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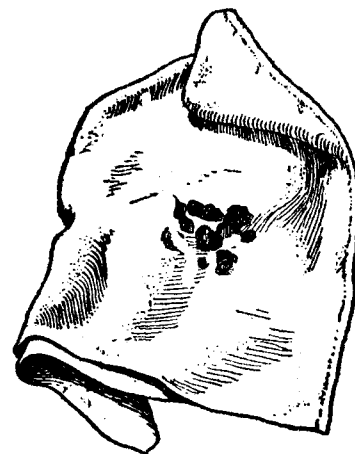
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The Madras Medical Journal.

JANUARY 1927.

ORIGINAL ARTICLES.

NEW IDEAS ABOUT THE DIAGNOSIS AND TREATMENT OF DIABETES MELLITUS - I*

(By Dr. B. V. VARADACHARIAR, B.A., M.B. & C.M.)

The subject matter of my lecture is based on the information contained in the recent books on Diabetes Mellitus. I do not lay any claim to originality in the views I am going to express, nor do I claim much practical experience. I have summarised the recent knowledge about the disease, more for my own information, than perhaps, for yours. For, I daresay, several of you have been closely following the development of the new ideas in this field and therefore what I am going to place before you is not new to you at all.

I know I am carrying coals to Newcastle, but the justification for this attempt of mine is based on the assumption that there may be a few in this learned assembly who, on account of pressure of professional duties, both private and public, have not found enough time to study the new literature on the subject. To such of them, the information I have gathered, might be of some use.

I have also another important object in view. It is this: From such of you, as are closely following the progress of research in this field, I should very much like to learn facts with which I am not yet familiar. From such

* A paper read before the Tanjore District Medical Association.

of you, as are putting these new ideas into practice in every day life, I should like to have the benefit of your valued experience.

About the importance of the subject itself, there can be no two opinions. This fell disease is carrying away from our midst so many precious lives, that we are not justified in being indifferent towards the havoc it is committing. We should put forth all our efforts and try to combat the disease with such new weapons as modern science has provided us with.

With these preliminary observations, I begin the subject of my theme.

Diabetes is to be defined, in the light of recent knowledge, as a disorder of the general metabolism of the body, characterised by an *excess of sugar in the blood and generally by the presence of sugar in the urine*. You all know that sugar is an intermediate product of carbohydrate metabolism. That is, in a healthy person, the starchy food that he takes, is first converted into sugar and this sugar is further broken up into CO_2 and H_2O in the tissues. But in a person suffering from diabetes, all the sugar that is formed from carbohydrates or starches, does not get converted into CO_2 and H_2O , but a large portion remains as sugar, gets concentrated in the blood and eventually passes in the urine. That is, in a diabetic, a large portion of sugar formed from starchy foods, is not burnt up in the body into its ultimate products. If this is so, the disease may be considered to be a disorder of carbohydrate metabolism. In fact, this idea prevailed till recently. But investigation has shown that in a diabetic, sugar is formed from protein as well and under some circumstances

sugar may be formed from even fats. As regards the protein forming the sugar, instances are common, where bad diabetics go on passing sugar in the urine, though they are put on an exclusively protein diet, entirely free from carbohydrates. In the case of fats, some sugar may be formed from the glycerine which they contain—You know that fats are compounds of glycerine and fatty acids—but it is questionable whether sugar is formed from fatty acids. But these fatty acids produce, in diabetics, substances called *Ketone bodies* namely— β -oxybutyric acid, diacetic acid and acetone. They are the intermediary products of fat metabolism. That is, as the fat metabolism, in a diabetic, is imperfect, these ketone bodies are formed from fat and *they remain as such* without being ultimately broken up into CO_2 and H_2O . In a normal person also, these ketone bodies are formed, but soon they get broken up into their simpler components, namely CO_2 and H_2O . A good deal of mischief done to a diabetic is caused by these ketone bodies concentrating in the blood. More mischief is caused from these and not from the sugar present in the blood. So, you see how fat metabolism also plays a very important part in a diabetic. From these considerations, we have to discard the old idea that diabetes is a disorder of carbohydrate metabolism only, and assume that it is a disorder or disturbance of not only carbohydrate metabolism but also of the protein and fat metabolism. In short, it is a disorder of the general metabolism of the body. Unless we grasp this idea, we will not be in a position to understand the *principles of the modern dietetic treatment of diabetes*.



Aetiology.—It has been admitted that sedentary habits associated with an excess of carbohydrate diet and mental work are the chief causes of diabetes. In Bengal, the common impression among the people is, that the District Munsiff gets the disease and the Deputy Magistrate does not. The District Munsiff sits at his table throughout the day, whereas the Deputy Magistrate leads an active life, as he goes on camping throughout the greater part of the year. It is also stated that it is a disease of the well-to-do who eat food rich in carbohydrate material. These factors may act as predisposing causes, but the essential cause is not yet known. For not only do we find the disease, in a well-to-do person with sedentary habits with or without plenty of brain work, but we find it also in a wandering beggar and in a toiling agriculturist. We have noticed it in persons whose habits are far from sedentary, but who on the other hand are very good sportsmen. It is found in the non-vegetarians as well as in vegetarians, though it is commoner in the latter. It is noticed in children, adults and old man. It is said to be more common in men than in women. The orthodox Hindu widow who does all the household work, lives on one meal a day and starves completely for 24 hours or more, once in a fortnight, or two, in strict accordance with her religious teachings, rarely suffers from the disease. There is no doubt that periodical starvation not only prevents the onset of the disease but actually checks its progress, as we shall see later on. These facts lead us to conclude that there are several factors involved in its causation, some of which act in combination in each particular case, or it may be that there is only a single exciting cause, with the other factors

acting as predisposing causes. In spite of all the research work done recently in connection with the disease, we are still in the dark with regard to its aetiology.

ABNORMAL SUBSTANCES PRESENT IN DIABETIC URINE.

(1) *Sugar*.—Presence of sugar in the urine is the most outstanding feature among the symptoms of the disease. The sugar is glucose or dextrose. We know that glucose is normally formed in the body from carbohydrates. It is stored in the form of glycogen in the liver. This glycogen gets converted into glucose whenever the tissues require it. The glucose is split up in the tissues into CO_2 and H_2O . Normal urine is said to contain 0.03 to 0.05 p.c. of sugar, but all authorities do not agree about this. MacLeod—one of the well-known physiologists—says “as the matter stands at present we must conclude that there is no unassailable evidence that dextrose (glucose) is a constituent of normal urine.” In diabetic urine, an appreciable quantity of glucose is present. The most common test used to detect glucose in the urine is what is called the Fehling’s test with which we are all familiar. Now-a-days, this solution is being given up in favour of the Benedict Solution, for the following reasons:—

(1) With Fehling’s test we cannot detect very small quantities of sugar in the urine. It is stated that with Fehling’s test smaller quantities of sugar than 0.1 p.c. cannot be detected, whereas with the Benedict’s the presence of 0.08 p.c. sugar can be easily detected. (2) Other abnormal constituents of urine such as urates, uric acid, creatinine, glycuronic acid, etc., reduce the copper in the Fehling’s solution, whereas Benedict’s solution does not get affected by these. (3) Benedict’s solution is a single solution consisting of sodii citras, sodii bicarb, and

copper sulphate and distilled water. It keeps well for an indefinite period. The principle of both these tests is well known to you. The sugar in the urine re-acts with the copper sulphate and reduces it to cuprous oxide (Cu_2O) which is insoluble in an alkaline solution and is therefore precipitated. Sometimes the re-action with the Fehling's solution or Benedict's is slightly modified, either with regard to the colour of the precipitate or the time taken to produce the same. For instance, when the urine is boiled with the re-agent, nothing happens immediately, but if the boiled urine is allowed to stand for a few minutes there is a yellowish green precipitate or a dirty greenish milky liquid. In these cases the modified re-action means that urine contains very small quantities of sugar. While speaking about the test for glucose in the urine, I should point out that the presence of *lactose* or milk sugar also gives the Fehling's test. Lactose frequently occurs in the urine of pregnant and nursing women. Lactosuria may be mistaken for glycosuria and treated as for glycosuria. Lactose may be distinguished from glucose as follows: (i) Glucose causes fermentation with yeast and lactose does not. (ii) Glucose when boiled with phenyl hydrazine, characteristic sheaflike crystals are produced within half an hour. Lactose also forms similar crystals, but not till the mixture is heated for several hours.

So much for the detection of glucose in the urine. There are other important substances also which are found, sometimes later on in the disease. These are called *ketone bodies*. They are the following:—B—oxybutyric acid, aceto-acetic (diacetic) acid and acetone. They are present in severe cases of diabetes and also in advanced

stages of ordinary cases, and are of *greater importance than glucose itself*. Their presence gives an indication that the disease is progressing rapidly and that the prognosis is bad. When these bodies are present we may take it for granted that diabetic coma is very likely to develop soon. These bodies are formed from the fat that the diabetic takes. They are the intermediary products of fat metabolism, just as glucose is the intermediary product of carbohydrate metabolism. (This has been already referred to above). When these bodies accumulate in the blood, the condition is known as *ketonæmia*. When they are passed in the urine, it is known as *ketonuria*.

TESTS FOR KETONES IN THE URINE.

1. *Test for di-acetic acid* (Gerhard's test). To some fresh urine in a test tube, add a few drops of 10 p.c. ferric chloride solution. Frequently a precipitate of ferric phosphate appears. Filter off and add more ferric chloride solution drop by drop. If a portwine or claret colour is produced, diacetic acid is present. It is unnecessary to filter, as the claret colour can be easily detected even in the presence of ferric phosphate.

Test for acetone.—Rothera's test.

Take a small quantity of urine in a test tube; saturate it with crystals of ammonium sulphate. To the mixture add a few drops of a recently prepared 10 p.c. solution of sodium nitroprusside, and as much ammonium hydrate as the amount of urine (caustic potash may also be substituted). Shake the test tube; the appearance of a *permanganate colour* indicates the presence of acetone.

Diacetic acid also gives this test; so this may be taken as a test for both diacetic acid and acetone or ketone

bodies. These tests for diacetic acid and acetone, are as important as, if not more important than, the test for sugar. Every medical man in charge of a diabetic patient, should be familiar with these tests.

Other tests.—There are other complicated tests such as the estimation of the acidity of the urine; estimation of CO_2 in alveolar air, estimation of total nitrogen, etc., which give a clue to the gravity of a case, but these tests can only be carried out in a fully equipped laboratory and will not be useful for ordinary clinical diagnostic purposes.

PATHOLOGY OF DIABETES.

Experimentally diabetes has been produced in animals by the removal of the pancreas from their bodies, *e.g.*, if the pancreas of a dog is removed, the animal develops diabetes. Again, in human beings, in some cases of death from diabetes, structural changes in the pancreas such as simple atrophy, fibrosis with atrophy, chronic interstitial pancreatitis have been found. Sometimes, a carcinoma or a calculus have been noticed. Acute lesions of the pancreas have also been observed. From these observations, it has been concluded that the *disease of the pancreas* and the *consequent suppression of its function* are the ultimate cause of diabetes. But in a certain number of cases of death from this disease, no lesions whatsoever have been found in the pancreas; the structure does not show any deviation from the normal. If the structure is normal, the function of the organ must necessarily be normal. In other words no disorder of the pancreas could be found in a number of cases. This means that the *disease of pancreas is not always the cause of the condition*.

