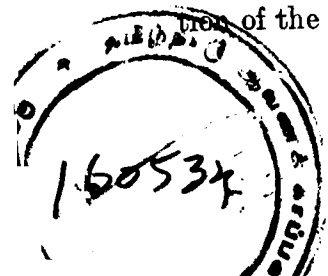


tuberculosis, but it is not unusual to find advanced cavity cases with a blood picture not far from the normal. There is no doubt an oligæmia which can be accounted for by the general loss of weight, sleep sweats, diarrhoea and expectoration, which must tend to lessen the quantity and drain the elements of the blood.

The hæmoglobin content shows no characteristic changes as a result of tuberculous infection. A mean of a large number of hæmoglobin values of patients in different stages of the pulmonary disease gave an average of 75 per cent. hæmoglobin in all irrespective of the stage of the disease.

There is no appearance which can be considered as pathognomonic of tuberculosis as regards the condition of the leucocytes of the blood. Craig (1905) has observed a leucocytosis in tuberculosis advancing with the stage of the disease from 10,000 in stage I to 14,000 in stage III with a decrease of the eosinophiles. Ullom and Craig (1905) shewed that increase of lymphocytes in the blood corresponded to increase of resistance in the patient to tuberculosis and Webb and Williams (1909) confirmed these results by their observations in a number of cases. These latter workers found that lymphocytes increased in number by residence in high altitudes and the correlation between such increase which has been confirmed by later observers and the improvement in the pulmonary condition by a change to the mountains is of interest from a pathological standpoint.

Watkins, Wright and King (1911) consider that the percentage of leucocytes closely corresponds to the condition of the patient and the activity of the disease.



Bender and De Witt by experiments on animals shewed that in acute tuberculous infection, there is either a terminal leucocytosis or a leucopenia, a progressive decrease of lymphocytes and an absence of eosinophiles, whilst in the chronic form no marked changes occur in the leucocytes.

Scholz (1912) showed that in cattle and other animals polymorphoneuclears decreased and lymphocytes, mononuclears and eosinophiles increased in recent tuberculous infection.

Murphy and Taylor (1914-1919) proved by experiments on animals that by increasing the lymphocytes by inoculation with cancer cells, resistance to tuberculosis was also increased in the animals.

Calmette (1920) by an exhaustive study of the pathology of the blood in tuberculosis gives 3 different formulæ for leucocytes—(1) moderate leucocytosis, pronounced lymphocytosis and slight eosinophilia in benign fibroid cases, (2) hyper-leucocytosis, abundant polynucleosis and diminution of lymphocytes and eosinophiles in massive infection and severe toxæmia, and (3) marked hyper-leucocytosis with 90 per cent. polymorphoneuclears and diminished mononuclears and lymphocytes and almost absent eosinophiles in sinking cases.

Bergel thinks that high mononucleosis is a defensive element in tuberculosis and is of favourable significance. Webb, Gilbert and Newman give the following lymphocyte counts from a survey of 400 cases of tuberculosis :—

- | | |
|-----------------------------------|-----------------|
| i. Normal individuals .. | .. 43 per cent. |
| ii. Arrested tuberculous cases .. | 45 .. |

iii. Improving cases	..	.. 37 per cent.
iv. Failing cases	..	.. 24 „
v. Dying cases..	..	.. 16 „

They found that in children lymphocytes are normally higher than in adults and in children with arrested tuberculosis lymphocytes are the highest. They stressed this point so much that they considered that children with less than the average number of lymphocytes in their blood required a thorough overhauling.

Rosenthal (1923) considered that a high lymphocyte count is a favourable sign in tuberculous infection, whilst a low count means a bad prognosis, though exceptions are occasionally seen to this rule. 30 per cent. lymphocytes and 4 per cent. eosinophiles was in their opinion a favourable count.

Eicke considers that marked leucocytosis and absence of eosinophiles is of bad augury and that a high lymphocyte count and eosinophilia are of good prognosis.

McDoughall says that when secondary infection of cavitation sets in, there is polymorphonuclear leucocytosis and Stein and Urbmann go to the extent of affirming that absence of leucocytosis means absence of cavities in pulmonary cases. Leucopenia however sets in in miliary tuberculosis.

Grawitz considers the degree of leucocytosis gives an indication of the stages of the pulmonary disease.

It is reasonable to conclude from the above observations that the tubercle toxin inhibits leucocyte production in the bone marrow and that the toxins of secondary

organisms like the pneumococcus, streptococcus, etc., stimulate the bone marrow to produce leucocytes.

An increase of blood platelets indicative of increase of opsonins has been described by Webb, Gilbert and Newman. The presence of the leucocytic inclusion bodies described by Dohle in tuberculosis and other infectious diseases has been confirmed by Tominaga and others. Their increase in a case is supposed to indicate the seriousness of the disease and to have a bearing on the prognosis. When Dohle's bodies are present in over 50 per cent. of the leucocytes the prognosis is said to be grave.

Arneth's Index.

Ever since Arneth (1904) described the leucocyte picture which he considered to be characteristic of tuberculosis and other infectious diseases, a good deal of work has been done to prove the correctness or otherwise of his views. Arneth considers that when an infectious disease gets worse, younger forms of leucocytes with less number of nuclear lobes are set free from the bone marrow in large numbers, so that an index 'shifting to the left' he considers to be of definitely bad prognosis. Arneth is of opinion that the myelocyte which is the youngest leucocyte gets its nucleus gradually indented and ultimately lobulated, so that in infectious diseases the immature forms are liberated in large numbers increasing the percentage of the uni- and bilobed leucocytes. Gulland and Goodall note that an unusually large number of neutrophils with rounded and kidney-shaped nuclei indicates severe infection. The standard in normal health of Arneth's count has differed with different observers (*vide* Table I).

