a

diabetic patient is well controlled when he excretes only 10 Gm. of sugar in twenty-four hours. Others permit him to excrete as much as 40 Gm. of sugar a day. Our patients excreted as much as 150 Gm. on occasions. Both of the patients whom we studied recently at this hospital excreted 100 Gm. daily for thirty days, a total of more than 6 pounds (3 Kg.) of sugar. They were in nitrogenous equilibrium and they felt perfectly well.

As to diabetic emergencies, there are in reality only two which are directly and intimately associated with diabetes. One of these is acid intoxication, or ketosis, and the other, which involves the insulin treated diabetic patient only, is the hypoglycæmic syndrome.

ACID INTOXICATION

The chief causes for ketosis, the more hazardous complication of the two diabetic emergencies, are either medical or surgical infections—a common cold, a boil, a sore throat or the more serious conditions such as pneumonia or an acute surgical state of the abdomen. All of these are potential causes of ketosis. Others are departure from dietary restrictions and eating too much, the omission of insulin, and hyperthyroidism. This year I have had an opportunity of seeing two patients in whom stubborn ketosis developed during severe sunburn. It is noteworthy that a simple condition such as pharyngitis may decompensate the diabetic patient so that it precipitates not only ketosis but coma as well.

We should differentiate between the two terms "ketosis" and "coma". The clinical differentiation is especially important. Coma is a state in which the patient is unable to respond to any and all stimuli. In ketosis he may be up and about. The distinction between coma and ketosis has been made by a laboratory test by Joslin and his school. They feel that if the carbon dioxide combining power is below 20 the patient is apt to be in coma; if it is above 20, he is not in coma.

Let us consider how trouble arises for the diabetic patient who contracts an infection. First he takes less food because he loses his appetite. He has been taught that in such a situation he should omit his insulin. Here the fault lies with the physician who fails to realize that even though the diabetic patient does not ingest any food he will draw on his own storehouse. These foods cannot be utilized properly without insulin. The result is that ketone bodies begin to form. The little endogenous insulin that he may have is rendered less potent by the infectious process, which thus furnishes another factor conducive to ketosis. As the ketone bodies begin to form, the patient shows a characteristic syndrome that sets in rather insidiously. At first he feels weak and listless. He is perfectly content to be left alone. A little later thirst develops, and frequency as well as polyuria. He may also complain of headache, backache and aches in the calves of both legs. These symptoms are annoying, but they do not alarm the patient particularly until he begins to vomit. With the vomiting there is epigastric pain. This pain may

further localize in the right lower quadrant. When the patient is seen at this time, the physician's attention is directed to the abdomen. An examination of the abdomen may show not only slight rigidity but even rebound tenderness. Appendicitis, of course, is the condition that suggests itself. At this point a blood count reveals leukocytosis with anywhere from 20,000 to 80,000 cells and a good percentage of immature neutrophils. Further examination of the patient will reveal that he is flushed; the tongue is parched, dry and beefy red, as is the throat; the eyeballs are soft; the skin is dry. The question arises: Is it uncomplicated ketosis or a surgical abdominal condition which has precipitated the ketosis?

Laboratory data at this point are of importance: in the blood sugar varying from 250 to 1,000 mg. per hundred cubic centimeters, low carbon dioxide combining power, elevated non-protein nitrogen, hemoconcentration; in the urine large quantities of sugar, large quantities of acetone and diacetic acid. These establish the diagnosis. What are we going to do with a patient in this situation?

First let us consider the one whose condition is not far advanced, the patient as we see him in the clinic. He may report a sore throat. Examination of the urine reveals a 4 plus acetone reaction and diacetic acid. The skin and the tongue are dry, there is dehydration and there are listlessness and the desire to be left alone. We tell the patient "Go home, take a tablet or two of table salt (1 Gm of salt) every hour, and follow that with a glass of water; in addition, take all the hot salt broth you can." We teach him to examine his urine for acetone and tell him to do so every two hours, and as long as acetone is present to give himself insulin (regular soluble) after each urine examination until the acetone bodies disappear. The dosage of insulin is determined by the urine analyses for sugar. He is told to take 25 units if the result of the test is yellow or red, 15 units if the specimen is green, and the juice of an orange if the Benedict solution is unchanged after boiling. This simple rule also protects the patients against insulin overdosage. We have had such a patient come to see us on Friday and return on Monday weighing 8 pounds (3.6 Kg.) more and acetone free. Although the glycosuria was still present, she retained most of the fluid taken.

In the more advanced stages of ketosis the patient is admitted to the hospital and put to bed. Warm bags are applied to the extremities and he is wrapped in blankets. We cannot give this patient fluids by mouth because he is vomiting, so fluids are introduced by clysis. What kind of fluids? Dextrose solution, and since the patient is losing salt while losing water we give a saline solution of dextrose. It is well to start with 1,000 cc. of 5 per cent dextrose in saline solution. How much insulin? That varies; anywhere from 25 to 50 units. All agree that an amount above 50 units is unnecessary. Perhaps even smaller doses at frequent intervals are more effective than a single large dose. We have given 35 units at the start. The urine specimen is examined every hour

or hour and a half for acetone bodies, and the administration of soluble insulin is continued until the ketone bodies vanish. As soon as the ketone bodies begin to disappear, the patient's flush vanishes, the tongue becomes moist, the cheeks fill out a bit and he is able to take some fluids by mouth. Salty broth is continued, also milk and orange juice are given, and the urine is re-examined frequently. The administration of insulin is continued for a day or two after the ketone bodies have disappeared.

How much insulin should be given after the ketone bodies have disappeared? We use the simple guide just mentioned: If the urine specimen is yellow by the Benedict test we give 25 units; if it is green, 15 units. If it is blue we give only orange juice. After a two-day period the patient will be able to take food by mouth, and he can then be regulated as any diabetic patient without ketosis.