A few words are necessary about the structure of the pancreas itself. This organ has two kinds of cells:—(1)

those that are regularly arranged into alveoli and provided with ducts; (ii) those which are irregularly grouped together in the midst of the alveoli and *not* provided with ducts. The latter are called “the islands or the islets of Langerhans.” From these two kinds of cells, two different kinds of secretions are formed, an external secretion and an internal secretion. It has been proved that the alveolar cells produce the external secretion and the islets of Langerhans produce the *internal secretion*. The external secretion is the *pancreatic (juice) fluid* which is led away by the pancreatic duct into the duodenum. We know the action of the pancreatic juice. It acts on all the three kinds of food stuffs—namely, proteins, fats and carbohydrates, and digests them. The *internal secretion* is absorbed into the blood. It is believed that this internal secretion regulates the carbohydrate metabolism, that is, it helps the sugar to be stored as glycogen in the liver and also helps tissue-cells to utilise the sugar whenever necessity arises. In pathological lesions of the pancreas, *this internal secretion is lacking* and therefore the tissues cannot utilise all the sugar in the body. The sugar consequently accumulates in the blood. When the blood is sufficiently concentrated with sugar, the latter is excreted by the kidneys. So, then, in a number of cases, diabetes is believed to be due to this deficiency of the internal secretion of the pancreas.

But I must tell you here what some well-known authorities, *e.g.*, Macleod, think about the structural changes found in the pancreas which are supposed to be the *cause of diabetes*. They think that they may *not be the cause at all* but on the other hand, *they may be the results of diabetes*. For instance, fibrosis or atrophy of “the islets

of Langerhans" may be the sequence of diabetes, as a carbuncle or a cataract follows diabetes.

The problem of the aetiology of diabetes is not therefore solved, by considering the pancreas to be responsible for the causation of the disease. As I have already suggested, there are a large number of cases in which no change in the histological structure of the gland could be noticed. In such cases the cause is to be sought elsewhere. Cammidge, one of the leading authorities on the subject, believes that there are several types of the disease:—

(i) a *pancreatic type* in which the internal secretion of the pancreas is wanting;

(ii) a *hepatic type* in which the liver does not store up sugar in the form of glycogen (as it does normally) but the sugar passes directly into the blood stream, on account of some defect of the liver;

(iii) a *glandular type*, where there is hyperactivity of the ductless glands such as the pituitary, thyroid, etc., leading to excess of concentration of sugar in the blood;

(iv) a *nervous type*, in which the impulses from the nervous system cause a discharge of stored glycogen from the tissues, which the brake-like action of the internal secretion of the pancreas is not able to control completely.

Thus it will be seen that the disease may be due to a variety of causes. This view is confirmed by the varying character of the symptoms, the different course and progress of the disease in different cases and the failure to bring on a cure with one particular kind of treatment. Cammidge thinks that, though the initial cause may be different in each case, a *deficiency of the internal secretion*

develops in each case *later on* in the course of the disease. This is very probably the reason why *insulin* has been of use in most cases of diabetes.

Other forms of glycosuria. I shall briefly refer here to several other conditions in which sugar may be present in the urine: (1) Glycosuria may result from nervous influences such as result from certain affections of the brain. The classical experiment of Claude Bernard who produced glycosuria, by puncturing the floor of the 4th ventricle, is an example of this. The cause of such glycosuria seems to be stimulation of the sympathetic nerves which go to the liver and the increased formation of glucose from the glycogen of the liver. (2) Hyper-secretion of some of the ductless glands such as pituitary, adrenals, thyroid, etc., leads to glycosuria, *e.g.*, exophthalmic goitre, acromegaly. (3) Sometimes, chloroform anaesthesia causes glycosuria. (4) Excessive use of alcohol during a dinner may sometimes cause glycosuria next morning. (5) In persons suffering from boils, carbuncles, septic wounds, etc., glycosuria is often present. (6) Sometimes mere emotional disturbance causes glycosuria. In 1914, Folin, Denis and Smethie tested the urine of a number of students after they sat for an examination and found sugar in 18 p.c. of them. Probably glycosuria found during the medical examination of a candidate for physical fitness may be only a mere indication of his mental excitement and nervousness. (7) In some cases called *renal glycosuria*, the carbohydrate metabolism is all right and there is no excess of concentration of sugar in the blood, as in real diabetes, but even then, the person passes sugar in the urine. In these cases the kidneys are supposed to let through the sugar more easily than in a

healthy person. To put it in other words the kidneys are supposed to be leaky. Hence this condition is known as "*leaky kidneys*." This is expressed by saying that the *kidney threshold* is lower than normal. I shall refer to this subject again in detail.

In all the above conditions, sugar appears in the urine and the list is a fairly long one. These are cases of glycosurias only and not of diabetes, and most of them are harmless. These harmless glycosurias must be distinguished from real cases of diabetes. The mere presence of sugar in the urine is not at all enough to warrant the conclusion that the person is suffering from actual diabetes. There are possibly a large number of persons who are passing sugar in the urine for the past several years and are the none the worse for this. They keep up excellent health and vigor. The fact is that, very probably, they are not suffering from diabetes but only from a harmless glycosuria, perhaps *renal glycosuria*. How can we diagnose these harmless cases of glycosuria from cases of real diabetes? Modern science and research have evolved a method of doing this and I shall very soon describe this method. This consists in estimating the amount of sugar present not in the urine but the *amount of sugar present in the blood of the person*. Before describing how this can be done, I shall make a few *observations on blood sugar*.

BLOOD SUGAR.

The blood of even a healthy individual contains a certain amount of sugar *as glucose* due to the carbohydrate food that he takes. The percentage of sugar in normal blood varies within certain limits. *When the individual is starving, his blood contains 0.09 to 0.1 p.c.*

of sugar. We may take the second figure, namely, 0.1 p.c. as the *average* for healthy persons. In Bengal McCay has found the average to be 0.13. Capt. Bhatia, I.M.S., Professor of Physiology, Grant Medical College, Bombay, has found the average for 18 non-vegetarians to be 0.116 p.c., and the same for 20 vegetarians to be 0.149 p.c. After the individual takes a starchy food, the p.c. of sugar in the blood goes up to about 0.17 within half to one hour and it suddenly drops down to the normal figure of 0.1 p.c. within 1½ to 2 hours after taking the meal. The same thing happens if the person takes a large quantity of sugar, instead of the carbohydrate meal. But when a diabetic takes a starchy meal, the p.c. of his blood sugar goes up to a much higher figure, say 0.2, 0.3, 0.4 p.c., etc. Not only the p.c. of blood sugar reaches a higher figure than in normal individuals, but something else also happens. The blood sugar takes a longer time to drop down to the normal level. That is, it may take 4 to 5 or 6 hours instead of 2 hours to drop to the normal level of 0.1 p.c. or it may continue to be slightly above the normal level, till he takes his next meal, when it will rise higher still.

In a normal person with healthy kidneys, no sugar passes in the urine even though he takes large quantities of carbohydrate food or sugar. It is said that 100 to 120 grammes of sugar can be taken by a normal person at a time without sugar appearing in the urine. McCay in Bengal has given 300 grammes of sugar at times to students, without any trace of sugar appearing in the urine. But in a diabetic, even small quantities of these foods will give rise to glycosuria. The reason is that in the case of the normal individual, his carbohydrate

mechanism is so perfect that it prevents such accumulation of sugar in the blood as will give rise to the passage of sugar in the urine. In the case of the diabetic, his carbohydrate mechanism or the sugar controlling mechanism is imperfect, so that a higher concentration of sugar takes place in his blood which causes the passage of the same in the urine. Generally speaking, in a healthy individual, no excretion of sugar takes place in the urine, unless his blood sugar reaches 0.18 p.c. or 0.2 p.c.; and with any amount of sugar or carbohydrate ingested, the blood sugar never exceeds this limit. The p.c. of blood sugar in a healthy person, at which sugar begins to appear in the urine is 0.18 or 0.2 and is called the *kidney threshold*. To explain clearly what this *kidney threshold* means, I quote a passage from Cammidge. "The kidneys act towards the sugar in the blood much in the same way as a dam or sluice to the water in a reservoir, only permitting an overflow, when more than a given height has been attained and this height in healthy persons appears to be a fairly constant one." In a diabetic even small quantities of carbohydrates may cause the blood sugar to go up far above the threshold value. Consequently sugar appears in the urine.

SUGAR TOLERANCE TEST.

I stated above that modern research has devised methods of accurately diagnosing diabetes from other forms of harmless glycosurias. I describe now the test employed for this purpose. This is known as *sugar tolerance test*. The test is carried out on an empty stomach or at least 3 hours after taking a meal. First the patient passes urine and his blood is taken for estimation of blood sugar. Then 50 grammes of commercial

glucose dissolved in 150 c.c. of water is given by the mouth. Samples of blood are taken every half an hour for 2 hours, i.e., they are taken 4 times during 2 hours. Urine is passed at the end of each hour. The p.c. of blood sugar obtained at each examination is marked in a chart and a curve thus obtained. In normal individuals, the blood sugar reaches a level of about 0.18 p.c. within $\frac{1}{2}$ to 1 hour and drops down to 0.1 or the normal, within $1\frac{1}{2}$ to 2 hours. In older persons, it may take about 3 hours but not longer. In a diabetic, the blood sugar may go up to 0.2, 0.4 p.c., etc., and takes several hours to reach the normal.

As a working rule we may assume that if the blood sugar fails to return to its original level or nearly so, *within 2 hours* after the administration of a single dose of 50 grammes of glucose, we may conclude that the person concerned is a diabetic with a faulty sugar storing metabolism.

RENAL GLYCOSURIA.

I stated above that the kidney threshold for a healthy person is 0.18 or 0.2. This threshold value in the case of Indians, especially vegetarians, appears to be low. Capt. Bhatia of Bombay has observed it to be 0.15 for Indians. That is, a healthy person begins to pass sugar in the urine, as soon as his blood sugar concentration goes up above 0.18 or 0.2 p.c. which generally occurs after a meal or ingestion of sugar. But some persons pass sugar in the urine even when their blood sugar concentration is lower than this figure, e.g., 0.14, 0.15, etc. That is to say, *their kidney threshold is low*. Or in other words, their kidneys are more permeable to sugar, than the kidneys of normal persons. They are said to have *leaky kidneys*

and the condition is called *Renal glycosuria*. Such persons evidently go on passing sugar in the urine after every meal. This is not diabetes at all and is a harmless glycosuria. How can we diagnose the condition? The mere test for sugar in the urine is absolutely useless in this case. It requires a blood-sugar estimation. If we make out a *blood sugar curve*, we notice that this curve is quite different from that of a diabetic. The curve does not reach a point higher than 0.16 or 0.14 and it drops to normal within 90 minutes or so. The condition can be diagnosed even without blood sugar examination, in the following way. Examine the urine after a carbohydrate meal or after giving 50 grammes of sugar by mouth. If you do not find any sugar in the urine, 2 hours after taking the meal or the sugar, and if he is quite healthy without any signs of diabetes, then such a person is only suffering from renal glycosuria and not diabetes. Some authorities think this is only a rare condition and can be neglected for practical purposes. But Cammidge and MacLean hold that the condition is very common; at least more common than hitherto recognised. In 48 cases which MacLean examined for official and general medical purposes 23 cases were renal glycosuria. As regards the causation of this condition, Cammidge believes that it is due to deficiency of calcium salts in the blood and therefore of Parathyroid secretion. Normal blood contains 5.3 to 6.8 p.c. of calcium salt; but in renal glycosuria only 2 p.c. is present. Cammidge has successfully treated these cases with the administration of calcium salts and parathyroid gland.