Table I—Arneth's count in Normal health.

	Arneth.	Cooke.	Miller & Reid.	Hamilton Black.	Minor and Ringer	McDougall	Ours.
Leucocyte of Class I	5	12	5	8	2.5	9	6
" " II	35	25	26	28	16.2	31	11
" " III	41	46	36	42	35.6	43	60
" " IV	17	15	28	22	18.6	15	18
" " V	2	2	5	22	5	2	5

The following figures represent the results obtained by different workers in the respective diseases indicated in the table. The index is obtained by adding up the percentages of Class I, Class II and $\frac{1}{2}$ of Class III, which is the method recognised by most people as the best one of representing the index :—

Table II—Arneth's count in Pulmonary Tuberculosis.

Authority.	Condition.	Class of leucocyte					Arneth's Index.
		I	II	III	IV	V	
Arneth	Normal health	5	35	41	17	2	60.5
"	Acute Miliary Tuberculosis.	36	56	8	0	0	96
"	Acute T.P.	14	56.5	24.5	4.5	0.5	82.7
"	Advanced Active T.P.	29.5	60	9	1.5	0	90
Cooke	T.P.	20	30	42	8	0	71
Hamilton Black	Chronic T.P.	31	34	28	7	0	79
Taylor	T.P.	31	57	10	2	0	93
Bushnell and Treuholtz.	T.P.	..	..	..	..	..	67
Webb & Williams	..	..	..	..	..	..	65
Minor & Ringer	..	..	..	..	..	..	43.5
McDougall	..	..	..	..	..	..	57
Miller & Reid	..	..	..	..	..	..	46.5
Ours	Normal health	6	11	60	18	5	47
	Early tuberculosis	7	16	55	17	5	50.5
	Advanced T. P. (anatomically)	8	14	58	16	4	51
	Advanced T. P. (toxaemic)	8	10.5	65	13	3.5	51
	Fibrosing T.P.	6	9	59	21	5	44.5

As will be seen from Tables I and II the range is so wide of the figures given by different observers for normal and for infective conditions, that each observer must construct his own figures in his own work.

Pappenheim gets his leucocyte index by totalling the number of segments in 100 neutrophile leucocytes so that his figures vary between the two theoretical extremes of 100 and 500.

Taylor and Wilson consider that an Arneth's index between 40 and 60 indicates normal health or very early T. P. and marked laevo-deviation and indices of 75 to 95 or more indicate severe infection. Prognosis can be given often without seeing the case by simply noting whether there is laevo or dextro deviation. Extensive laevo deviation even with an apparently early condition is of bad prognosis and absence of laevo deviation in anatomically advanced disease is of favourable prognosis.

Our figures obtained from an analysis of 122 cases though quite different from Arneth's and others' have been fairly consistent as regards the deviations though the very great laevo deviation observed by Arneth is not so evident. The counts of the unilobed and bilobed nuclei have been very low in our series and the Arneth's index has in consequence been much lower. It is interesting to note that an index of 47 in normal health, rises to 50.5 in the early cases, 51 in cases advanced either from the anatomical or systemic sense, falling to 44.5 again in fibrosing and recovering cases. That the index in fibrosing cases is even lower than in normal subjects, is probably due to the fact that a good number of our normal subjects were from amongst patients who attended the Tuberculosis Institute for chest complaints and were diagnosed

as non-tuberculous respiratory affections. The possibility of a few of these cases being incipient or hilus infections and the fact that they were not strictly normal subjects even if non-tuberculous, coupled with the fact that most of the fibrosing cases were under the best dietetic and hygienic conditions of the Tuberculosis Hospital, presumably accounts for the apparently higher index in the normals than in the 'fibrosing cases.'

We agree with McDoughall that there is a paucity of class IV of the polymorphoneuclear in advanced tuberculous conditions the figure for our toxæmic group being the smallest, *viz.*, 13% and that for the fibrosing cases being the largest, *viz.*, 21%. It means that the leucocytes with the most defensive power are scarce and the mono and bi-lobed leucocytes with poor phagocytic power are more abundant.

Differential Count.

This has given quite consistent results in our hands. An increase of polymorphoneuclears is synchronous with advanced disease coexisting with a diminution of the lymphocytes. There is an increase of lymphocytes in the early stages of tuberculosis (21.5%) and the same high lymphocyte count (22%) is observed in the fibrosing cases as well. There is marked diminution in their number in the more advanced and toxic types of the disease (17% to 15%). Eosinophiles which work out at 2% in the normal subject, form 3.5% in the early stages of tuberculosis going down to 2.5% in the more advanced stages and 2% in the sinking cases. The persistence at the comparatively low figure of 2.5 in the fibrosing cases is probably due to experimental error, the number of such cases in our series having been small.

Table III—the differential Leucocyte Count in different stages of Pulmonary tuberculosis.

Class of cases.	Differential count.			
	Polymorphos.	Large monos.	Lymphocytes.	Eosinophiles.
Non-tuberculous cases ..	72%	4.5%	21%	2%
Early tuberculous cases ..	69.5%	5.5%	21.5%	3.5%
Advanced tuberculous cases (anatomical) ..	75.5%	5%	17%	2.5%
Advanced tuberculous cases (toxæmic) ..	78%	5%	15%	2%
Fibrosing cases ..	71%	4.5	22%	2.5%

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NEW IDEAS ABOUT THE DIAGNOSIS AND TREATMENT OF DIABETES MELLITUS—II.