As to the differential diagnosis of acute surgical conditions of the abdomen and ketosis, when a patient is admitted presenting the syndrome which I have outlined and we do not know the diagnosis exactly, my policy has been to postpone the operation if possible until the patient is ketone free. If he is ketone free and the abdominal symptoms persist, then I think he is entitled to an exploratory operation. There is one other test which I have found useful; namely, study of the leukocytes, not their number but rather the characteristic toxic granulation. I recall two instances of diabetic ketosis simulating surgical abdominal involvement without elevation of temperature or pulse rate, and with only 8,000 leukocytes in the peripheral circulation. The abdomen was tender with some rebound tenderness, and many granules were seen in the leukocytes. The acetonuria disappeared after insulin therapy, and as the patient appeared to improve surgical intervention was postponed. After two days of relative quiescence the temperature suddenly rose and an exploration revealed a gangrenous appendix. The other patient showed the classic picture of diabetic ketosis with dominant abdominal symptoms. The leukocyte count was not high, but toxic granules were present. In this case we did not wait too long. Insulin and an infusion were given and an acutely involved appendix was then removed at operation. The patient in whom ketosis is present without abdominal complications may show an even more marked leukocytosis, with a count up to 50,000 and more but, the absence of toxic granulation is conspicuous. I do not wish to convey the thought that the presence or absence of cells containing toxic granules is an infallible guide. It must be evaluated in relation to the other clinical and laboratory data.

HYPOGLYCEMIA

The other diabetic emergency is hypoglycemia. The syndrome which is caused by hypoglycemia is not the same with regular insulin as with protamine zinc insulin. The differentiation is very important. With regular insulin the patient has ample warning. He becomes nervous and appre-

hensive, begins to sweat, has headache, his memory becomes foggy, there are visual disturbances, and he realizes that something is wrong. Orange juice will correct the difficulty immediately. With protamine zinc insulin the onset of these symptoms is rather insidious. The only trouble may be headache; it may continue for six, eight or ten hours, and then the patient may go precipitously into hypoglycemic coma from which he cannot be aroused. We therefore tell patients who are receiving protamine zinc insulin "If and when you experience any unusual symptoms, no matter what they are, please take some orange juice at once and follow that up with milk and crackers." The reasons for that are probably obvious to all of you. The orange juice will counteract the existing hypoglycemia, while the milk and crackers, a cereal or bread will continue to supply carbohydrate to combat the prolonged action of the protamine zinc insulin.

The preparation of the surgical patient is a problem that all of you will encounter, and it is not difficult. With the emergency surgical condition such as acute appendicitis in diabetes, the patient is taken to the operating room. There he is given an intravenous clysis of 5 per cent dextrose in 1,000 cc. of saline solution, together with 25 units of insulin hypodermically. When he is returned from the operating room, the procedure is repeated. That will insure a flow of urine, which is frequently examined. The dose of insulin which is then given is determined by the rule I have already stated. 25 units if the reaction is yellow, 15 units if it is green, and only orange juice if the patient can take fluids by mouth and the reaction is blue. Continue with that until the patient is able to eat. Then give a simple diet and regulate it in the customary manner.

With the surgical condition in diabetes which is not an emergency, the procedure is slightly different. We all know that patients in the operating room lose a good deal of fluid, and I prefer to hydrate them for a longer period before operation. With those patients we advise the ingestion of table salt in a dose of 1 Gm. (two half gram tablets) every hour with a glass of water, for about ten or twelve hours before operation.

To recapitulate briefly: The most serious emergencies of the diabetic are (1) ketosis and (2) hypoglycemia. The treatment of ketosis is based on two definite scientific principles, the replacement of fluid and salts and the use of insulin. The treatment of hypoglycemia is based on a sound scientific principle as well, the administration of sugar. For the rapidly acting insulin sugar may be given in the form of orange juice and for the slowly acting protamine zinc insulin milk and crackers or some form of carbohydrate that is slowly absorbed so that it can neutralize the prolonged effect of the insulin.

There appears to be more to the mechanism and the treatment of diabetic coma than has been indicated in this discussion. There are factors about which a good deal still needs to be learned. We should not gain the impression that by the

treatment outlined we are able to save all patients with diabetic coma. Under the most favourable conditions and the best treatment known, many of the patients still die, for reasons which in the present state of our knowledge remain unknown. This appears to be the general experience.

DISCUSSION OF QUESTIONS

Dr. Du Bois: Do you have much difficulty in getting a patient to take tablets containing salt (sodium chloride)?

Dr. Tolstoi: No.

Dr. Du Bois: How do you give them?

Dr. Tolstoi: Just let the patient swallow the tablet. If he cannot swallow the tablet, crush it and follow it with a drink of water.

Dr. Harry Gold: One might give about one-sixth teaspoonful of ordinary table salt, could one not?

Dr. Tolstoi: Yes. We prefer the tablet form. It is convenient. However, salt in any form will do.

Dr. McKeen Cattell: Your procedure is based entirely on the matter of replacing salt and fluid which is below normal?

Dr. Tolstoi: That is right. The diabetic patient in coma does not retain dextrose solutions alone for any length of time. The fluid is rapidly lost. The addition of salt helps to fix the fluid in the body. I am sure that some of the hospital staff will recall the patients who have come into the house as little shriveled up individuals and by the next day they seem like balloons, with big puffy cheeks and eyes that seem small, from edema resulting from the salt water treatment. Of course, as soon as the salt is discontinued the water flows off and the patient seems more normal.

Dr. Samuel B. Barker: Don't you think that the salt may have some effect in replacing the deficient base and in the differential excretion of chloride perhaps as ammonia chloride? That, of course, would not be a rapid process. Undoubtedly the main thing would be the salt itself, but don't you think the retention of base from the chloride would be an important effect of salt administration?

Dr. Tolstoi: That is quite true, because in these cases acetone is being produced all the time, and to neutralize that we draw on the body's supply of base. There are other points, too controversial, perhaps, to discuss here, namely, the administration of sodium bicarbonate, gastric lavage and lactate-Ringer (Hartmann's) solution. I don't recommend them nor do I object to their use.

Dr. Du Bois: Of course, the question as to whether or not sodium bicarbonate should be used is at least thirty years old. The point about keeping the diabetic patient wet is also fairly old. I remember that at least twenty-five years ago Theodore Janeway used to insist on that: the wet diabetic patient did not go into coma.

Dr. Barker: will you continue the discussion of diabetic acidosis?

Dr. Barker: The term "acidosis" is rather unfortunate. It was introduced by Naunyn, who

intended it to designate the production in diabetes of beta hydroxybutyric and acetoacetic acids, a situation which is now rather well covered by "ketosis." Acidosis has come to have such broad implications that is often used to include practically all aspects of the condition resulting from the production of these two acids. The word itself is an exaggeration: the blood is seldom actually acid, in the sense that its pH falls below 7.0, and often considerable amounts of ketone bodies are found in the blood without any deviation from the normal range of 7.3 to 7.5. Probably the best definition of the present-day use of the term is that suggested by Van Slyke and Cullen: a lowered alkali reserve resulting from acid production. In the case of diabetes, acid production would be the production of beta hydroxybutyric and acetoacetic acids.