Here then is the great importance of blood sugar examination in all cases of glycosuria. When we examine

persons for physical fitness or for purposes of treatment and when we find sugar in the urine, we at once conclude that such persons are diabetics. We reject them for purposes of service or insurance. We put them probably on a rigorous dietetic treatment or sometimes we may even advise them injections of insulin. If some of these cases are only harmless glycosurias such as renal glycosuria, we shall be doing them immense harm. Perhaps we shall be responsible for causing much mental worry and a great deal of physical discomfort to a number of persons who are not real diabetics at all. I think it is high time that we do things more carefully. We must do sugar tolerance tests and carry out blood sugar estimations. Only then, can we diagnose real cases of diabetes from harmless glycosurias. I quote below, the case of a man who was rejected by a Life Insurance Co., on the plea that his urine contained sugar, though he had only a harmless renal glycosuria.

"A healthy man of wiry physique and clean family and personal history was recently totally rejected by a well-known Insurance Co., on account of alleged glycosuria. He was subsequently sent to me by his medical advisor, when I estimated the sugar content of his blood and urine, after a meal containing a very large quantity of carbohydrate and sugar. The urine contained 0.08 p.c. and the blood 0.101 p.c. respectively. This man was therefore not a diabetic, but his renal threshold was lower than normal. These facts were given to the company, who simply retorted that they intended to abide by their decision. The injustice of this procedure is evident, especially as the patient's prospects in regard to assurance in any other company are prejudiced, by his having to

disclose his previous rejection. What remedy has this man against the autocratic ignorance? "I should like to know." I am yours faithfully. A Knyvett Gordon, M.B.

Lancet Dec. 19, 1925. Correspondence : "*Glycosuria and Life-Insurance.*"

ALIMENTARY GLYCOSURIA.

A few words should be said about the so-called alimentary glycosuria. In this condition, it is supposed that the ingestion of a very large amount of sugar causes a portion of the latter to be absorbed into the blood directly from the intestines (i.e., without passing through the liver) and thus lead to hyperglycaemia and glycosuria. This is a wrong idea. It is also wrongly presumed that in this condition there is no defect in the carbohydrate metabolism. With a normal carbohydrate metabolism, a healthy person is capable of dealing with very large quantities of sugar without having hyperglycaemia and glycosuria. Hence there can be no such thing as 'alimentary glycosuria.' MacLean suggests that "this term might be dropped with advantage."

METHODS OF EXAMINATION OF BLOOD SUGAR.

For these I want you to refer to the books. The most common method employed in England is MacLean's method, which consists of precipitating the proteins in the blood by means of acid sodium sulphate and dialysed iron and then estimating the sugar in the filtrate by means of a copper solution. MacLean has recently devised a handy apparatus for this purpose which can be used by any practitioner. I think its price is Rs. 80 or so.

(To be continued.)

COMPLEMENT FIXATION IN TUBERCULOSIS 19

COMPLEMENT FIXATION IN TUBERCULOSIS.

(BY RAO BAHADUR DR. M. KESAVA PAI, M.D.,
Director, Tuberculosis Institute and Superintendent,
Tuberculosis Hospital, Madras,
and

K. VENKAT RAU, M.B. B.S., Research Laboratory, Madras.)

Bordet and Gengou⁶ (1903) first demonstrated the presence of antibodies for the bacillus of tuberculosis in the sera of tuberculous animals.

Wassermann and Brucke³² (1906) working with the sera of patients treated with tuberculin showed that tuberculin fixed complement in the presence of these sera.

Besredka⁴ and Manoukhine⁵ (1914) obtained complement fixation reactions in a large number of tuberculous sera of patients not treated with tuberculin.

Kuss, Inman²⁰ and others have shown that tubercle antigen does not give an absolutely specific result in complement fixation reactions, but that a number of non-tubercular sera, especially those of syphilitics, give a complement fixation reaction with the antigen.

Nishida and Petroff²⁴ have demonstrated complement fixation with the blood of sheep immunised with living tubercle bacilli. A very large amount of work has been done during recent years on the complement fixation reaction in tuberculosis and the results obtained by various workers, though differing somewhat as regards the reliability of the test, go to prove that the test has a definite though not absolute clinical value when considered along with other clinical data.

Caulfield and Peatty¹¹ using tubercle bacillary emulsion and old tuberculin as antigens obtained 56% positive reactions in tuberculous cases. They concluded that the test was therefore not reliable.

Dudgeon, Meek and Weir^{14, 15}, using different kinds of antigens found that a killed suspension of tubercle bacilli gave 84% positive results in pulmonary tuberculosis and 57% in surgical cases. The same workers employed an alcoholic extract of tubercle bacilli as antigen and obtained 89.3% positive results in tuberculosis.

Elizabeth Fraser¹⁸ working with a living emulsion of bacilli grown on glycerine potato medium obtained only 68% positive reactions in tuberculous cases.

McIntosh, Fildes and Radcliffe²¹ used emulsions of the tubercle bacillus in saline and obtained 76.7% positive results in pulmonary tuberculosis and 80.7% in surgical cases. These percentages were based on comparatively small figures.

Radcliffe,²⁷ from an analysis of 568 cases of pulmonary tuberculosis in which he used fresh suspension of bacilli, obtained 90% positive reactions.

Calmette and Messo¹⁸ made a 1% peptone extract of tubercle bacilli and using the concentrated filtrate as antigen obtained 92% positive results in 134 cases of tuberculosis.

Wang and Crocket³¹ using a glycerine veal culture of tubercle bacilli and extracting it with ether and chloroform obtained 84% positive results in patients treated with tuberculin and 87% in cases not so treated.

Watkins and Boynton³³ basing their results on an exhaustive investigation of 6,500 cases, obtained 78% positive results in clinically tuberculous cases with bacilli; 64.6% in clinically positive but bacillus negative cases; 37% on suspected cases and 4.2% in clinically non-tuberculous cases.

Fidlar¹⁷ using tubercle bacillus suspension in saline obtained 74 to 77% positive results in clinically active pulmonary tuberculosis.

Alcock, Douglas and Luey,¹ from a synopsis of 6,522 cases in which Besredka's antigen was used, obtained 90.6% positive reactions in bacillus positive cases, 47% in the clinically tuberculous in whom bacilli were present at some time previously, and 36.5% in clinically tuberculous cases in whom bacilli could never be demonstrated.

Nimmo Smith and Hewat²⁸ using Besredka's antigen got 89.8% positives amongst bacillus positive cases, 55.8% amongst clinically positive but bacillus negative cases and 14.2% in arrested cases.

Craig and Miller obtained 98% positives amongst tuberculous and 4% amongst non-tuberculous cases.

It will be seen from the above resume of the literature on the subject that the results have varied fairly widely in various hands. The explanation is not far to seek. The varying factor has been the antigen; the complement, tuberculous serum and hæmolytic system having been more or less identical. Thus Wassermann used old Tuberculin; Nesfield and Emery emulsions of dried tubercle bacilli; Dudgeon killed but fresh emulsions of bacilli; Inman²⁰ emulsion of living bacilli; Wang and

Crockett³¹ a glycerine veal culture defatted with ether and chloroform; Craig and Dudgeon an alcoholic extract of tubercle bacilli; Casper autolysates of tubercle bacillus emulsions; Besredka⁴ a specially prepared emulsion from growths on his egg medium; Renaux²⁸ and Bronfennbrenner the same antigen previously extracted with ether or chloroform or both to remove the lipoids and prevent 'Cross-Fixation' with syphilitic sera; Petroff a glycerine extract of tubercle bacilli and so on; each claiming good results and advantages for his own antigen. Polyvalency of antigen is stressed by Miller, whilst Cooper and Sweany,¹² consider most antigens equally good. Aronson and Lewis³ with an antitubercular goat serum obtained steady results with all antigens. Variations on the results may also be partly due to differences in the technique, specially the temperature, method and duration of incubation during the different stages of the reaction. It has been asserted by some that the incubation of the tubes in the first stage of the reaction should be much longer than in the Wassermann reaction and that the hamolytic results should be read immediately after the completion of the second stage and not after allowing the tubes to settle overnight. Others again have used smaller doses of complement to add to the delicacy of the test. These details severally or jointly are no doubt very important in the case of tubercle complement fixations.

Serum from venous blood drawn by puncture has been used by the vast majority of workers. Sillers, Ramsbotham and others have obtained good results with cerebro-spinal fluid.

COMPLEMENT FIXATION IN TUBERCULOSIS 23

In view of the above points in the conduct of the test, we find it necessary that the technique employed in our work should be described in some detail. Three parallel series were done with every sample of blood, *viz.*, complement fixation with Besredka's antigen, the same with a living emulsion of tubercle bacilli grown on Petroff's egg medium, and a control series of Wassermann reactions with the same sera. The reactions in every case were carried out in small tubules of about 3 millimetre calibre volumes being measured out with capillary pipettes so that 5 c.c. of blood drawn from each case gave quite sufficient serum for all the three parallel series together. The serum was allowed to be in contact with the clot for 24 hours as a rule over ice and this seems in the opinion of Punch and others, to draw the maximum amount of immune body from the leucocytes in the clot.

Decomplementing was done by heating for $\frac{1}{2}$ hour at 55° C. The pooled serum of four guinea pigs left in contact with the clot for 4 to 5 hours was used. Titration of this complement was done on each occasion with sheep cells sensitised with 10 M.H.D. of anti-sheepamboceptor from the rabbit. For the Wassermann series 3, 5, and 8 M.H.D. of complement were respectively used for indicating weak, medium and strong reactions, whilst $1\frac{1}{2}$, 2 and $2\frac{1}{2}$ M.H.D. of complement were used in the tubercle complement fixation series. Incubation of the serum + complement + antigen system was done in the water bath at 37° C. for half an hour when the sensitised sheep cells were added. The mixture was agitated every few minutes by a capillary glass rod placed in each tube and the results were read at the end of half an hour of the second incubation. Complete

absence of hæmolysis with a clear, colourless supernatant liquid was indicated by + + +, slight hæmolysis with a heavy deposit of red cells by + +, a fair amount of hæmolysis with slight deposit of red cells by + and complete hæmolysis by—. Every test was controlled by positive and negative sera from a previous occasion. The following is a scheme of the reaction.