BY DR. B.V. VARADACHARIAR, B.A., M.B. & C.M.
THE MODERN TREATMENT OF DIABETES BY DIET.

Till recently, the dietetic treatment of the disease consisted in limiting the carbohydrate or the starchy foods and giving plenty of fats and proteins instead.

This was based on the assumption that the carbohydrate metabolism was the only one at fault. Now it is established that all the three kinds of foodstuffs, namely, carbohydrates, fats and proteins are involved in the disordered metabolism. (This was already pointed out in the beginning of this lecture.) Consequently the principle of restricting the carbohydrate and giving excess of the other kinds of foods, has now been abandoned and a well-balanced diet consisting of all the three kinds of foodstuffs mixed in proper proportion based on the metabolic needs of the body, is now advised.

It is Dr. Allen, the great American worker in diabetes who conducted experiments on animals and placed the dietetic treatment of diabetes on a scientific basis. A brief reference to his experiments is not out of place. He removed a large portion, *i.e.* 9/10ths of the pancreas of a dog leaving behind a small portion, *i.e.* 1/10th only. This produced diabetes in the animal. When the dog was given a large carbohydrate or a fat diet, the animal got thin gradually and died soon. But when it was fed on a low diet containing all the three kinds of foodstuffs, it kept up its health and no glycosuria followed. When the carbohydrate was restricted, but the fats and proteins were in excess diabetes followed. This showed that restriction of carbohydrate alone did not help in the experimental diabetes, but all the foodstuffs had to be proportionately limited. From these observations, Allen inferred that human diabetes also should be treated on the same lines, *i.e.*, the minimum of food necessary for the requirements of the body containing all the foodstuffs should be given, if improvement were to follow. It is on

this principle that the modern dietetic treatment of diabetes is founded.

Allen's dietetic treatment—Starvation treatment—Treatment by alimentary rest.

The principle of this treatment consists of two main stages :—(1) to starve the patient till his urine is free from sugar and then (2) to give him gradually increasing amounts of different foodstuffs, in order to find out how much diet he can tolerate, without passing sugar in the urine. *That diet* with which he just begins to pass sugar, is his tolerance limit and *that* will be his final diet, which he should go on taking for ever afterwards.

The patient should stay in a hospital for the first few days or he must stay quietly at home without doing any work. For the first 2 or 3 days no treatment is given. The patient is given his ordinary food, his symptoms and physical signs carefully studied. A specimen of 24 hours urine is examined and the amount of sugar per ounce estimated. Presence of albumen, diastetic acid, etc., noted. The condition of teeth should be attended to and a dentist may be called if necessary. The patient's blood-pressure and weight are taken. We should record the daily output of sugar. If possible a blood-sugar estimation is made. All this will take 2 or 3 days. After all this is done, the *treatment is commenced*. The patient is confined to bed for 2 or 3 days and the bowels are kept open daily with salts. This is a very important point and should not be neglected. *He is made to starve till his urine is free from sugar for 24 hours*. The period, during which urine becomes free from sugar varies with cases. It generally takes 1 to 3 days. *Starvation* does not mean *absolute starvation*. He can take tea or coffee without sugar as much

as he likes. He can have 2 or 3 cups of Bovril or clear soup or *leuco*. He can take plenty of water or soda water. He can have about 6 to 8 ounces of *twice boiled green vegetables* or what are called 5 p.c. vegetables such as cabbage, spinach, French beans, etc., or any of our ordinary *கீரை*. Very little carbohydrate is left in a green vegetable of this kind when it is boiled twice, but the patient has the mental satisfaction of taking some bulky food. If there is acidosis or the patient is feeble, he may be given 4 oz. of whisky or brandy well diluted with water and distributed over 24 hours. Under this treatment, sugar disappears from urine within 1 to 2 or 3 days. At the end of this period, the patient may feel a bit weak, but has the supreme satisfaction of finding that the sugar in the urine has dropped *from 2 to 3 thousand grains to zero*. When the urine is sugar free, we give small quantities of only carbohydrates of the 5 p.c. **class* for 2 or 3 days and add proteins gradually for the *next 2 or 3 days* and then commence adding *fat very gradually*. The idea of giving fat after so many days is not to increase the tendency to acidosis. After the urine remains sugar free for 24 hours for 2 days, we give about 250-300 grammes, *i.e.*, 8 to 10 ounces of twice boiled 5 p.c. vegetable, with plenty of weak tea or coffee without sugar. This amount of carbohydrate should be divided over the three or four meals of the day. On the third day, a little protein and fat are added, *e.g.* one egg is given twice in the course of the day.

* Vegetables are divided into 4 main classes : 4 p.c. vegetables, 10 p.c., 15 p.c., 20 p.c. vegetables.

5 p.c. vegetables, spinach, cabbage, tomato, radishes, pumpkin, vegetable marrow, French beans, brinjals, cauliflower, Brussels sprouts, sorrel.

10 p.c. : onion, carrots, beetroot, turnips.

15 p.c. : Green peas, artichokes.

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10 p.c.: onion, carrots, beetroot, turnips.

15 p.c.: Green peas, artichokes.

20 p.c.: Potatoes, boiled rice,

On the 4th day 3 eggs are added. On the 5th day a little cream and a small quantity of meat is added. On the 7th day the patient starves again, i.e., he takes coffee, tea, clear broth, or Bovril or Lemco, etc. On the 8th day he takes a little more diet than on the 6th day. In this way, the diet may be increased gradually in carbohydrate, fat and protein, if he does not happen to pass sugar in the urine. Every day the urine should be examined and if on any day there is a trace of sugar, he should starve one day and on the next day when the urine is sugar-free, he should go back to the diet with which he did not pass sugar.