At the start of this short discussion on diabetic acidosis I should remind you that the fundamental cause of the extensive acid-base disturbances found in diabetes is the deficient carbohydrate oxidation. This leads to a deranged fat metabolism, with large amounts of the ketone bodies being produced from the incomplete oxidation of fatty acids. These are neutralized by means of base taken from BHCOS_3 , the carbon dioxide freed being eliminated by the lungs. The kidneys assist in the attempted regulation of normal equilibrium by excreting the acids partly in the form of free acid and partly in the form of ammonium salts. The kidney is able to form ammonia in order to conserve fixed base (that is, sodium). At this stage no change has yet been produced which is not rather easily reversible by remedying the primary cause, namely, the poor oxidation of carbohydrate. The proper procedure is, of course, the administration of carbohydrate and insulin. This will stop the acid production and assist in the oxidation of any accumulated ketone bodies. The base combined with these acids is thereupon released and serves to return the carbon dioxide combining power of the blood to normal.

However, if the diabetic condition is allowed to continue unabated, the organic acid load increases and the alkali reserve of the blood is still further reduced. Ammonia production by the kidneys is unable to keep up with acid excretion, and the use of fixed base is resorted to. This still further reduces the ability of the blood to carry carbon dioxide and at the same time lowers the total salt concentration. This hypotonicity is at once remedied by the excretion of water until the proper salt concentration is reached. This results in a temporary isotonicity but also produces a lowered total content of electrolyte and a lowered blood volume.

As the condition progresses still further, the amount of base available to carry carbon dioxide becomes small and the base from chloride seems to be freed to neutralize acid material. Hydrochloric acid often is vomited or may be excreted by the kidneys as ammonium chloride. The depletion of alkali and long continued glycosuria eventually lead to serious dehydration, hyperpyrexia

and hyperpnea. In the extreme condition of diabetic coma, desiccation is pronounced, blood volume, alkali reserve and base content are very low, and the blood pH may be below 7.1.

The changes as sketched here have been related to blood but of course apply to all extracellular body fluids. The cells also are hit by the loss of salt and water, but they are most affected by the self destruction induced by the increased breakdown of body protein. All the changes described are gradual; conditions merge into one another with no sharp demarcation. In addition to the tissue changes, there is pronounced dyspnea caused by the need of eliminating large amounts of carbon dioxide aggravated by hyperthermia and sometimes by increased metabolism.

The first line of treatment must still remain the alleviation of the defective carbohydrate oxidation with insulin, coupled with the administration of carbohydrate to replace the lowered body stores. Beyond that, one may look toward restoring the water and the electrolyte balances. The patient is dehydrated but, as Dr. Tolstoi has mentioned, the administration of water alone will not afford any permanent relief. The need for base is acute, but it is undoubtedly best to supply this as sodium chloride rather than as sodium bicarbonate, for two principal reasons: First, there is often a need for chloride as well as for base. If base is needed in larger amounts than chloride, the kidney is quite capable of excreting the excess of chloride, as ammonium chloride, and the fixed base is retained. It is furthermore quite possible that the administration of sodium bicarbonate even in extreme diabetic acidosis may overcompensate and cause a dangerous alkalosis, when insulin is also used.

It may be reemphasized that the most logical treatment for the alleviation of diabetic acidosis is the use of insulin plus sugar, combined with salt administration.

Student: I should like to ask Dr. Barker if he considers that the danger of acidosis is due primarily to dehydration.

Dr. Barker: Dehydration certainly is an exceedingly serious aspect of untreated acidosis.

The diminished alkali reserve, lowered blood pH, hyperglycemia and other carbohydrate disabilities, fatty infiltration and degeneration are all important involvements in diabetes and all are precursors of dehydration. But the dehydration and lowered blood volume are undoubtedly the factors which hit the hardest, in the final analysis.

The administration of base was an early treatment, but bicarbonate administration did not help permanently. Magnus Levy's old idea was that one should cause the patient to excrete as much acetone as possible; that it was more important to eliminate it than to keep it. Actually, an excretion of acetone bodies in the urine of a normal persons can be caused by giving a large amount of base.

Dr. Tolstoi: Isn't there a feeling among cardiologists that the reduction of blood volume is



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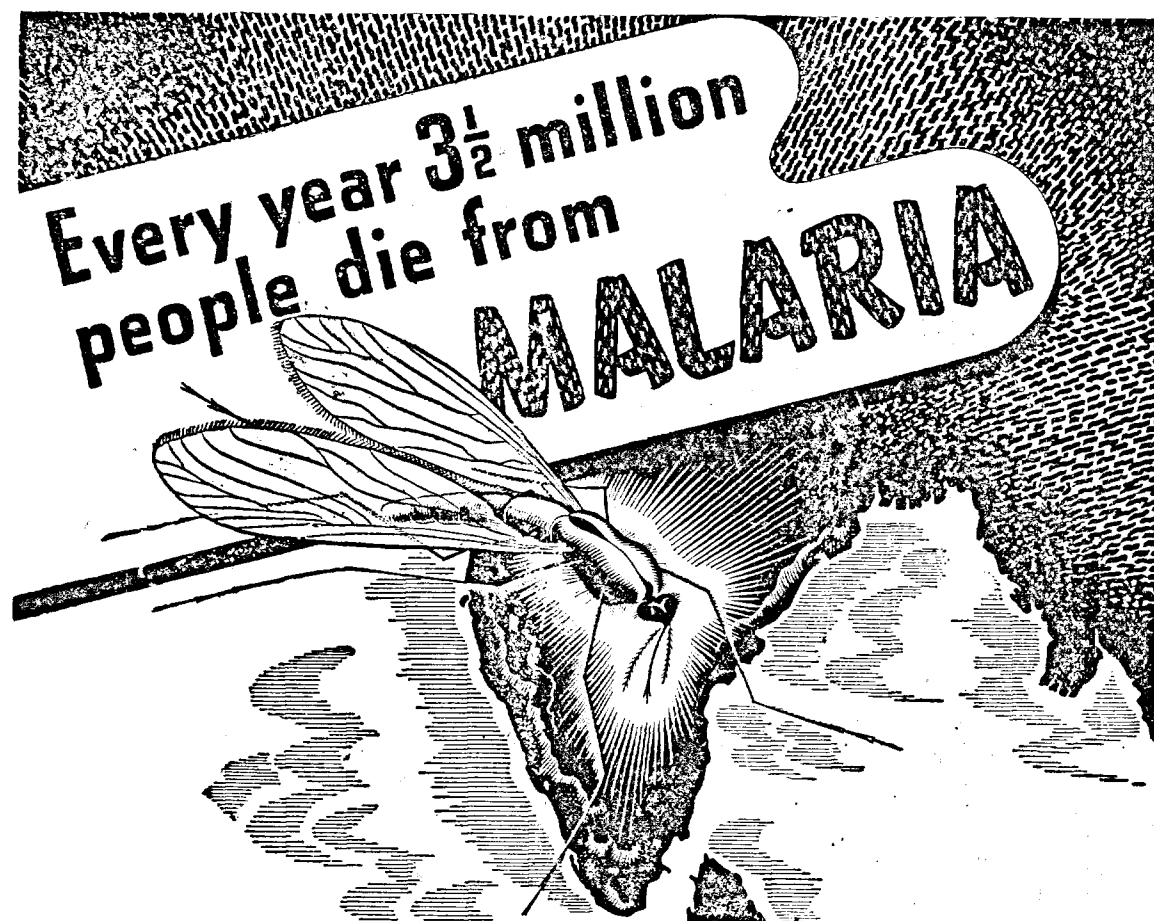
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detrimental to the heart of the diabetic patient who has lost a lot of fluid and is one of the causes which finally is responsible for death?