—	Wassermann test				T. C. F. R. with Besredka antigen				T. C. F. R. with T. B. E. antigen			
	1	2	3	4	1	2	3	4	1	2	3	4
Tube ..	1	2	3	4	1	2	3	4	1	2	3	4
Antigen ..	0	1	1	1	0	1	1	1	0	1	1	1
Complement ..	3	3	5	8	1½	1½	2	2½	1½	1½	2	2½
Saline ..	1	0	0	0	2	1	½	0	2	1	½	0
Patient's serum ..	1	1	1	1	1	1	1	1	1	1	1	1

Incubate for ½ hour

Sensitised sheep R. B. C.'s ..	1	1	1	1	1	1	1	1	1	1	1	1
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Incubate for ½ hour.

The reactions were read out immediately at the end of the experiment and again after standing the tubes overnight. In the latter case it was sometimes found that a weak positive reaction of the previous evening was apt to show a negative next morning as hæmolysis possibly non-specific in character is apt to take place by longer keeping especially in a tropical climate. Our results therefore represent the observations at the termination of the second incubation on the evening of the test.

The uniformity of the bacillary suspensions was assured by an opacity test on the same lines as Brown's

standardisation of bacillary vaccines. Controls were done with known sera which were kept for subsequent examination. It was found that the opacity standardisation of the antigen was quite reliable for all experimental purposes.

The Specificity of the Reaction.

That a large percentage of syphilitic sera give a complement fixation reaction with tubercle antigen is a well recognised fact. It has therefore been a rule that a control series should always be done for the Wassermann reaction when conducting a tubercle complement fixation test and no serum should be declared positive unless it gives a negative Wassermann reaction. Wang and Crockett³¹ found that, whilst non-tuberculous and non-syphilitic sera gave a negative complement fixation for tuberculosis, syphilitic non-tuberculous sera gave 41% positive T. C. F. R. (tubercle complement fixation reaction). Conversely tuberculous, non-syphilitic sera have been known to give a Wassermann positive reaction, Fidler¹⁷ and Dulaney¹⁶ found 7 to 8% of tuberculous non-syphilitic cases give a Wassermann positive reaction, and Von Adelung³⁰ 8.7%. The latter however explains this by saying that this figure represents the percentage of syphilitics in a 'normal' population. This kind of 'cross-fixation' in tuberculosis and syphilis has not been correctly explained though the lipoids in the antigen and insufficient heating of serum seem to have something to do with it, as proved by defatting of the tubercle antigen and prolonged heating of the serum in decomplementing it, when a lower percentage of 'cross-fixation' is obtained. Again anti-syphilitic treatment has often been found to render a positive T.C.F.R. into a negative one.

The percentage of positive T.C.F.R. in non-tuberculous cases has varied in the hands of different workers. Inman²⁰ obtained 24%, Nimmo Smith²³ 26.4%, Debaines and Jupille¹³ 24% in Wassermann positive sera, 17.3% in non-syphilitic sera and 3.2% in perfectly normal individuals; Miller 44% amongst leutics; Craig 5.4%, and Fidler¹⁷ 28.3%; Besredka with his egg medium antigen which contains lipoids found 79 positives amongst 750 bloods sent for the Wassermann test. Cooke examined the blood of 75 cases of congenital syphilis and found the T.C.F.R. positive in all of them.

Tubercle complement fixation has also been noted by different observers in a variety of diseases including diphtheria, epidemic encephalitis and leprosy (84.2%).

Significance of the reaction.

There is no universal agreement regarding the interpellation of results obtained in tubercle complement fixation reactions and in this particular the test is of definitely less value than the Wassermann test in syphilis. Punch^{25, 26}, and Inman hold that a positive reaction is a reliable test for active and recently active tuberculosis and a negative reaction excludes the diagnosis of tuberculosis. In Punch's first series published in 1920 he refers to 50 cases of proved tuberculosis in which 49 gave a positive T.C.F.R. whilst the only negative case was found to be one of bronchiectasis. He also found clinically positive but bacillus negative cases giving a 100% positive reactions and that positive reactions persisted even after arrest. He found moreover that cases giving positive Von Pirquet's test but clinically non-tuberculous gave a negative reaction. No other worker has been so lucky in obtaining such consistent results.

Heckman¹⁹ found that T.C.F.R. is negative in many cases of tuberculosis, especially at the onset of pleurisy, peritonitis, meningitis and acute miliary infection. He observed that a persistently strong positive reaction indicates a grave prognosis and that a positive becoming negative is usually a bad omen. According to Watkins and Boynton¹³ this last phenomenon is a sure indication of the exhaustion of the immunising mechanism.

Besredka⁴ tested the blood of 12 infants suffering from tuberculous meningitis and found the reaction positive in every one of them.

TABLE I.

Result	Clinically and bacteriologically positive cases	Clinically positive but bacteriologically negative cases	Total clinically tuberculous cases	Clinically non-tuberculous cases
T.C.F.R. with Besredka antigen and bacillary emulsion positive ..	94 (62.2%)	43 (87.7%)	137 (68.5)	19 (28%)
T.C.F.R. with Besredka antigen positive but bacillary emulsion negative ..	14 (9.30%)	3 (6.1%)	17 (8.5%)	5 (7.3%)
T.C.F.R. with Besredka antigen negative but with bacillary emulsion positive ..	14 (9.3%)	1 (2.1%)	17 (7.5%)	3 (4.4%)
T.C.F.R. negative with both Besredka and bacillary emulsion antigens	29 (19.2%)	2 (4.1%)	31 (15.5%)	11 (60.3%)

A different view is held by Brown⁷ and his co-workers who found relapses were more often found amongst patients with positive T.C.F.R. than with a negative T.C.F.R. Caulfield⁹ also asserts that when a patient gives a strong T.C.F.R. time after time he is almost certainly going downhill, though it is a fact that in the last stages

the reaction becomes negative. It is however agreed on all hands that a strong T.C.F.R. is diagnostic of tuberculosis, though the opposite does not hold good, *i.e.*, a negative T.C.F.R. does not exclude the possibility of active tuberculosis. On the other hand it has been found that out of the positive T.C.F.R.'s in the apparently non-tuberculous several persons have developed tuberculosis soon after the test and a good number of others were contacts.

Other observers like Wang and Crockett³¹ consider that there is no relationship between the strength of the reaction and the stage of the disease. Andrus² thinks that prognosis can be based on the degree of reaction time after time and that though the presence of the reaction is more an indication of infection than of active disease a strong persistent reaction means definitely clinical tuberculosis.

TABLE II.

Results	Tuberculous cases Bacillus + ve.	Tuberculous cases Bacillus - ve.	Total Tuberculous cases	Clinically non-tuberculous cases
T.C.F.R. +ve c. Besredka and bacillary antigen ..	62.2%	87.7%	68.5%	28%
T.C.F.R. +ve c. Besredka antigen ..	71.5%	99.8%	77%	35.3%
T.C.F.R. +ve c. T.B.E. antigen ..	71.5%	89.8%	76%	32.4%
T.C.F.R. +ve c. either or both antigens ..	80.8%	95.6%	84.5%	39.7%
T.C.F.R. negative c. both antigens ..	19.2%	4.1%	15.5%	60.3%

It will be seen from Table II that whilst 80.8% only of the bacillus positive cases give a positive T.C.F.R.

with either antigen, 95.9% give a positive reaction in the bacillus negative cases. This apparent discrepancy is accounted for by the fact that most of the bacillus positive cases in the series were advanced III stage cases wherein a negative reaction was elicited with either or both antigens. The percentage of 84.5 positives by the use of both the antigens as against 77% and 76% respectively with either of the antigens is of special interest indicating that more reliable results can be obtained by a duplicate series than by the use of a single antigen. It is also seen that the delicacy of the test with the Besredka and bacillary emulsion antigens is about the same, being 71.5% in both in the bacillus positive cases, 93.8% and 89.8% respectively in the bacillus negative cases and 77% and 76% respectively in the total clinically tuberculous cases. The percentage of cases undetected by the use of the test, *viz.*, 15.5% compares favourably with the results obtained by other workers.

It is seen from the table, that the percentage of positive results in clinically non-tuberculous cases is comparatively high being 39.7 when either or both antigens are used and 35.3% and 32.4% respectively with Besredka antigen and bacillary emulsions. This apparently high error of experimental observation is due to the high percentage of Wassermann positive sera in the series. Thus 13 out of the 19 non-tuberculous cases in which both antigens gave a positive T.C.F.R. gave a Wassermann positive reaction; of the 5 cases in which the Besredka antigen alone gave a positive T.C.F.R. 3 were Wassermann positive and of the 3 cases in which the bacillary antigen alone gave a positive T.C.F.R. 1 was Wassermann. Leaving the Wassermann positive

cases therefore out of consideration, for reasons already stated, the real error works out at 19.6% as against the apparent error of 39.7%. The test can therefore, be considered to be of definite diagnostic value when carried out in two parallel series, some of the cases missed in one series being detected in the other, though the percentage obtained in either series is approximately the same. Thus 15.5% only are negative in the clinically tuberculous and 19.6% positive in the clinically non-tuberculous Wassermann negative cases. These figures, we consider, are of definite clinical value especially considering the fact that the incidence of tuberculous infection in the community in which this research was conducted is, as one of us has shown in another paper, very high.

*Incidence of Syphilis amongst the persons tested for
T.C.F.R.*

Excluding the borderland cases wherein the faint reaction by the Wassermann test ought to be excluded, 23.5% were found to give a positive Wassermann reaction. This is undoubtedly a higher percentage than that obtained by other observers and we do not propose to explain it.

The prognostic value of the T.C.F.R.

We have not been able to confirm the views held by some that the test has any prognostic value. We have not been able to correlate the strength of the reaction with the stage or activity of the disease or the toxæmia accompanying the condition. We have had a number of negative reactions in very advanced and moribund cases which we first imagined could be explained, in parallel with the tuberculin reaction, by a deficiency of the immune bodies in the late stages of the disease. But

not only have we seen that the degree of reaction has no relation to the condition either anatomical or toxic, but we have seen it vary from time to time in the same case regardless of whether the case is progressing favourably or otherwise. Consecutive tests conducted during the progress of a number of cases favourable or unfavourable in the hospital under our direct observation have failed to shew any relationship between the degree of reaction and the progress of the case, so that we consider that the reaction is of no value in prognosis.

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A FEW OBSERVATIONS ON PUERPERAL FEVER.

(By DR. M. B. PRABHU, M.B., B.S.)

Puerperal fever is one of the most dreaded complication after child birth. The importance of this subject lies in the fact that more women die from this than from any other single accident or disease during pregnancy, child birth or puerperium. Some observers go to the extent of saying that the death from this condition alone is nearly equal if not greater than the mortality from placenta previa, post partum hæmorrhage, rupture of the uterus, Eclampsia in short, all other conditions put together. In England it has been observed that the mortality from puerperal fever from 1911 to 1922 is nearly 30 per cent. of the total maternal mortality. In the city of Madras puerperal fevers alone accounted for 43 per cent. of the mortality connected with child birth during the last 5 years. The total number of deaths from child birth

being 1,395 and deaths from fevers after child birth being 663 (nearly half).