This gradual process of increasing the different foodstuffs goes on till the *calory value* of his food reaches the maximum which he can have. He cannot go on increasing his food to the level which he was having prior to the treatment. There is a certain limit fixed for him and his maximum food should be this limit. *This limit is fixed in the beginning of the treatment* alone, as follows:—It is calculated that a man who is doing no work, but is staying at home quietly, requires at the rate of 15 *calories* per lb. of his body weight. That is, if the man's weight is 120 lbs., he requires $120 \times 15 = 1,800$ calories* for maintaining his health. In ordinary life, a man who stays quiet at home takes much more food than this, but for a diabetic of this weight—120 lbs.—this is the maximum food that is necessary. During the treatment, a record of the total quantity of the food he takes daily in terms of grammes of carbohydrate, fat and protein and also the *total calory*

* *One caloric*.—The amount of heat required to raise the temperature of Kilogramme of water through one degree centigrade.

1 gramme of fat—9 calories.

1 gramme of protein—4 calories.

1 gramme of carbohydrate—4 calories.

value of the food, should be kept. If we want to determine the values of different foods in grames of carbohydrate, fat and protein, we have to refer to the tables published in books on physiology. Suitable diets can also be built up by reference to tables giving the composition of different foodstuffs. The physician should get familiar with the necessary calculations.

Let us see how far this fasting treatment is practicable. It is suitable only in the case of intelligent persons who co-operate with the doctor and who carry out this instructions to the very letter. It is quite unsuitable in the ordinary type of hospital patient who is ever ready to beg, borrow, or steal his food from his neighbour to satisfy the cravings of his stomach. Another great objection to this treatment is that it is rather complicated, requiring the knowledge of the composition of the different foodstuffs and their calory values. As regards the success that has attended this treatment, a large percentage of cases have been permanently cured, but a certain number fail to respond to the treatment. In these unsuccessful cases, recourse should be had to *insulin*. The starvation treatment is found to be specially suitable to the mild form of diabetes which is prevalent in India. I say that it is specially suitable to Indian cases, because it can be easily reconciled to their traditional fasting habits. Fasting is enjoined by the Hindu religion on special days such as Ekadasi, and therefore, there will not much objection for fasting for the sake of treatment. Our ancestors attached a lot of importance to fasting, and periodical starving was one of their religious duties. Fasting certainly gives alimentary rest and is extremely useful especially in the case of the vegetarians of South India who

are habituated to take a large carbohydrate diet twice or three times a day. It may not be a panacea for all political and social evils of the present day as Mahatma Gandhi thinks. But it appears to be certainly a very useful adjunct to treatment, in a number of diseases connected with faulty dieting.

Regarding the starvation treatment of diabetes, Lt.-Col. Waters says "The more experience I have in using the method, the more convinced I am that it is the method of election for use in India." Lt.-Col. Waters has published in his book on "Diabetes," a number of cases which are striking examples of cure of the disease by this method. I quote here one case only, for want of time. A Brahmin N. D., aged 40, a head constable, was admitted for symptoms of diabetes. His weight was 109½ lbs. sugar in the urine varied from 1,700 grains to 2,400 grains daily. After one day's starvation, sugar disappeared, and remained absent afterwards.

TREATMENT OF DIABETES BY INSULIN.

It has been well established that diabetes is associated with a deficiency of the internal secretion of the pancreas in a large number of cases. In a certain number of cases the deficiency may result later on in the disease. It has been proved that this internal secretion is produced by the islets of Langerhans. It is also known, that want of this secretion prevents the sugar being stored and utilised or burnt up fully in the tissues, so that sugar accumulates in the blood and then passes in the urine. Scientists have been trying to isolate this internal secretion of the pancreas from the other substances contained in this organ and recently Banting in America has been successful in doing it. He has isolated this internal secretion from the pan-

creas of animals. This substance which is an extract of the islets of Langerhans is known as insulin. The exact chemical nature of this substance is not understood but it is probably a mixture of several substances and not a single substance. As regards the method of preparation of insulin, we need not trouble ourselves, but leave the matter in the hands of the chemists. Banting found by experiment that the administration of insulin reduced the blood sugar in animals and when a large dose was administered, the animal succumbed to its effects, that is, it died of *hypo-glycæmia*. Now at this stage I want to explain this term. It means that the blood sugar of the animal is reduced below its normal level. When the blood sugar is above the normal level, as it always occurs in diabetes or soon after the ingestion of food in normal individuals, the condition is known as *hyper-glycæmia*. We know that the normal blood sugar is about 0.1 p.c. Therefore a blood sugar of say 0.14 is hyper-glycæmia, whereas say 0.05 or 0.06 is hypo-glycæmia. So then, if a large dose of insulin is given to a man or animal, death will occur from the sudden reduction of blood sugar, i.e., hypo-glycæmia. The following are the symptoms of this condition; hot flushes in the face, weakness of limbs, giddiness, excessive sweating, tremors, coolness of extremities, a fear of some impending disaster.

EFFECT OF INSULIN ON DIABETES.

Insulin reduces the sugar in the urine and gradually makes the sugar disappear. It also reduces the high blood sugar or hyper-glycæmia which is always present in a diabetic. It removes the ketone bodies from urine: Therefore the patient's condition improves rapidly. He puts on weight, feels stronger and brighter. The increase

in strength and energy produced by insulin treatment is very marked. Many diabetics who were given up for lost, have recovered to such an extent as to resume their work and earn their livelihood. It should be noted that insulin will not effect a permanent cure of diabetes.