Dr. Ephraim Shorr: With respect to the cardiac accidents, we should remember that the proper function of the heart is dependent on a normal type of metabolism, a situation which does not exist in the absence of insulin. It seems to me that to regard cardiac failures and irregularities that occur as due to just one factor, such as the dehydration, is possibly a little unsafe. In working with hearts of diabetic dogs we have been struck with the rather profound changes which occur in the texture of the muscle. The diabetic heart becomes fragile and shreds easily. There are also some histologic deviations from the normal. Therefore I think more than just one factor is responsible for the failures which occur.

ROLE OF THE PITUITARY

Dr. Du Bois: I will ask Dr. Chambers to tell us something of his work on the role of the pituitary.

Dr. William H. Chambers: There are several excellent reviews of this subject, by Houssay, in the May 1937 issue of the *American Journal of the Medical Sciences*, also by Long and Britton and by Riddle in the *Cold Spring Harbor Symposia* for the summer of 1937 and a review article by Russell in *Physiological Reviews* for January 1938. These reviews discuss the effects of the different endocrines on diabetes. Many different types of animals, from the toad to the dog, are used and it is difficult to put the data together and form any consistent picture.

The depancreatized dog is in a completely diabetic condition when insulin has been withheld for three or four days, or three or four days after the operation when the animal has been given no food. You are quite familiar with the picture: respiratory quotients of 0.70 to 0.73 in the basal condition and no rise in quotient with the introduction of dextrose. The D:N ratio is 2.8. This condition prevails for about two weeks of fasting, until the animal loses from 45 to 50 per cent of its body weight. At that time the creatine excretion rises very definitely in the depancreatized animal. Sometimes the nitrogen excreting increases. This is associated with a rise in the respiratory quotient to the protein level, indicating that the protein is handled as a normal animal would take care of it.

Barker has studied this condition in our laboratory and found that the rise in the protein metabolism is not an essential part of the picture. At this time the acetone bodies may disappear completely and the D:N ratio may fall to zero, meaning no dextrose excretion at all. When dextrose is given the respiratory quotient shows a rise, indicating that some of the dextrose can be oxidized. Here is a condition where the animal in the absence of insulin can oxidize some sugar.

Now, a similar situation is found, as you know, in the Houssay animal. Houssay in 1929 pointed

out that the removal of the pituitary would change the diabetic picture, lowering the D:N ratio, reducing the ketosis almost to zero and reducing the glycosuria. We studied several animals of this type in the calorimeter with the help of Dr. Chandler and Dr. Sweet and found that the respiratory quotients under basal conditions would range between 0.74 and 0.78. That is, the animals under basal conditions were burning some sugar at all times.

But when we gave them dextrose by mouth the injected sugar apparently was not burned. That is, there was little rise in the respiratory quotient, only five out of fifteen experiments showing any definite increase. About 60 per cent of the sugar was excreted. So we attempted a different type of experiment, one of which may serve as an illustration. The data are from one of nine dogs with which balance studies were made in the following fashion: The animal was given meat twice daily and put in the calorimeter for eight hours during the course of the day. The metabolism when the animal was quite in the cage at night was probably quiet equivalent to that in the calorimeter in the dark during the daytime.

We found that when the animal was on a pure meat diet for five days the D:N ratio was 2.45, that it excreted 40 Gm. of dextrose and the equivalent of 16 Gm. of nitrogen a day and that the respiratory quotient during the day averaged 0.75. This was not changed by the introduction of the meat. On the basis of a respiratory quotient of 0.75, in twenty-four hours the animal would oxidize about 30 Gm. of sugar.

In a subsequent period of three days we added to the meat, given twice daily, 30 Gm. of dextrose and some lard. The animal then excreted about 15 Gm. of the 30 Gm. which was fed over the amount which might be derived from the protein according to the D:N ratio. In other words, the animal was oxidizing 30 Gm. from the protein and 15 more from the extra dextrose, and 15 Gm. was recovered in the urine. The respiratory quotient rose to 0.78. Calculating from the heat production and the respiratory quotient, we find the dextrose oxidized in twenty-four hours to be about 50 Gm.

In a later experiment the animal was given 100 Gm. of dextrose a day for four days and 85 Gm. was excreted; the oxidation remained practically the same and the respiratory quotient about the same.

In other words, these animals lacking a pituitary and a pancreas are able to oxidize a limited amount of dextrose. An increased amount in the diet does not change the amount oxidized, but it is merely excreted.

You recall that Long has removed the adrenals from depancreatized cats and found that the diabetes was ameliorated in much the same way that Houssay showed with the hypophysectomized diabetic dogs. We have studied four adrenalectomized depancreatized dogs. The data from these animals could be put with the data from the Houssay animals and no distinction found. The

D : N ratios and the respiratory quotients are about the same. The respiratory quotient shows no particular rise when dextrose is ingested. The removal of the adrenal seems to be quite comparable to the removal of the pituitary as far as carbohydrate oxidation is concerned.