Besides this we have to consider a still larger number of women who recover from this fever but who are more or less invalidated for life by the train of symptoms following incomplete resolution of septic infection of the uterus and its appendages. Many a woman will recall her pelvic pain, discomfort, and menstrual irregularities, to her fever after confinement.

Ætiology.—A short resume here of the different views held at different times on this subject may not be without interest or benefit. The earliest account of puerperal fever is from Hippocrates who lived in the fourth century before Christ. He attributed it to suppression of loctial discharge. Then for a long time there was no advance on this subject. In the 16th century it was supposed to be due to inflammation of the womb as a result of entry of cold air inside. In the 17th century a very ingenuous theory came into vogue and flourished for a time. It was that of milk metastasis. It was believed that milk which was normally circulating in the body was attracted towards the uterus during pregnancy and towards the breasts during lactation and that when it found its way into the peritoneal cavity it gave rise to puerperal fever. In favour of this it was observed that a milky white fluid was found in the peritoneal cavity of women who died after this fever. This theory though it attracted some notice for a time was exploded when it was found that the milky fluid was present in the peritoneal cavity of non-pregnant women dying after certain conditions and that it was found in the peritoneal cavity of men as well. During the 18th century this fever was definitely associated

with putridity of uterine contents but it was during the last century that most of our knowledge on this subject was worked out. Dr. Gordon in Scotland and Holmes in U.S.A. established beyond doubt the contagious nature of this disease; but it was Siennmeelyweiss in Vienna who found out how the contagion spread from one person to another. He noticed that the incidence of fever in women attended to by medical students was as high as 11·4 per cent. whereas it was in case of women attended by midwives 2·7 per cent. In those days the students attended the Maternity ward straight from the dissection rooms. He thought that the infection was due to some material introduced from without by students and made them wash their hands with chlorine water and later with chloride of lime and found the incidence of sepsis fell down from 11·4 per cent. to 3 per cent. He inoculated animals with the puerperal discharges and observed lesions similar to those found in women dying from puerperal fever. Then he formulated that the essential cause was introduction from outside of decomposing organic matter and it was in the latter half of the last century that the actual organisms were discovered.

The chances of a woman to become septic are very great. Organisms are normally present at the vulva, staphylococci of various kinds are almost constantly present and bacillus coli in 20 to 30 per cent. and streptococci in 30 to 55 per cent. Staphylococci, streptococci and bacillus coli are also present in the lower part of the vagina and less frequently in the upper part. The vagina during 24 hours after delivery contains relatively few organisms. This is apparently the result of cleansing process which occurs during labour by the passage of the child's

body with placenta and membranes. It is possible also that the bactericidal power of fresh blood may exert some influence. But after second day large number of organisms are present. The local discharge alkaline in reaction containing as it does blood, epithelial cells and granular debris forms a very favourable ground for bacterial growth. The woman especially during the first 48 hours of puerperium has to pass urine in the recumbent posture and invariably some of it will contaminate the vaginal orifice. In cases where the urine is septic as in cases of pyelitis, which is not an uncommon complication of pregnancy, the genital canal will invariably be infected. After each defecation if the parts are not properly cleaned the organisms may get directly into the vulval orifice or the organisms may migrate from the rectum where the R. V. Septum has been severely bruised. In addition to all this in our country where the barber midwife is the village obstetrician one to whom asepsis is as foreign as aviation or astronomy the liability for infection is very great. In spite of all this we find cases of sepsis are not so numerous as we should expect on account of the aforesaid reasons. It is because there are the natural powers of defence that have to be taken into consideration. First and foremost there is the natural resistance of the individual. This is more marked amongst those classes who spend most of their lives in the open. Thus the poorer and lower classes have greater stamina than the richer classes. We have a wandering class of beggars who earn their livelihood by selling beads, begging or thieving. Their women sometimes get themselves confined behind a bush or a tree, pick up the baby and resume their march with probably as little concern as if they have completed defecation. Then there

is the protection afforded by the lining membrane of the genital tract. The perineum is covered by the skin, the mucous membranes of the vagina and vaginal portion of the cervix are covered by several layers of squamous Epithelium and though the cervical canal easily admits a finger after delivery it is not patent but closed. Moreover the direction of the lochial flow is from the uterus towards the outlet, from the aseptic region to the septic and so long as this lining membrane is intact the septic organisms even if they get in, do not find a suitable place to grow and multiply. As regards the uterus I believe it has sufficient resistance against the pathogenic organisms if it has not been already damaged by prolonged labour or internal manipulations during extraction of child in abnormal delivery. In cases where the uterine wall has not been subjected to much strain it is not uncommon to find pus in uterus and sometimes offensive too, though the patient's condition is good and pulse and temperature normal. If the uterine wall had no such resistance there would have been severe signs of infection in these cases.

Bacteriology.—The organisms in puerperal fever are mainly streptococci, staphylococci, *B. Coli*, Pneumococci, Gonococci more rarely Diptheroid, Tetanus and Typhoid. Streptococci are the most important of these and last year we carried out some investigations about their incidence and variety in puerperal fever. They are generally classified as Hemolytic and Non-Hemolytic depending upon their power of Hemolysing human blood and the generally accepted view is that the Hemolytic variety is associated with the more virulent type of fevers. Without going into the details of our investigation our observations briefly put have been as follows. Streptococci

have been present in 61 per cent. of our cases. Among these cases the Hemolytic variety were found in 85 per cent. Among the cases where the Hemolytic variety was found about 65 per cent. were mild cases. Our conclusions have been—

1. That Streptococci are present in the majority of our cases particularly of the more severe types.
2. The Hemolytic variety are the predominating type in our cases here and that they are present in mild as well as in severe cases. In severe cases the Hemolytic streptococci being the Anginosus, the Subacidus and the Pyogenus.
3. The non-hemolytic are found in comparatively few cases and where they were present they gave rise to severe infection in 5 cases and mild infections in 6 cases. We had so few cases of non-hemolytic infections that it is not correct to draw any conclusions about them.

Staphylococci.—These are often present with Streptococci, when alone they usually give rise to mild form of sepsis.

B. Coli. This is not very common. When present I believe it gives rise to a Subacute type of fever which does not come down so readily as in other types.

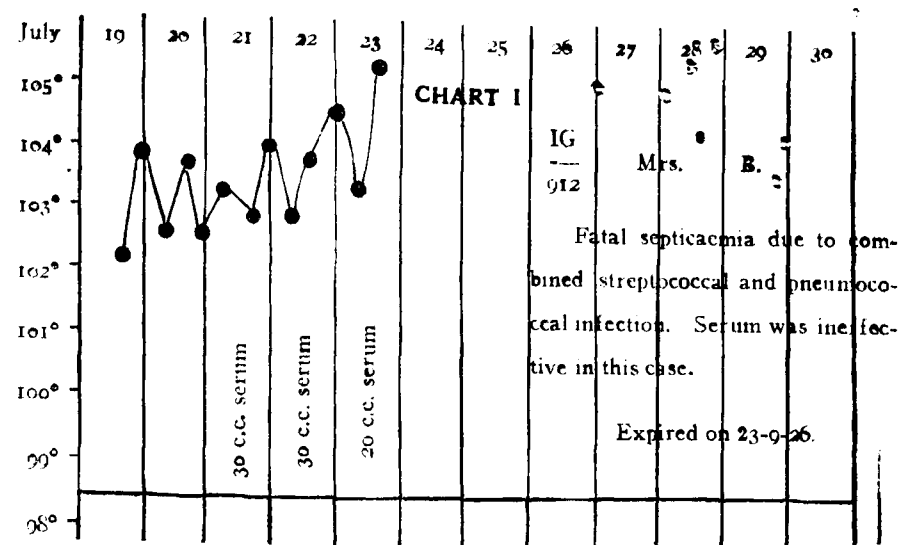
Pneumococci—are relatively uncommon. In the two cases where they were present in my series from June last they were associated with streptococci and one ended fatally and another was taken home in a low condition.

Gonococci. It is hard to isolate Gonococci in culture.

They are easily overgrown by other organisms! probably they give rise to the subacute type of fever.

Typhoid. Typhoid organisms may be isolated from uterine discharge where blood culture is positive.

Very often two or more varieties are associated together and give rise to a severe infection. See Chart I.



I would like here to touch upon the interesting question how far a case of puerperal sepsis is infective. Before the discovery of the true cause of septic fever people thought it spread like wild fire. Outbreaks of fever would arise in Maternity wards and in a mysterious way spread from patient to patient. Hence the septic cases were immediately isolated and nurses and doctors who attended on them were looked upon as if they had attended a case of small-pox. Prejudices die hard and even to-day the fears and doubts survive. As this fever can be spread only by the introduction of infective materials from outside into the genital tract there is no danger of sepsis spreading from patient to patient as long as no septic material passes from one to another. A case of puerperal fever is as infective as a case of Enteric or Dysentery. If you

take proper precautions, the chances of infection are almost nil. This is important because the obstetrician may be faced with the question whether he should undertake a maternity case if at the same time he is attending on another septic confinement case. I believe and I say it after considering well on the subject that if you take proper precautions you may undertake the case. The chances of infection are through your hands and if you properly disinfect them, then, there is no danger. I have had occasion to attend on normal labours after attending the septic cases and I have noticed no untoward results. Removal of placenta, Forceps delivery, internal podalic version, incomplete abortion completed, all these cases were followed up by me after the confinement till they left the Hospital and there was no septic trouble. If gloves are worn on all occasions the risk of infecting is practically nil.

Pathology.—Lesions may be divided into three groups.

1. Primary, 2. Local Secondary, 3. Remote Secondary.

The primary lesion may be at the placental site, at the cervix, vagina or perineum. Local secondary lesion is a result of infections spreading by contiguity. This may be in nature of Thrombo phlebitis or lymphangitis. The inflammation of the veins and lymphatics give rise to pelvic cellulitis or parametritis where the cellular tissues become oedematous infiltrated and swollen.

Peritonitis.—This may be *via* lymphatics or fallopian tubes. In mild cases it is localised. In severe cases it is diffuse there being practically no protective reaction to combat the infection. There may be inflammation of the tube giving rise to pyosalpinx. Ovary may suppurate and form abscess.

Remote lesions arise from Metastasis. These toxæmic lesions are practically the so-called "cloudy swelling" in the internal organ as a result of the toxins so that the affected organ looks swollen pale and soft whilst microscopically its cells are granular and stain badly. Septicæmic lesions in lungs or blood vessels or brain or joints may give rise to septic pneumonia, infective endocarditis, phlebitis, meningitis and or arthritis.