CHOICE OF A CASE FOR INSULIN TREATMENT.

First of all we should make sure that the case is one of real diabetes and not glycosuria such as renal glycosuria. If insulin is given in the latter case, the consequences will be disastrous. The blood sugar which is not high in this condition will suddenly fall and the result is hypo-glycæmia. To find out whether a case is real diabetes we can either do a blood sugar estimation or do a 'sugar tolerance test.' With this test, in most of the cases of renal glycosuria, no sugar passes at the end of 3 hours after giving 50 grms. of sugar.

*The following are the conditions in which insulin is indicated :—*1. If the patient is young and the disease is running a very rapid course.

2. If the urine contains ketone bodies in large amount and if coma is threatening.

3. If coma has actually supervened.

4. If the person has developed complications such as carbuncles, boils, cellulitis, gangrene, etc.

5. If dietetic treatment has been tried and found unsuccessful.

HOW TO TREAT A CASE WITH INSULIN ?

Before the treatment is begun, the patient should be asked to stay at home for a few days—4 to 5 days. A record of 24 hours urine should be kept and the quantity of sugar passed daily should be estimated. Test for ketone bodies is made and the blood sugar is recorded.

We next decide what diet to give him. It is very important to regulate the diet during the insulin treatment. Otherwise the treatment will not benefit the patient. We should give him the *minimum amount of diet* required for his health or body metabolism. I have already stated that the minimum diet is calculated at the rate of 10 to 15 calories per lb. of body weight. That is, if the person weighs 120 lbs., he should get sufficient food, equivalent to 120×10 to 120×15 calories, *i.e.*, 1,200 to 1,800 calories. From the diet tables we should work up a suitable diet which would be equivalent to 1,200 to 1,800 calories. Throughout the course of insulin treatment, he should have only so much and nothing more. But if he is losing weight, we may increase the diet, though with this increased diet, he may pass some sugar in the urine.

Insulin should be administered only by hypodermic injection in ordinary cases and not by mouth. It does not act if given by mouth. The injection should be given $\frac{1}{2}$ hour before the two principal meals of the day, *i.e.*, once in the morning and once in the evening. It is better to begin with 5 units and go on increasing the dose by 2 or 3 units every 2 or 3 days. We may go up to a maximum dose of 30 units, twice a day. If this does not remove the sugar from the urine, the diet should be altered in such a way as to reduce the carbohydrate in the food. *For ordinary cases 15 to 20 minims will suffice as a maximum dose.* When we arrive at a dose which keeps the urine sugar free and also the blood sugar normal, this dose may be continued. But how long we are to go on with the treatment? Well, in severe cases the injections will have to be kept up daily for an indefinite period. If at any time in the course of the treatment, the patient begins to get hypo-glycæmia,

he should take 1 to 2 ounces of sugar immediately. If this is done, the symptoms disappear at once. During the course of treatment with insulin, the patient should always have 1 or 2 ounces of sugar by his side, so that he may take it as soon as untoward symptoms appear.

CAN INSULIN BE GIVEN WITHOUT BLOOD SUGAR EXAMINATIONS ?

It is not always possible to conduct blood sugar examinations. Facilities for this may not be available for most practitioners. Moreover they may not have the special training required for the purpose, at least to begin with. Under these circumstances, is insulin, which is such a valuable remedy, to be withheld, especially when it is a question of life and death for the patient ? *The answer is 'no'.* MacLean says that it is quite possible to administer the drug without blood sugar estimations, if we follow the following instructions :—

(i) In ordinary uncomplicated but severe cases, begin with a small dose say 5 units and go on increasing the dose by 2 or 3 units once in 2 or 3 days, the injections being given twice daily $\frac{1}{2}$ hour before food.

(ii) A 24 hour sample of urine should be examined daily for sugar and ketone bodies. As long as there is just a trace of sugar present, we are safe and we know that the patient is not having hypo-glycæmia.

(iii) If the urine is sugar-free, do not increase the dose of insulin but give the same dose.

(iv) Under such careful treatment, hypo-glycæmia is very rare. If the symptoms do appear, it is quite an easy matter to deal with them by giving an ounce or two of sugar.

DIABETIC COMA.

I feel that I should make a special reference to this dreaded complication, regarding the views that are held about its causation and its modern treatment. The most commonly held view about the causation of coma, is that it is an acid intoxication, that is, the acid bodies or the ketones to which I have already referred, namely diacetic acid and acetone, accumulate in the blood as a result of which poisonous symptoms are produced. This view is held by the English and American doctors, and is probably true as regards diabetic coma that occurs in the severe cases in western countries. As regards the Indian cases, McCay and others who investigated 400 to 500 cases in Bengal in 1919 in connection with the Indian research Fund Association, have come to the conclusion that diabetic coma as it occurs in India, *is not an acidosis or acid intoxication, but is due to accumulation of nitrogenous waste products in the blood.* In other words it is a kind of *uræmic coma*. They say that the blood picture of diabetic coma resembles exactly that of uræmic coma which is the result of kidney disease. They have observed that the kidneys get affected very early in most of the cases of Indian diabetes as is shown by the presence of albumin and in cases which develop coma, the kidney function fails, leading to a high concentration of nitrogenous waste products such as urea in the blood.