In an article published in 1937, Long reported that adrenal cortex extract given in large amounts to rats which had been partially depancreatized produces a definite glycosuria. In the earlier reports, small amounts of cortical extract showed no influence on carbohydrate metabolism. We are not prepared to say from our data to what extent cortical extract may influence carbohydrate metabolism. In one adrenalectomized depancreatized animal cortical extract was withheld for six days. There was no difference in the carbohydrate metabolism that we could determine.

Another animal was hypophysectomized and studied, and later the adrenals were removed and the same experiments performed. The results were quite comparable.

So the removal of the adrenals after taking out the pituitary apparently does not change the picture further. Long has suggested, I believe, that the pituitary acts through the adrenal cortex. I think that this still remains to be demonstrated.

Now I want to take just a minute to show quantitative relationships in these different animals. The first animal—and these are round figures taken from the experimental data, estimates rather than actual figures—is a normal animal fed with plenty of carbohydrate in the diet. When given 50 Gm. of dextrose, it will absorb about a gram per kilogram hourly in the course of four hours. Of that amount 40 per cent is oxidized and 60 per cent deposited as glycogen. Whenever dextrose is given we find that it is not immediately completely oxidized but is partly deposited as glycogen. The Houssay animal will oxidize about 10 per cent of the dextrose given. According to Houssay's figures about 20 per cent in this four-hour period may be deposited as glycogen and the remainder, or about 70 per cent excreted.

The completely depancreatized animal will oxidize none. Glycogen deposition has been shown to take place in the absence of insulin if the blood sugar is raised to an abnormally high level. But when the dextrose is given by mouth the blood sugar is not high enough to show a definite deposition of the glycogen and 100 per cent of the dextrose can be recovered in the urine.

We see on a quantitative basis that removal of the pituitary, and we might put the adrenal in the same category, does not by any means restore the animal to a normal position, either with respect to oxidation of dextrose or with respect to the deposition of glycogen.

There is one other complicating factor in the relationship of the pituitary and the adrenal. It is apparent from this picture that we did not obtain 100 per cent recovery from the diabetic condition by any means. In two animals we did find practically a complete restoration of ability to oxidize dextrose. One of these was a Houssay

animal which showed in the histologic sections a very definite lesion in the region of the hypothalamus but a perfectly normal adrenal cortex so far as we could tell. The other animal was a depancreatized animal which never showed glycosuria. We could find no pancreas at autopsy. The animal burned sugar in normal fashion but the autopsy showed a cyst in the posterior lobe of the pituitary and marked atrophy of the adrenal cortex. So I think that the explanation of the parts played by the pituitary and the adrenal cortex is still decidedly theoretical.

DIABETIC TISSUE

Dr. Du Bois: We have heard about the diabetic patient and the diabetic dog. I wonder if Dr. Shorr will say a little about the diabetic tissue that has been studied.

Dr. Ephraim Shorr: We have come to regard diabetes as a disturbance of the mechanism by which the various ductless glands regulate sugar metabolism, rather than a defect exclusively associated with an insulin deficiency.

It seems probable that tissues have a fundamental capacity to metabolize a wide variety of foodstuffs, including carbohydrates, which is essentially independent of the secretions of the ductless glands. This is evidenced by the observation that embryonic tissues are capable of sugar oxidation before the ductless glands appear. As organisms became more complex with evolution, the ductless glands developed and assumed control over this function, among others. This loss of independence was one of the penalties the tissues had to pay for their greater complexity and higher organization. It is amazing that, despite the millions of years of such servitude, an organism as well as isolated tissues can still give evidence of this primitive independence and burn sugar without the assistance of insulin.

Some experiments in our laboratory have a bearing on this point. You will recall that Dr. Chambers showed that animals with pancreas and pituitaries removed were capable of oxidizing more carbohydrate than animals with pancreatic diabetes. The improvement was, however, only partial. We studied the respiratory metabolism of tissues isolated from these animals and found that they were capable of an entirely normal sugar metabolism. It is apparent from these experiments in the Houssay animal that there must still exist other factors which inhibit sugar metabolism apart from the pituitary secretions. We then asked ourselves whether the superiority of isolated tissues over the whole animal might not reside in the degradation of these other inhibiting factors with time, when the tissues were removed from the body. This theory was then tested by the study of tissues from diabetic animals in the following manner. They were removed from the animals under sterile conditions. One portion of tissue was studied at once and found, as one might expect, to have no capacity to oxidize sugar. Another portion was incubated for ten hours at body temperature under proper

conditions. At the end of ten hours, these tissues were found to have regained the normal capacity to oxidize carbohydrate. This occurred independently of insulin. Our working hypothesis is that during the period of incubation the inhibiting influences, whether pituitary or adrenal in origin, were used up. Now the tissues were free both of the sugar-accelerating hormone (insulin) and the inhibiting hormones and had resumed their innate capacity to oxidize this foodstuff without any hormonal help.

In view of what I have said, you may have been puzzled about the absence of any influence of this conception on the present treatment of diabetes, which you have just heard outlined. What you have heard has been a careful therapeutic regimen based largely on the conception of diabetes in the human being as a disease due to deficient insulin production. This does, indeed, represent a lag between the results of animal experimentation and their application to human disease. It is warranted, however, for the present, on several counts. The chief reason for this apparent disregard of polyglandular factors in human diabetes is that we know but little about the role of the factors, other than insulin. This difficulty in correlating animal experiments with human disease is also seen in exophthalmic goiter, in which, despite our considerable knowledge of the presence of thyrotropic pituitary factors which produce a comparable condition in animals, little or nothing is known of the way in which these pituitary factors act in the production of exophthalmic goiter.

We must also recognize that, while human disease is in many instances comparable to that produced in animals, there is a tendency for conditions in the human body to become more stable after an initial disturbance than in animals, where it is possible to influence the ductless glands in a much more profound manner. This stabilization of the human organism can also be seen in disturbances of sex physiology, in whose treatment various hormonal principles, particularly those of pituitary origin, have been much less successful than in animals.