Clinical types.—The severity of infection is largely determined by the nature of the infective organisms and the site and extension of the infection. Thus tears and lacerations of perineum are not attended clinically with severe symptoms. Bruises of the vagina are not so serious. It is the cervical tears and the placental site that give rise to more severe types. The reason is not far to seek. The cervix which during pregnancy is soft and vascular is in close relation with enlarged pelvic blood vessels and lymphatics. The raw surface at the placental site is in intimate contact with the hypertrophied highly vascular blood vessels of the uterus. Hence any toxins developed are readily absorbed and give rise to severe symptoms. Clinically the most severe types are found in cases of incomplete abortion, adherent placenta and prolonged labour, and after instrumental deliveries. Cases of incomplete abortions sometimes of several days' duration especially where an attempt had been made to get rid off the ovum for social reasons as in widows and unmarried girls, and in severe types of sepsis associated with peritonitis general or local. Adherent placenta is also a cause of graver type of sepsis because in the majority of cases the patient on admission is in a weak exhausted condition and stands the manual removal badly. Many cases come in a collapsed

condition and have to be treated for collapse before removal could be attempted. In such cases any septic infection is likely to spread unchecked due to the low powers of resistance on the part of the patient. Prolonged labour is by far the most common and most important cause of sepsis. The bruising of the soft parts due to the foetal head being jammed against them for a long time together with the lowered vitality of the mother due to exhaustion is a very favourable condition for the occurrence of sepsis. Next in importance come obstetrical operation particularly high forceps which is a fruitful cause of severe cervical tears. All these cases are divided into two types. The sapræmic and septicæmic, sapræmic where the organisms multiply locally and septicæmic where the organisms get into the blood streams or lymphatics and give rise to general infection. In severe types of sapræmia some organisms are likely to get into the blood and in septicæmia there is always a local site. Hence there is no hard and fast line of demarcation between the two.

Signs and symptoms.—The cardinal signs are fever, subinvolved uterus and offensive discharge. Fever is constant and is the earliest indication of septic trouble. If you cannot account a fever during puerperium say as due to mastitis or malaria, you should consider it as probably due to septic infection even if lochia is not offensive. It ranges from 99° to 102° in mild cases, it may go up to 104° or 105° in severe cases. With this fever there is rapid pulse. The pulse is rapid but not in proportion to the height of fever. You may find a temperature of 102° with a pulse of 100 or 110. In severe cases it may rise to 140 or even 160. A pulse rate above 140 is not of a favourable import. Hyper-pyrexia is rare and is one of the

terminations in our fatal cases. The fever shoots up to 106° and refuses to come down in spite of hydrotherapy or iced enemas. The uterus is large and slightly tender. Pain is not such a prominent symptom. There is a certain amount of distension except in mild cases. Diarrhœa is present in very severe cases and resists the usual treatment. It is a sign of bad import.

Lung signs are rare. There may be a certain amount of bronchitis or congestion at the bases in cases which have been in their back for a long time. Septic pneumonia may be present. Urine is high coloured, contains traces of albumen and is sometimes turbid. Sleeplessness is common and headache is sometimes present. In bad cases there is restlessness and delirium. There may be convulsions and coma especially towards the end. There may be phlebitis. It has not been so common in our cases. Some of our cases complain of shooting pain down the thigh from the hip. Sometimes it is only on one side, sometimes on both sides. On examination there are no signs of redness swelling or tenderness.

Hip joints appear normal. I do not know whether it is a neuritis, whether it is due to pressure of pelvic exudates in the nerves or whether it is due to tenderness in bones due to deficiency of calcium salts. It disappears as the septic trouble subsides. Some cases I treated on analgesics and some on calcium lactate. There was no rapid improvement and they took their own time to recover.

Lochial discharge—is offensive in most of the cases. It is purulent or mucopurulent. Always examine the diaper on the patient. The discharge on a diaper kept out and exposed, decomposes and smells readily. In very

septic cases it is brown or even black. In a few cases the discharge is purulent even where the temperature is normal. It is in cases where absorption has been little, e.g., where the pus is from infected perineal tears. In some cases of severe infection the discharge is not purulent. This is a thing to be remembered. Whereas an offensive discharge is a positive sign of sepsis, a non-offensive discharge does not disprove sepsis. Non-offensive red discharge is present in septicaemia condition. Nevertheless it is possible to distinguish the healthy red discharge and the septic red discharge. In healthy discharge there is a small circle of deep red in the centre and a thinner and paler area around it. The septic red discharge is uniformly deep red throughout and does not dry so readily on the diaper. There is a moderate degree of anæmia in all severe cases. In some serious cases the anæmic condition becomes visibly worse day by day. In other serious cases the anæmic condition is not so noticeable. Probably some toxins have greater destructive power on R. B. C. than others. According to the signs and symptoms cases can be classified into 2 varieties, the moderate and severe. There is one type about which I do not find any mention in the textbooks. I call it the fulminant type. I have seen only 2 cases. Both of them were natural deliveries. Both had not been handled by barber midwives. Both were well nourished and middle class patients. For 12 to 24 hours after delivery the temperature was normal. The temperature shot up to 104 or 106. The toxin seems to have a special action on the heart. The pulse rose to 160 or 170, got imperceptible and fluttering. Together with that, there was great restlessness, prostration, loss of consciousness and in spite of all treatment they succumbed in 48 hours.

Prognosis.—In sapraemia the prognosis is generally good. 90 per cent. recover. In septicæmia it is grave; mortality ranging from 50 to 75 per cent. according to books. In our hospital it is slightly less than 50 per cent.

Treatment.—It is a matter of grave reproach on our vaunted progress in all fields of medicine that a woman in the discharge of the highest and noblest of her duties, i.e., in entering into motherhood, should be jeopardizing her life. So great and definite is this risk that many of the Insurance Companies who are ready to accept the lives of people going to war provided an extra premium is paid, decline to accept the life of a woman unless she is a widow or spinster and puerperal sepsis has contributed not a little towards making child birth such a dread and danger.

The treatment can be divided into prophylactic and curative. Prophylactic consists in giving every woman clean healthy surroundings and proper attendance and help during confinement. The homes of our poorer classes are very insanitary and those of the middle classes too, are far from satisfactory. In these surroundings the chances of infection are great. To remedy this there should be sufficient number of Maternity Homes distributed in several centres where treatment is given free or for a nominal fee. The word hospital brings to one's mind a place where disease or abnormal conditions are met with and a person who hesitates to send his wife into a hospital may not have as much hesitation to send her to a Maternity Home. Secondly there should be a sufficient number of properly trained nurses who could be called at any time to attend confinements at homes. These two could be carried out only by the united efforts of community or

society and would go a long way in minimising the incidence of sepsis. As regards individual efforts to minimise

sepsis—

(1) Strictly adhere to aseptic and antiseptic measures paying minute attention to every detail.

(2) Avoid prolonged labour.

(3) At the same time avoid unnecessary interference remembering that *labour is a physiological and not a pathological process.*

Curative treatment.—On referring to several books on the subject about the treatment of puerperal fever one is surprised that when they are more or less in complete accord as regards aetiology, pathology, signs and symptoms no two books are unanimous or definite about any one line of treatment. One advocates in every case of sapraemia exploration of the uterus under anæsthesia. Another advocates in septic cases that have resisted milder treatment besides dressing the uterine cavity incision of the posterior fornix and drainage of the peritoneal cavity (Pryer's treatment). A third recommends douching the uterus with weak antiseptic as a routine measure whereas a fourth thinks it dangerous to give intra-uterine douches as a rule. Lastly there are not wanting some who decline to interfere with the uterus in any case. Such being the case it is no wonder that the reader who goes through several books on the subject is left in a state of suspense as to what he should do when he meets with a case of puerperal fever.

In this hospital which is probably the biggest maternity clinic in the East we have far greater facilities for studying and treating cases of puerperal fever than elsewhere and I dare say we get more severe types than in

the West due to lack of adequate medical aid coupled with poverty and ignorance of the masses. I shall therefore in the ensuing pages describe at some length the treatment carried out here which from actual experience has been found to give us most satisfactory results.

Indications for treatment are :

1. Remove the focus of infection and eliminate the toxins already absorbed.
2. Support the strength of the patient.

Treatment.—Carefully go through the history of the case, ascertain whether the woman had been examined outside by untrained midwives whether membranes had ruptured before or after admission into the hospital, whether she had been in labour for a prolonged period and if any assistance instrumental or otherwise had to be given before delivery. If delivered outside the hospital whether the after birth came soon after, if it was complete or incomplete. Find out also if there was hæmorrhage before and after delivery of the child. This will give you a clue as to the nature of the infection and the treatment you have to adopt. Then carefully examine the patient system by system because the fever might be due to causes other than sepsis and what is more important to remember besides puerperal fever there might be other intercurrent fevers.

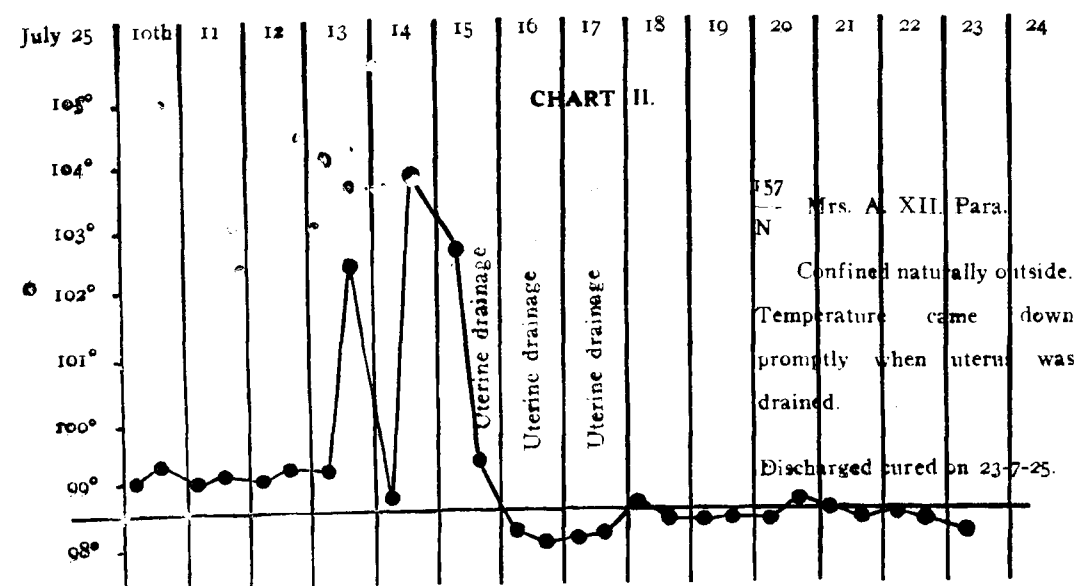
Mild Sæpraemia.—If on examining the patient you find she does not look ill, her pulse ranges between 80 to 100 per minute, temperature is normal or 99 in the morning and up to 100 in the evening, the uterus involuting fairly but the lochia being slightly offensive, order a brisk

purge, raise the head of the bed, put her on ergot and quinine mixture, Ergot $\frac{1}{2}$ Drm., Quinine 5 grains T.D. and fourth hourly hot vaginal douches. If the temperature comes down and the lochia is less offensive, continue the same treatment and the patient will be all right in a few days.