This view is being recently expressed even by English authorities. Payne and Poulton writing about the treatment of diabetic coma in the issue of *Lancet* dated September 26, 1925, state as follows :—“ The failure of renal function seems to be common in diabetic coma, as shown by oliguria, albuminuria, urea retention in the blood and

failure to excrete ketone bodies and occasionally even sugar. In some cases renal failure is so decided as to cause a marked resemblance to *uræmic coma*. Out of 9 cases examined by us, in 6 oliguria was present, in 8 albuminuria. Out of 7 cases examined for blood urea, 6 showed abnormally *high blood urea concentration*."

Regarding the *diacetic acid and acetone*, the Bengal investigators found these only in a very small number of Indian cases. Lt.-Col. Waters does not agree with this observation regarding the presence of these ketone bodies in Indian cases.

TREATMENT OF DIABETIC COMA.

The treatment of diabetic coma hitherto adopted consisted chiefly of elimination of the poison by introducing as much fluid as possible into the circulation, either by mouth or through the subcutaneous route or through the veins, and neutralising the excess of acid in the tissues by administering *sodii bicarb*, and also by starving the patient. No drug that could influence the disordered metabolism was available. So cases of diabetic coma never recovered. In fact the onset of coma meant impending death.

But now the conditions have entirely changed. In *insulin* we have a powerful drug which has a marked influence over the defective metabolic processes that prevail in coma. Insulin has proved much more valuable in treating cases of coma than in treating cases of ordinary uncomplicated diabetis. It is in diabetic coma that "the most brilliant results" and "certainly the most spectacular effects of treatment with insulin" are noticeable. Patients who are completely unconscious and on the point of death have recovered quickly after being treated with *insulin*.

As regards the details of the treatment, in actual cases of coma, we should give 20 units of *insulin* intravenously at once. Smaller doses are useless. Wait for an hour and if there is no improvement give a second dose of 20 units intravenously. Further injections are given at intervals of 1 to 3 hours *subcutaneously*, according to the improvement that follows. When the case is not so urgent or when the patient is not actually comatose, subcutaneous injections only are enough from the commencement. Give plenty of fluid by mouth or if this is not possible on account of the unconscious state of the patient, give saline rectally or subcutaneously or even intravenously. Weak coffee and tea can be given in plenty. Stimulants such as camphor and strychnine can be given alternately every hour or so, if heart shows signs of failing. Bowels should be opened by enemas or purgatives such as *croton oil*. *Do not give any food* by mouth till improvement sets in. When the patient is better, keep him on a low diet and continue to give as much *insulin* as you think necessary. 10 to 20 units twice daily will be probably enough.

Cambridge and several other authorities believe in the administration of glucose 5 p.c. solution intravenously, side by side with *insulin*, in cases of coma to avoid the danger of hypo-glycæmia. A pint or more of this glucose solution can be given at a time every 6 hours. This appears to be a safe procedure. But MacLean objects to this method of treatment. He does not see the rationale of giving glucose in a case in which there is already hyper-glycæmia.

As regards the question of giving alkalies in coma, all are practically agreed that *sodii bicarb* should not be

given intravenously in coma. MacLean says, "This drug appears only to accelerate the fatal issue. Intravenous injection of alkali seems not only useless but dangerous, and sometimes sudden death takes place."

Joslin, one of the greatest authorities, says as follows:—"The dangers attendant upon the use of alkalis in the treatment of acid intoxication far outweigh their advantages.....I believe that often, a patient threatened with diabetic coma is sent into actual coma by the careless administration of alkalies.....The results obtained since the routine administration of alkali has been abandoned, have been so satisfactory that I shall not willingly return to their employment."

So this seems to be the prevailing opinion regarding the administration of sodii bicarb intravenously in cases of coma. As regards giving it by mouth in severe cases for diabetes uncomplicated with coma, there appears to be difference of opinion. MacLean thinks that it is very useful in some cases when given by mouth. But Joslin will not have even this. I shall quote him again:—"One must bear in mind that it is possible that the administration of small quantities of alkalies over long periods may set free acid bodies existing combined quiescent and harmless in the body and thus do harm. Furthermore, the administration of alkalies over long periods may deplete the body salts such as chlorides which are distinctly useful.Finally the constant use of an alkali appears to promote the constant excretion of acid bodies. I have known a moderate acidosis of a month's duration to vanish with the omission of soda".

Conclusions. (1) Diabetes is not a disorder of carbohydrate metabolism alone as was hitherto thought; but it

is a disturbance of the general metabolism of the body in which all the three kinds of foodstuffs are involved.

(2) As regards the ætiology, the pancreas plays an important part, there being always either a primary or a secondary affection of this gland. The actual causation of the disease is still obscure, though much new light has been thrown recently as regards its pathology.

(3) The presence of ketones in the blood and urine is considered to be a more important factor as regards the prognosis of the disease than the presence of sugar itself.

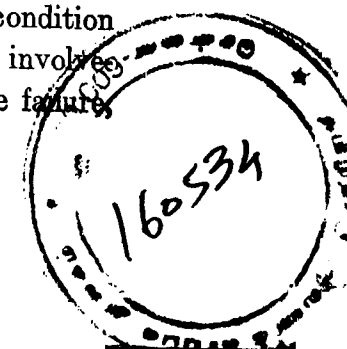
(4) There are different kinds of glycosurias. Great harm is done to a patient by diagnosing a harmless glycosuria as diabetes. By means of sugar tolerance tests and blood sugar estimation, simple glycosurias can be diagnosed from diabetes proper.

(5) Ordinary cases can be successfully treated by Allen's starvation treatment. For the mild form of diabetes which prevails in India, this method is very suited and successful.

(6) If the dietic treatment fails or when complications such as carbuncles, abscesses, coma etc. set in, insulin should be employed and gives very good results especially in coma.