Finally, there is some suggestion from the work of Young as to the way in which these two points of view, the polyglandular disorder as against the relatively stable metabolic disturbance dominated by one secretion, can be reconciled. Young has been able, by injection of pancreatic extracts into animals, to produce degenerative changes in the pancreas which are followed by diabetes. This diabetes differs in no way from the diabetes produced in animals by complete removal of the pancreas and is, once degeneration is extreme, a relatively stable condition. This state produced by pancreatic extracts might be the prototype of human diabetes.

The newer conception of diabetes, however, is so recent and so inadequately explored in the human being up to the present that we should not be discouraged about the fact that it is not reflected in our standard treatment of the disease. A few years may well bring about an entirely new picture

as a result of insight gained in the possible mechanisms underlying this disease.

NATURE OF INSULIN

Dr. Du Bois: I would appreciate it if Dr. Du Vigneaud would make a few comments on the present stage of development of the chemistry of insulin.

Dr. Vincent Du Vigneaud: Insulin is a protein. The molecule is made up of amino acids, and of such a number of amino acids that immediately possibility of synthesis is out of the question at present. When you hear individuals talking about synthetic insulin you can rest assured that they have no concept of the subject, because that is out of the question at the present stage of development. Probably no subject in the field of biochemistry is at present being attacked more diligently than the chemistry of the proteins. It is particularly interesting to note that various laboratories of classic physical chemistry which just a few years ago would not think of working on a biologic substance are working on proteins from a physicochemical standpoint. This is indeed a significant development. Anything done on the chemistry of proteins will in turn throw light on the chemistry of insulin. Also anything done on the chemistry of insulin in turn has its significance to the chemistry of proteins.

The fact that insulin is a large protein molecule with molecular weight of about 35,000 leaves the hope of oral administration at the present time somewhat discouraging. It is true that insulin is destroyed by certain proteolytic enzymes, but that is not such a formidable barrier as the size of the molecule because there are various ways to get around the enzyme destruction. The real problem is the fact that the molecule is so large that it is difficult for appreciable amounts to pass through the membrane of this intestine. One approach is to attempt to dissociate this large molecule into smaller units and still retain activity.

An interesting chemical development has been the recognition by many that zinc must be present in order to obtain a form of crystalline insulin. The crystalline insulin is a zinc complex of this protein. Much work has been done to find out whether other metals will serve. Of all the other metals that have been tried, cadmium, cobalt and nickel are the only ones that enable the insulin to be crystallized. The crystallization of insulin is now so successful that it is being done commercially and a solution of crystalline insulin is on the market. The relationship of these metals to the insulin and the nature of the linkage are attracting the attention of our group and many others.

Much work is still going forward on the composition of insulin. The entire insulin molecule is still not completely accounted for. Considerable work is also going forward on various physicochemical properties of this interesting protein.

Student: Why is zinc used rather than any of the other metals you mentioned?

Dr. Du Vigneaud: The fact that zinc was the first metal to be encountered in the work is

perhaps one of the reasons. Furthermore, the pancreas contains a considerable amount of zinc and this likewise might have influenced the choice of zinc. The crystals we have so far made with cobalt are tinged a light pink and the nickel crystals have had a green tinge. The fact that the zinc crystals are colourless and the fact that zinc occurs in the pancreas are perhaps the main factors in favour of the choice of zinc.

Dr. Cattell: Is it known how the zinc acts to change the character of the molecule in relation to the matter of absorption?

Dr. Du Vigneaud: Insulin free of zinc is physiologically active. If you assay one against the other carefully you do not find much difference in spite of the claims to the contrary. However, there is a real difference between protamine insulin and insulin. It is suspected that zinc aids in the combination of protamine and insulin. If there is any difference between zinc insulin and zinc free insulin, which as I say is questionable, I think it would rest on the possibility that zinc insulin has more of a chance of attaching itself to certain proteins at the site of injection and delaying absorption a little bit.

(Continued from page 362)

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Chrysotherapy in pulmonary fibrosis with cavitation—Dastidar ... 155

(4) Vitamin C in pulmonary tuberculosis—Hai ... 163

(5) Some experimental observation on sulphonamide drugs in rabies—Prasad ... 175

(4) Hai points out that marked vitamin C sub-nutrition has been found in patients with severe pulmonary tuberculosis; that daily excretion of ascorbic acid in the urine of these patients have been found to be markedly decreased; and that therapeutic trials lend support to the heavy demand of vitamin C by these patients.

(5) Prasad states that solu-septasin appears to have a bactericidal effect in vitro in dilutions up to 1:160.

The Editors of the *Patna Journal of Medicine* announce that a medico-legal supplement will be appearing henceforth with the Journal, more or less as the mouth-piece of the Medico-Legal Society Bihar. The first supplement presents the following articles:

- (1) Determination of age by X-ray examination
- (2) The doctor in court
- (3) Crime and insanity
- (4) Modern Indian problems in psychiatry and criminality.

We believe that this supplement will be greatly appreciated by the profession.—*Ed. I. M. J.*

JR. OF SOUTH IND. MED. OCT. '40

Pulmonary tuberculosis in infancy and childhood—Srinivasan ... 61
Anemia—Subramanyam ... 68

Special Journals

IND. JR. OF OPHTHALMOLOGY OCT. '40

- Tonometric observation of the normal intra-ocular pressure of Indian eyes—D. D. Sathaye... 150
(6) Treatment of trachoma—Kothare ... 155
Sclero-malaria perforens—Hansraj & Dalal ... 157
Sulphanilamide in eye diseases—Krishnamurthi ... 159
Discussion for high myopia—V. D. Sathaye ... 164
(6) Kothare writes that the model treatment of trachoma will be silver iodide in acute stage continued for a period varying from a fortnight to a couple of months according to the severity of the case, and glycerine copper in chronic cases in the third stage.

INDIAN MEDICAL NEWS

(From Our Own Correspondent)

LOCATION OF TUBERCULOSIS CLINICS

That if a tuberculosis clinic is to be of maximum benefit, it should be situated in, or as close as possible to, thickly populated area, is the unanimous opinion of the Committee of Experts recently appointed by the Tuberculosis Association of India at the instance of the Government of India to consider what condition should govern the selection of sites for such clinics and whether any particular precautions were necessary in the case of clinics situated in populated areas.

The Committee were also of the view that no particular conditions are necessary regarding the distance of a well-conducted clinic from the nearest house. They recommended, however, that if a clinic is located in a part of a building used for other purposes, the clinic should have a separate entrance.

PROGRESS OF INDIGENOUS MANUFACTURE

Owing to the extended production of drugs in India as many as 92 various drugs have been taken off the import list.