Moderate type.—If on the other hand the lochia becomes more offensive or if the fever rises above 100° or if the patient when you first see her has temperature running from 100° to 100° or even 102°, uterus large, lochia offensive, pulse anywhere between 100 to 120 per minute with tension fair and the patient looking quiet and not distressed, bring her to the table, wash the vulva very thoroughly, open the labia to see whether the urethral orifice and the vestibule are bruised. Pass a catheter and note the odour and color of the urine, see if it is cloudy, purulent or blood stained.

Look at the perineum for tears and see if there are any stitches which have gone septic and remove them and put in Sims Duckbill speculum and examine the vaginal walls, see if they are bruised and note the nature of the discharge in the vagina. Next take a sponge holder and gently lift up the anterior vaginal wall when the cervix will come into view. Note its condition whether it is inflamed, swollen, or torn or covered with sloughs. Give a vaginal douche thoroughly and pass in the Budin's tube. Some say to study the cervix with the Vulsellum. This is unnecessary and inadvisable. The cervix is congested, inflamed, readily friable and probably septic. And if the Vulsellum is used it will cause bleeding and will be a fresh place for absorption of infection. If the Budin's tube has to be passed for

several days the use of Vulsellum every day is to be deprecated. In a few cases the lips are so much torn and high up that you cannot study the cervix properly with the Vulsellum. Instead of that if the speculum rests properly with its end in the posterior fornix the cervix resting on it and if the anterior vaginal wall is lifted with the sponge holder you see the cervix clearly. This is painless, easy and gives the least amount of discomfort to the patient. When the cervix is in view pass the Budin's tube gently as far as it will go. When it has gone in close the outer end of that tube and draw it out and you will see the nature of the discharge. It may be merely red discharge or it may be yellow and purulent or with blood and pus mixed, together. Wash the Budin's tube and re-introduce it gently, press on the fundus and drain the pus; in mild or favourable cases purulent discharge comes first and then red non-purulent, non-offensive discharge or else only a small amount of pus slightly offensive. In such cases draw the Budin's tube, give a hot vaginal douche, touch any cervical tears or vaginal bruises with Tincture Benzoin Co., or Chloramine T. 1% solution. Pack the vagina lightly with gauze. Send the patient to bed, put her on E & Q., mixture fourth hourly, very probably the temperature will come down in the evening and will not rise so high next day. The general condition of the patient too will be appreciably better. Next day bring her up to the table and do similarly and the temperature will come down to normal in a few days. These are generally cases of mild Sapræmia where when the discharge in the uterus is properly drained the temperature comes down and patient improves (See Chart II). After delivery



the heavy fundus falls down on the lower uterine segment and sometimes gives rise to acute ante flexion or retroflexion and the kink produced interferes with the proper drainage of the uterus and if any infection gets in the uterine contents retained inside give rise to symptoms. It is especially in such cases that the simple introduction of Budin's tube clears away the accumulated discharge. This coupled with mixture and douches suffices in all mild cases. This can be done daily if necessary and requires no anaesthetic. In this connection a word must be said about the Budin's tube. The metal tube is not preferable to glass tube. In the latter you can clearly see what the discharge in the lumen of the tube is and you can see no particles of blood and pus adhere inside. The metal tube offers no such advantages. The gauze put in the vagina is taken 6 hours later when a hot vaginal douche is given. If the vaginal wall is very sore and bruised there is no need to put in the speculum.

Introduce your finger up to the cervix and glide the Budin's tube along into the uterus. This is an easy procedure and disturbs the patient least.

Severe Type.—In cases where uterus is large and tender with offensive discharge temperature ranging from 100° to 102° in the morning and up to 103° or 104° in the evening with pulse ranging from 120 or 140 or more per minute with rapid respiration and patient in distress, or, even in milder cases where there is profuse purulent discharge from the uterus, some further toilet of the uterus is necessary. It is very likely in these cases that the organisms are more virulent or pieces of membrane and blood clots have been left in the uterus. In such cases bring the patient to the table, give her 2 drams of brandy well diluted if the pulse is not full. Give a vaginal douche and introduce the Budin's tube and drain the contents. Now take a piece of sterilized gauze and with the uterine forceps introduce into the uterus. Twist it round and round when pieces of blood clots membranes, etc., will adhere and come out. Draw the piece of gauze and introduce another till no more foreign matter adheres to the gauze. Now take another piece, dip it in Tr. Iodine or any other antiseptic, introduce it into the uterus turn it round and round and round and apply to the interior thoroughly. Dress the cervix and vagina as before and the temperature is sure to come down. This procedure is not required more than once or twice and an anaesthetic is not necessary. Digital exploration of the uterus is not necessary in the most of the cases. Where there is a reasonable doubt that the placenta is not come out entire especially after abortions and premature labour, anaesthetic should be given and the uterus thoroughly explored

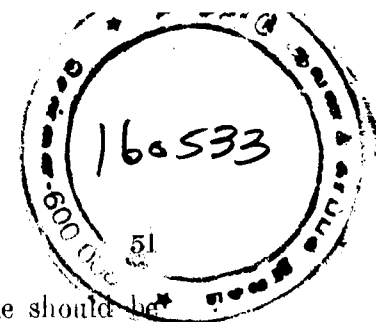
PUERPERAL FEVER.

with the finger. Any pieces remaining inside should be removed, the interior of the uterus stroked with a blunt flushing current if necessary and an intra uterine douche is given to wash out the contents.

The offensive lochia is a sure sign of puerperal sepsis but it is as well to remember that the degree of offensiveness is not proportional to the degree of infection. Offensive pus in the uterus is compatible with normal temperature and there may be hardly any smell in the lochial discharge in a case of Septicaemia.

Uterine douche as a routine measure in every case of infection has been given up for several reasons. One is shock especially in feeble and anaemic patients. Very often the temperature shoots up in the evenings. This is because the disturbance of uterine cavity causes increased absorption of toxins resulting in a negative phase. There is probably the danger of infection being carried upwards into the tube, but douching may be given in a few cases where the condition of the patient is very fair but the lochia exceedingly offensive. In such cases douching the uterus with a few pints of lysol or preferably eusol stops the smell and diminishes the discharge. These are cases of Sepsaemia where the walls of uterus have absorbed very little infection.

Septicaemia. In these cases patient looks very ill with temperature ranging from 102° to 105° with very little remission, pulse rapid, feeble, patient semiconscious or fully unconscious, tongue tremulous, furred, dry and often covered with sordes, the forehead extremities are often covered with perspiration. These cases are very few and are those that are admitted after incomplete abortion outside. Lochia may be suppressed or if present



at all is scanty and slightly offensive. In such cases local treatment is not of much avail. The treatment is to be carried on general lines. Head of the bed is to be raised, iron, quinine and ergot mixture to be given internally. Hydro therapy, nourishing diet and stimulants to be attended to. The only special treatment is administration of 10 to 20 c.c. antistreptococcus serum every day to be repeated if the patient's condition is hopeful. More attention should be paid to keep up the patient's strength and to put her on nourishing diet than on local treatment.

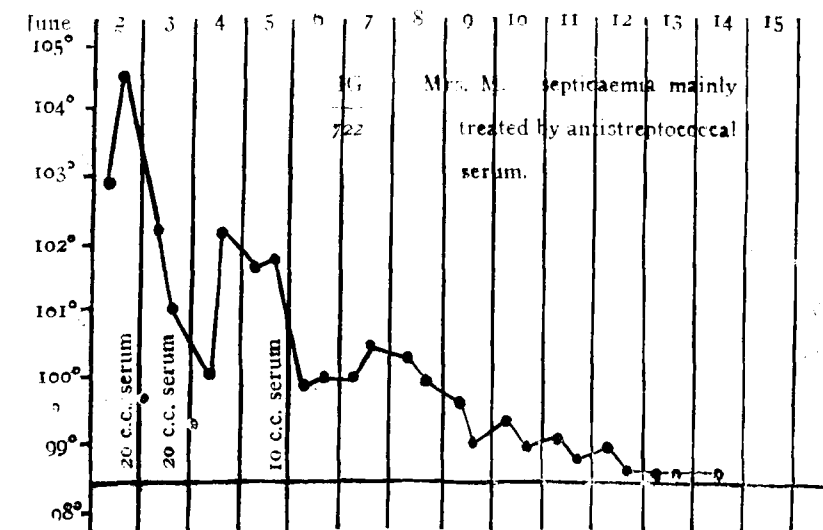
Cases with signs of local peritonitis and parametritis. In these cases the adjoining peritoneum and cellular tissues are also involved. Besides usual signs of a severe type of uterine infection there is pain, tenderness and board like rigidity of the lower abdomen. In these cases if the condition of the patient permits, bring her to the table and examine the parts. Give a hot vaginal douche and drain the contents of the uterus with the Budin's tube. Put her to bed in Fowler's position. Give her soda sulph. dram 1, every four hours, fourth hourly hot vaginal douches. Fourth hourly turpentine stupes to the lower abdomen with second hourly stimulants like glucose brandy, chicken essence to be supplemented with glucose saline per rectum and pituitrin one c.c. morning and evening. When the patient improves order glycerine and ichthyol tampons at bed time to hasten the absorption of inflammatory exudate in the pelvis. Cases which looked very bad on admission have come round under this line of treatment.

Cystitis.—In every case of fever where the uterus is involuting properly and the discharge is not offensive it is always necessary to remember about infection of

urine. Organisms get into the bladder through the urethra or directly as a result of bruising of the vesico-vaginal wall. Put the patient on Hexamine and acid sodium phosphate grains 10 T.D. and if necessary when the urine is turbid or offensive, wash out the bladder with warm boric lotion. The urine will clear completely in a few days.

One must always be on the look out in cases of puerperal fever for other inter-current fevers as well, Malaria, Enteric, may exist with puerperal fever. We had one case of puerperal fever complicated with enteric which but for the timely diagnosis would have fared worse by frequently handling the patient to the table and back.

Serum and Vaccine.—Antistreptococcal serum was used more frequently this year than during last year and we have received very encouraging results. It is effective as a prophylactic in cases where you expect the case to become very septic and earlier it is given when sepsis supervenes, better are the results. See Chart III.



In cases badly handled outside and then admitted to hospital or in cases of adherent placenta where the child has been confined at home and the mother is brought to the hospital sometime afterwards it is given as a routine. We find that the supervening sepsis is not severe and the patients recover quicker than we anticipated. Of course there are cases where it has failed. Why, it is not definitely known. It may be that the serum has no effect in neutralising the activities of the particular strain of Streptococci present. See Chart I. It may be there is a mixed infection like strepto, staphylo and pneumo cocci or that the vital centres have been already damaged. It is given now in 20 to 25 c.c. doses every day or every alternate day generally 2 to 4 doses suffice.