(7) Insulin can be used best with blood sugar examinations conducted from time to time to watch the effects of treatment.

(8) Regarding the causation of coma, ideas are changing. McCay and others hold that in Indian cases there is no acidosis at all, but there is a uræmic condition of the blood present. Importance of the early involvement of the kidneys in diabetes and their ultimate failure,



has not been hitherto properly observed. But recently European observers are realising this.

(9) Regarding the treatment of coma, insulin has brought on a great revolution. Marvellous results have been obtained in this condition. The treatment of coma with alkalies is not considered to be rational and is being abandoned.

CERTAIN COMPLICATIONS OF PREGNANCY*

(By DR. P. R. VENKATARAMA AIYAR AVL., M.B. & C.M.)

ANTEPARTUM HÆMORRHAGE.

There are two varieties, the accidental and the unavoidable. The nomenclature sufficiently indicates what they are. In the former bleeding takes place from the separation of a placenta normally situated and in the latter from the low implantation of the placenta in an area which forms the chief dilating passage in labour, and which takes place as a matter of consequence with each contraction of the uterus, dilating the cervix. The placenta is attached wholly or in part to the lower uterine segment which is clinically that part of the uterine cavity which dilates to permit the passage of the child. Anatomically this area spreads for a distance upwards of about three inches along the wall of the internal os and almost corresponds to the area of the uterus where the peritoneum is loosely attached and where muscle bundles are arranged longitudinally. Antepartum hæmorrhage is more frequent in Multiparæ and multiple pregnancies.

Frequency.—Guy's hospital gives 1 in 534; Rotunda 1 in 227; Glasgow 1 in 391; Tanjore 1 in 40. The

* A paper read before the Tanjore District Medical Association.

abnormally high figure in Tanjore is probably because that almost all the cases which come, do so for some abnormality.

Ætiology.—That accidental hæmorrhage is due to some accidental cause as shock, fright, and trauma has been handed over to us from posterity. A view of recent origin is that Toxæmia especially albuminuria of pregnancy plays an important role in its production. This view in my opinion cannot be accepted without discussion for if it was due to Toxæmia all cases which disclosed albumin in the urine together with all the signs and symptoms of the defective function of the Kidneys and all cases of eclampsia, should develop hæmorrhage and my experience seems to negative such a view as no such case developed accidental hæmorrhage. Further, albumin was not detectable in the very few accidental hæmorrhage cases we have had. One won't be far from right when one says that the diseased condition of the uterine wall is responsible for the accidental hæmorrhage and placenta prævia and a condition of Syphilis in the mother as a likely cause cannot be denied without hesitation. The view I hold is that the unhealthy condition of the uterine endometrium is a responsible factor in the production of accidental and unavoidable hæmorrhage. It can be well illustrated if an analogy is made between the fertilised ovum and the uterine endometrium, on the one hand, to a traveller and a rest house on the other. Just as admission is refused by the manager of the rest house to a traveller who frequently seeks admission, lest he should be a waste on the finances of the rest house, similarly the uterus refuses admission to a fertilised ovum when it is in an unhealthy condition in order that this pregnancy will not

be a waste on the life of the woman, especially when the soil is unsuitable. Its noticeability in multiparæ shows the refusal of the uterus to be the undesirability on the part of nature to avoid pregnancy when the mother is enfeebled on account of frequency of child-birth, and that she should not now be subjected to pregnancy as her health does not admit of such a condition when the uterine endometrium is also diseased.

The ovum, the fertilised ovum, like a vicious traveller forgetting the previous charity of the endometrium to the previous ova, acquires acquaintance along its passage out from the fundus, somewhere near the cervix and shows its vengeance to the uterus by subjecting it to chances of pregnancy and bringing about the disastrous condition of placenta prævia. This is an instance where nature's aid in repulsing pregnancy out of its usual site in the interests of the mother is overcome by the destiny of the mother who becomes subject to placenta prævia, mild or serious as the case may be, thereby proving the biblical saying "In sorrow, if I can add, and in misery, shall thou bring forth." The unhealthy condition of the uterus, the debility consequent on child birth and the unique loss of the uterine tone incidental to repeated pregnancies all combine to form adverse conditions and are analogous to a financially rich feeding house, which has become poorer day by day by feeding travellers. If pregnancy is considered as a harmonious, homogenous symbiosis in which the mother and the unborn infant reciprocate their help, the former supplying from her natural resources much that the latter is in need of, the latter stimulating the maternal tissues and functioning them to greater activity so that their stock of resources is made good, if wide acceptance can be

given to this view, then to me it seems that the previous born infant, the paying guest, had failed to do its duty as a result of which the host, the mother, is loath to give a lodging to the present ovum and this is expressed by the flat refusal of admission in the fundus of the uterus, its usual site, and the ovum in its turn converts its well intentioned hospitality into acts of vengeance and implanting low near the cervix subjects the mother to the misery and trouble of the placenta prævia. With the same analogy, accidental hæmorrhage can be explained in that the unwelcome guest having somehow excited the sympathy of the host gains admission to the fundus but is subsequently detected to be an undesirable guest and therefore attempts at expulsion are made by premature separation of the placenta. That placenta prævia is not a recurring affection seems to support the view that only at the particular period, the uterine endometrium was not in a fit state to receive the ovum. The hæmorrhage in both the conditions takes place from the torn utero-placental vessels. Some consider that bleeding in placenta prævia is an accidental hæmorrhage to placenta prævia by shocks, and jars and that the detachment of the placenta results from the uterus growing away from the placenta or the placenta growing away from the uterus. These are no doubt views and explanations of an ingenious nature.