The manufacture in India of suitable disinfectants has been taken up by the Medical Stores Supplies Committee under the Chairmanship of Lieut-General G. G. Jolly, I.M.S., Director General, Indian Medical Service. Attempts are being made to produce a complex chlorine compound hitherto imported which is essential for the manufacture of many non-irritating efficient disinfectants which are now so largely used. A dye called acriflavine is also an important disinfectant, particularly in war time, and its manufacture in India from locally produced materials is under investigation.

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A matter of Taste

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● Literature with full citations of scientific work and publications on Scott's Emulsion will gladly be sent to any medical practitioner on application to Imperial Chemical Industries (India) Ltd., Calcutta, Bombay, Madras, Rangoon or Colombo.

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Bulk of the Indian diet consists of carbohydrates. These carbohydrates comprise of rice, bread, potato, all sugars, sooji, etc. These are digested by the enzymes of a special type such as diastase or amylase present in the saliva, intestinal juice and the pancreatic secretion.

Carbohydrate dyspepsia of the small intestine and carbohydrate fermentation and acidity are very common. They are due to the faulty secretion of these enzymes in quality and quantity. Bulk of the diet, particularly carbohydrates, tax the secreting mechanism of the glands by putting constant strain on these. The result is the undigested foods particularly carbohydrates, kept long in the stomach, produce heaviness, acidity and heart-burn.

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Samples have already been produced in an Indian laboratory.

Troops in the field deprived of fresh fruit or vegetables develop scurvy. To prevent this disease tablets of vitamin C are made up and India's most concentrated form of vitamin C is "amla". Amla berries are collected in the Nilgris under the supervision of the Director, Nutrition Laboratories, Coonoor, dried and made into tablets of suitable size. The Committee is now contemplating the production of a more concentrated form of vitamin C.

Substitutes for corks are being considered. These have become expensive and difficult to obtain owing to the war. Samples of wooden corks made by the Forest Research Institute, Dehra Dun, having two circular slots to increase resilience are being examined and are to be tried out on a large scale. Bottles with threaded glass necks and metal caps as well as bottles with crown corks are also considered as possible substitutes for bottles with corks.

MEDICAL STORES SUPPLY COMMITTEE MEETING

Many problems affecting medical supplies to the Army were considered at the third meeting of the Drugs Supplies Committee, now called the Medical Stores Supply Committee, held recently under the chairmanship of Lieut-General G. G. Jolly, I.M.S., Director-General, Indian Medical Service.

Samples were shown of Emetine Hydrochloride, Apomorphine Hydrochloride, Acriflavine and Dried Blood Plasma made in India under schemes of investigation fostered by this Committee in close liaison with the Board of Scientific and Industrial Research of the Commerce Department.

To combat shock through loss of blood, blood transfusion is largely used in modern medicine. Such blood is difficult to obtain in quantity for the treatment of the wounded in the field and other methods are being carefully considered. One of the most successful and promising of these methods is the use of dried blood plasma. Blood to which sodium citrate has been added to prevent coagulation is allowed to settle and the clear liquid drained off. This liquid is dried *in vacuo* to a granular powder and packed aseptically in containers. Before use it is made into a solution with sterile water and given to the patient in the same manner as a blood transfusion. A sample of dried blood plasma, the first to be made in India, was shown to the Committee which discussed the question of its manufacture in India.

The production of emetine hydrochloride which is still required for the treatment of amoebic dysentery is closely associated with the growth of ipecacacuanha. This drug grows wild in the forests of South America but is more and more difficult to obtain, and further the forests have to be penetrated. Ipecacacuanha however has been demonstrated to grow well in Mungpoo, Bengal, and the question of encouraging its cultivation in India is under consideration.

Much progress has recently been made in Travancore, Bombay and Madras in the development of fish liver oil schemes for supplying India with substitutes for Cod Liver Oil. The essential factor in these oils is their content of vitamin A and vitamin D. As the shark Liver oil has ten times the vitamin A content of Cod Liver Oil it forms a valuable source of vitamin A. This oil however is lacking in vitamin D so that it is necessary to add this vitamin to it. Vitamin D, a complex chemical substance, has now for the first time been made in India at the Indian Institute of Science.

MEDICAL RELIEF IN MYSORE

It is interesting to note from the report of the Senior Surgeon in Mysore for the calendar year 1939, that Mysore occupied a unique position in the field of medical relief.

The report revealed that in Mysore there was one medical institution on an average for every 87 square miles, one for every 20,653 of the population and spent Re. 0.4-6 per capita. Comparatively speaking, Mysore stood in the forefront of all other British Indian Provinces and States in this respect.

The report further stated that last year 20 new institutions were opened including two tuberculosis institutions, six local fund dispensaries on a reduced scale, nine subsidised rural dispensaries and three malaria relief centres. On the whole, as many as 3,210 beds were available for in-patients.

The Government spent on the medical department Rs. 19,82,217 during the year, the contributions from local bodies and the receipts from hospital fees being respectively Rs. 3,40,000 and Rs. 1,17,213. A special grant was made during the year amounting to one lakh of rupees, for providing up-to-date surgical instruments and other equipments to the major hospitals in Bangalore and Mysore, the district hospitals and certain selected Local Fund dispensaries; so as to bring the most modern method of diagnosis and treatment within the reach of the rural population.

U. P. MEDICAL CONFERENCE

The seventh U. P. Medical Conference held under the presidency Dr. Banarsi Das, Superintendent of the Agra Mental Hospital, concluded its session on 29th October.

The conference urged that, whereas the present Indian Medical Council Act excluded a large number of qualified medical men possessing registrable qualification in the various provinces of India from its purview and thus imposed a severe disability on them, the Act should be so amended as to include all grades of qualified medical practitioners in its fold, to permit recognition of the medical qualifications of all other countries on the basis of reciprocity alone, and to provide for a majority of elected members on the Council. The Conference requested the Provincial Government to approach the Central Government to amend the Indian Medical Council Act accordingly.