Vaccines.—Vaccines we try only in subacute cases. We have now a case of coli infection who has been in the wards for some weeks getting-examine and auto-vaccine. We want to make a vaccine from the various strains of streptococci isolated from septic cases and then try them intravenous therapy. I have been asked what has been my experience of intravenous therapy like injections of Tr. Iodine, Chloramine T, Acriflavine and Urotropine. I am not able to give you a definite report about these. Most of our cases respond favourably to the line of treatment described above and there has been no necessity for this. Where the cases did not respond favourably to our line of treatment these were tried with no better results. Hexamine must be good in coli infection but in the very acute types I had tried, it gave no good result. Where the patient gets low fever after confinement and where apparently no cause can be found it is well to

remember particularly two things—B. Coli infection and incipient tuberculosis. Have the urine examined bacteriologically and give a few injections of auto-vaccine or stock vaccine if it is coli infection. If this fails watch for signs of tubercle.

I shall close this paper by mentioning a few words about the disinfectants and stimulants used in the treatment of sepsis.

Disinfectant.—Lysol dram 1 to 2 per pint. Tr. Iodine same strength. Eusol Tr. Benzoin Co., and lately Chloramine T 1 to 2 per cent. have been used with very encouraging results.

Stimulants.—We rely chiefly on brandy, chicken essence and glucose, dram 2 each every four hours. Arrack though not so good as brandy gives satisfactory results. Rectal saline is a very valuable stimulant in puerperal condition. Pituitrin has a peculiar value in obstetrics as it promotes involution of the uterus, acts as stimulant and prevents tympanitis.

In conclusion my best thanks are due to Dr. A. Lakshmanasami Mudaliar, B.A., M.D., our Asst. Superintendent, for having kindly gone through this paper and to Lt.-Col. C. A. F. Hingston, C.I.E., O.B.E., I.M.S., our Superintendent, for having given me permission to publish this paper.

CLINICAL NOTES.

A CASE OF CIRCUMSCRIBED SCLERODERMIA
OR MORPHEA.

(By CAPT. M. NARSING RAO, L.M. & S.)

Miss L. D. aged 25 has had coloured and white patches on her body for a long time. She began getting these when she was a child of three years. The patches make their appearance as brownish coloured patches, hard and thickened and "caught-up-like." They vary in size from that of a quarter anna piece to that of the palm. Some time after the appearance of these brown patches, appears over the middle of the brown a white, shiny, "Ivory-like" patch, the whole thing now looking like an ivory patch with a pigmented brown coloured ring round it. Patches like these make their appearance in successive crops while slight improvement takes place over the older patches; although they do not completely disappear. The brown patches in the early stages are slightly itchy. She also complains that the hair on her head is rather thin.

She has never worn a corset.

Family History. Nobody in the family has similar patches on the body. Nobody in the family has ever suffered from Leprosy. Mother has had cystic goitre for the last thirty-two years and during the last twenty years or so has been suffering from a severely itching chronic eczema over both legs and ankles. The constant irritation and scratching has made the skin over the area quite full of warts.

Physical Examination. The patient is a well-developed and plump person, rather inclined to be fat. The brown ringed white patches are seen all over the body

except the face and hands. They are well marked over the breasts, thighs, abdomen and legs. One or two white patches on the legs are white and shiny, look slightly depressed, and are hard and "parchment-like" to the feel. Examined with a lens, these shiny white patches are found to be without any hairs towards their centre; but towards the periphery hairs can be seen. These hairs are not discoloured. Round the white patches can be seen a circle, coloured slightly brown, about a quarter of an inch in width. Running across the white patches and standing out in bold relief, a blue vein can be seen. Neither the white nor the brown portion of the skin showed any scaly appearance, and examination of the superficial epidermis under the microscope did not reveal any fungus or spores. The cutaneous sensation is absolutely normal to touch, heat and cold and pain.

Most of the white patches which are improving, show the presence of distinctly coloured hairs over them. They have lost their ivory appearance and parchment feel. Pigment has not yet made its appearance over these patches. Otherwise they are quite normal. The secretion of sweat over these areas could not be ascertained definitely.

No patches could be definitely located as running in the course of any definite nerves as described by Sequera.

No enlargement of any glands.

Pathology of the disease has been described by various authors as a neuro-trophic condition resulting in an infiltration of patches of skin by small cells which by physical pressure over the arterioles obstructs them causing a failure of blood supply to the patch of skin thus producing atrophy. Each patch of skin is described as

the base of an infarct in the skin. The white patch is described as having a lilac or rose coloured margin on account of underlying blood capillaries. (The description of colour referring as it does, to European and American cases, a little allowance has to be made about the colour of the surrounding area, which in this case is more of a brown.)

Aetiology.—The disease is described as being more common among women than among men and more frequent during young age being most common between the ages of 10 and 30.

Diagnosis.—*Teanea Versicolor* has a scaly appearance of superficial epidermis. The fungi and spores are easily seen under the Microscope. Has no ivory patches over the discoloured patches.

Luecoderma.—Here the appearance of skin is quite normal except for the loss of colour surrounded by a ring of hyper pigmentation. There is no ivory appearance over the patch. Nor does the skin at any time get infiltrated and thickened giving a "caught-up-like" feeling. The hairs always lose their pigment and generally have the same discoloured appearance.

Leprosy.—There is always loss of sensation. The discoloured patch never gains an ivory appearance. The hairs over the area completely disappear. The preliminary pigmentation with infiltration and thickening is not noted.

Note.—The case has been for a long time suspected to be Leprosy. Patches of skin cut out of the body, nasal, mucous, etc., have been examined by competent persons and declared to be negative.

The disease appears to be rare, only a very few cases having been mentioned in literature. In this country, owing to the wide prevalence of luecoderma and leprosy, it is in all probability mistaken for the one or the other.

Treatment.—McCarrison mentions scleroderma as one of the diseases caused by hypothyroidism. Harrower and Sajous report four cases of scleroderma as having been cured with Thyroid gland by Levi and de Rothschild. Other dermatologists also recommend Thyroid gland treatment. All these successful cases, together with the fact that the mother is suffering from cystic goitre and was suffering from the disease at the time the person was born, has lead me to adopt treatment with Thyroid gland. To avoid any unpleasant results from Thyroid, it has been combined with liquor arsenicalis as recommended by Sir Norman Walker. Thus $1\frac{1}{2}$ grains of Thyroid with Liquor Arsenicalis m. xii daily is being given.

Special points noted in connection with this particular case are (1) The patches may appear in successive crops and as some of the patches improve spontaneously others may make their appearance. (2) The disease is very chronic, in this case having lasted for 22 years and yet showing no signs of complete disappearance spontaneously. (3) The patches have a special predilection for the breasts. This has been attributed to irritation from pressure of the corset. In this case as the person has never worn a corset, it is quite clear that pressure from corset is not an essential factor.

I am thankful to the lady for permitting me to report the case.

CORRESPONDENCE.

The following has been sent to us with a request for publication:—

BOMBAY MEDICAL COUNCIL.

In accordance with a resolution passed by the Bombay Medical Council at a meeting held on 20th September 1926, the following summary of its proceedings is published in the medical press for general information.

1. Mr. Joseph Michael Pereira, M.B., C.M. (Edin.), was called before the Council to answer a charge of issuing sick certificates in a case in which he had not been in personal attendance on the patient during the period covered by the certificates.

Mr. Pereira was cross-examined and questioned personally and after deliberation on the previous correspondence and the personal explanation offered, the Council decided that Mr. Joseph Michael Pereira had been guilty of infamous conduct in a professional respect and directed the Registrar to erase his name from the Medical Register.

2. Mr. Purushotam Narotam Patel, a registered practitioner, was called before the Council, but stated that he was unable to appear in person or by a legal representative. The Council therefore considered the charge, namely, that he had been convicted by a First Class Magistrate of making a false statement and fabricating his register of patients to support this false statement. On appeal to the Sessions Judge, the conviction was upheld.

The Council decided that Mr. Purushotam Narotam Patel had been guilty of infamous conduct in a professional respect and directed the Registrar to erase his name from the Medical Register.

3. Mr. Dharamdas Kaushalyadas, an unqualified medical practitioner at Bulsar, submitted an application

CORRESPONDENCE.

for recognition and registration. This was supported by certificates as to capacity for medical practice signed by Mr. B. M. Khambatta, L.M.S., and Mr. T. C. Khandwala, L.M.S. The Council decided that as the applicant had not had training in any medical school, he cannot be registered.

Mr. Khambatta and Mr. Khandwala who issued the certificates submitted were warned that their action in associating themselves with an unqualified practitioner was highly irregular [*vide* Ethical Suggestions No. 1 (c)].

4. It was decided that the draft of a revised Code of Medical Ethics be circulated to the various medical societies in the Presidency for their opinion and remarks. This will be considered at a subsequent meeting of the Council.

5. An application from Dr. R. H. Bhadkamkar, M.D. (Bom.), whose name was erased from the Register on 17th February 1921, for restoration, was considered.

Dr. Bhadkamkar having given an unqualified assurance that he would sever all connection with Messrs. N. Powell & Co., where he had been acting as Director of their Laboratories, the Council, after due deliberation, decided to permit Dr. Bhadkamkar's name to be restored to the Register.

ASSOCIATION NOTES.

The following are the replies received for some of the representations made to the authorities on behalf of the Association:—

(1) *Representation.*—Extending the Government orders granting daily allowances to officers studying at the Tropical School of Medicine, Calcutta, so as to make it applicable to officers studying at any other recognised institution in India.

Reply.—Members of the Provincial Medical service are at liberty to take leave for post-graduate study in any medical institution in India which provides *recognised* post-graduate courses.

(2) *Representation.*—The disciplinary rules applicable to gazetted officers of the rank of Civil Assistant Surgeons shall secure to them the same safeguards as those enjoyed by gazetted officers of all departments in general, *viz.*, that orders of suspension, stoppage of increments and the like shall not be passed without the sanction of the Local Government.

Reply.—The powers of appointing and posting Civil Assistant Surgeons have been delegated by Government to the Surgeon-General. The powers of punishing Civil Assistant Surgeon by reduction, stoppage of increment and suspension lie at present with the Local Government.

A REQUEST.

Members who have not yet sent in the subscriptions due from them are requested to send the same as early as possible to Dr. A. S. Mannady Nayar, M.B., PH.D., Secretary (Collections), Professor of Bio-Chemistry, Medical College, Park Town, Madras. The next issue of the Journal will be sent per V.P.P. to those in arrears whose remittances do not reach the Secretary before the despatch of the said issue. Members in arrears are earnestly requested either to send their remittances early or to accept the V.P.P. when it reaches them.

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The only member of our Service who has been included in the recent Honours List is **Khan Sahib Muhammad Aziz-ul-lah Sahib Bahadur**, who has been promoted to be "**Khan Bahadur**". Our warmest congratulations to him.

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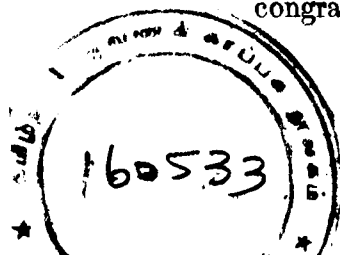
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