Epitome of Indian Medical Literature

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This is a Special Number devoted to the discussion of certain aspects of tuberculosis. Frimodt-Møller stresses on the need for organized home treatment. He warns against the imitation of Western methods of control, and advises a 'balanced scheme' of tuberculosis policy, propaganda, clinics, colonies, and home patients. Ukil draws attention to some chronic non-tuberculous infections of the lung simulating pulmonary tuberculosis in symptoms and signs, clinical manifestations and even X-ray findings. These diseases are according to him: (1) unresolved pneumonia, (2) some sequelae of broncho or lobular pneumonia, (3) chronic bronchitis of infective origin, (4) bronchiectasis, (5) chronic pulmonary abscess, (6) pulmonary syphilis, (7) hydatid cysts of the lung, (8) pulmonary mycoses, (9) injected pulmonary neoplasms. He gives illustrative case under each head. Benjamin and Verghese discuss pulmonary tuberculosis in relation with a co-existing diabetes mellitus, and are of opinion that the type of tuberculosis in diabetics is not materially different from that in non-diabetics in India except that exudative reactions are more severe. They also believe that diabetes is not a contra-indication to any of the usual forms of treatment of tuberculosis, including surgery. They however point out that haemoptysis is seen more frequently in the diabetic group and the immediate results of treatment are slightly below the average for non-diabetics. Jones writes that spontaneous pneumothorax, in the normal as well as in the tuberculous, is not uncommon, but that the treatment is often difficult and requires prompt attention. Samuel discusses the treatment of tuberculous empyema after classifying it into four groups:

- (1) pure tuberculous empyema without pulmonary tuberculosis,
- (2) mixed pyogenic and tuberculous empyema without pulmonary tuberculosis,
- (3) pure tuberculous empyema with active pulmonary tuberculosis,
- (4) mixed tuberculous and pyogenic empyema with active pulmonary tuberculosis.

He enunciates the following principles of treatment:

- (1) if the underlying lung is tuberculous and requires thoraco-plasty for itself, then the empyema of the lung should be treated by thoracoplasty.
- (2) if the underlying lung is not diseased or if it is tuberculous and for itself does not require thoracoplasty, the empyema should not be treated by thoraco-plasty unless aspiration treatment fails.
- (3) Thoraco-plasty should be used only when mixed infection exists and high toxemia therefrom, when it should be done immediately.

Ghosh, discussing the effects of tuberculosis on pregnant women, urges great care before and after parturi-

tion. He also describes the effects of pregnancy, delivery and puerperium upon the course of the disease. Two forms of tuberculous attack, he writes, seem likely to affect appreciably the fertility of women of child-bearing age. Ray and Sen describe a balanced diet for the tuberculous patient. They warn against "roughage" in diet for patient suffering from tuberculous enteritis.

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(2) Madan enumerates various anesthetic agents and describes the precautions to be taken to decrease the toxicity and complications of each.

He lays down certain general rules. In shock, or when it is anticipated preliminary intravenous glucose should be given with insulin 1 unit for every 5 g. of glucose. In operations involving hæmorrhage, transfusion of blood by continuous drip method is useful during the operation. In cases where liquid volatile anesthetics, like chloroform, ether, cyclopropane, etc. are used. Soda bicarb a few days before operation should be given. Ketosis is combated by a preliminary saline enema. Rectal olive oil is useful after operation, as it ensures the restoration of certain properties in the blood which are inhibited by the anesthetic, e.g. phagocytosis, and it speedily restores the opsonic index. Pre-medication is also advised. Omnopon, nembutal, sodium soneryl and di landid hydrochl. are some of the drugs used for pre-medication, but the author recommends the last as the safest and best.

He warns against the use of avertin when there is even a slight dysfunction of liver or kidneys.

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(3) Ajinkya points out that the most common type of lesion to be met with in the cervix of a multiparous woman is the erosion. Careful treatment of tears and cervicitis might play an important part in the prophylaxis of cancer in addition to preventing and even curing many minor discomforts such as backache and leucorrhoea. He classifies cervical lesions as follows: traumatic, infective, neoplastic and neuropathic.

Cervicitis is often a cause of sterility. High cervical tears give rise to habitual abortions.

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CORRIGENDA

Vol. 34 No. 11

(November Issue 1940)

[Article on Gargoylism by G. R. Dinker Rau and A. V. S. Sarma]

- (1) In page 302, please read G. R. Dinker Rau instead of "C. R. Dinker Rao."
- (2) In page 306, legend to the upper right illustration, please read *Note the absence of hook-like deformity of the lumbar vertebrae* instead of "Note the hook-like deformity of the lumbar vertebrae."

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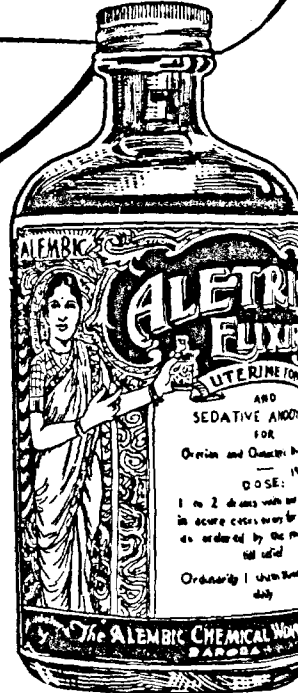
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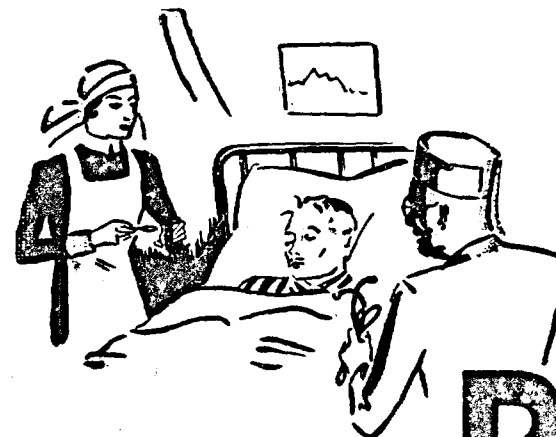
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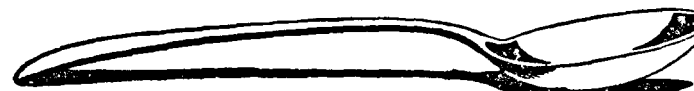
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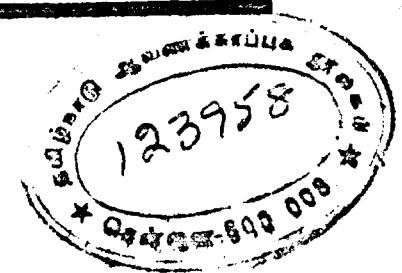
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