VARIETIES.

(a) *Accidental*—(1) *Open*, (2) *concealed*, (3) *mixed*.

(b) *Placenta Prævia* 1. Lateral, 2. marginal, and 3. central. Clinically the variety in placenta prævia depends upon the nearest edge of the placenta to the internal os whatever may be the dilatation of the cervix.

SYMPTOMS.

The one common symptom in placenta prævia and the accidental, save in the concealed, is external bleeding. The loss of blood may be slight or profuse and alarming, anæmia and collapse may be greater in accidental than in placenta prævia, owing to a sudden and extensive separation of the placenta. Sudden and severe hæmorrhage in the later weeks of pregnancy (last six weeks) without an adequate cause, taking place while lying in bed or asleep or on slight exertion such as dressing or walking should warrant an undoubted diagnosis of accidental or unavoidable hæmorrhage. The recurrence of the bleeding is in favour of placenta prævia and the severity of bleeding in placenta prævia depends upon the extent of overlapping of the placenta over the os. Although the initial hæmorrhage may be slight in placenta prævia, the subsequent hæmorrhage may be a menace to life. The severity of the hæmorrhage is also governed by the duration of pregnancy; the greater, the loss, the nearer is the pregnancy to full term. In view of this characteristic recurrence of bleeding in placenta prævia, failure to take steps to prevent a second bleeding may be a sin of omission of a fatal nature.

In two-thirds of the cases, premature labour sets in and only a third reach full term. The statement that abnormal presentations as pelvic and transverse are noticeable in placenta prævia could not be justified by my experience in this hospital. Almost all the cases were of head presentation. Inertia of the uterus in the second and third stages due to feebleness of the mother and a rigid cervix may be formidable foes in the management and tax our resources. In concealed accidental hæmorrhage

signs of internal bleeding such as faintness, rapid, feeble pulse, vomiting, severe abdominal pain from over-distention of the uterus the pain being of a tearing nature, rapid development of extreme pallor and collapse (some women dying undelivered), tense tender and wooden feel of the uterus, absence of uterine contractions, absence of foetal parts, inaudibility of the foetal heart sounds, all these will disclose the sad tale of concealed accidental; and in the mixed variety of accidental, shock and collapse out of all proportion to external bleeding will give a clue.

DIAGNOSIS.

Placenta prævia must be diagnosed from accidental hæmorrhage. A sure diagnosis can be made when the placenta can be felt; and when the placenta is felt, the statement that it can be mistaken for a blood clot is to underestimate the diagnostic dexterity and feelings of the observer. However, the firmness of the placenta and its continuity with the membranes will give a correct inkling. In cases where the placenta could not be felt on account of the closure of cervix a very probable diagnosis of the placenta prævia can be made, if the presenting part is head and this is not engaged and is freely moveable in a case where the woman is bleeding in the last weeks of pregnancy. Help might be available in the detection of albumin in the urine, pointing to the condition being one of accidental. In all cases in the later months of pregnancy a correct and early diagnosis should be made with the least possible delay, and the absence of dilatation of the cervix cannot be considered to be a reasonable argument for failure to diagnose the condition; and facilities for diagnosing the condition are available in metallic dilators with which the cervix may be dilated, and the condition

can be diagnosed. After having correctly identified the condition whether hæmorrhage is due to placenta prævia or accidental, there are yet other conditions resulting from symptoms of shock or from symptoms of hæmorrhage which might mimic prævia or accidental.

And these are :—(a) simulating conditions presenting symptoms of shock.

- (i) Fulminating appendicitis.
- (ii) Perforation of a gastro-duodenal ulcer.
- (iii) Acute intestinal obstruction.
- (iv) Rupture of ovaries.
- (v) Tortion of Myoma or ovarian cyst.

(b) Simulating conditions presenting symptoms of hæmorrhage.

- (i) Rupture of abdominal aneurism.
- (ii) Rupture of the uterus.
- (iii) Intra-peritoneal hæmorrhage.
- (iv) Rupture of tubal gestation.
- (v) Thrombosis, of mesenteric vessels.

In concealed accidental which might simulate some of the above conditions the decisive factor in diagnosis is that the signs centre round the uterus and, elsewhere in the abdomen, nothing is discovered.

PROGNOSIS.

No language can suitably embody the seriousness of Ante-partum hæmorrhage and in the light of existing conditions and want of dissemination of knowledge among the lay public about the gravity of the cases where bleeding takes place before the birth of the baby, who are apt to regard the condition as one of the woman being possessed and that incantations and charms alone will remedy, the prognosis cannot be anything favourable or commendable.

Despite the best help and skill in attendance the woman might succumb simply because she was allowed to loose more blood than what her system could allow or tolerate, to admit of interference by way of management. I can dogmatically dictate that the chance of a woman falling a victim to this dire condition will be few and far between if the management of the case is undertaken at the first loss without losing any more time. The experience in my private cases justifies this view. Cases are brought to the hospital in extremis and at times moribund, when interference will only prove to be the proverbial last straw. The maternal mortality according to well equipped hospitals is about 4 per cent. Here it lamentably works about to a percentage of twenty. This twenty per cent is too high a record and it can still further be reduced if the public are made to understand the importance of early transporting the patients to the hospital at the very first loss of blood or summoning medical aid promptly when there is every reason to be sanguine of attaining a lowered mortality rate. Very often, cases are brought in a condition of collapse and after several days of bleeding and it is high time that we should see that the treatment of cases is not postponed to a dangerous delay. The foetal mortality is also equally horrible. In many cases the death of the child is an inevitable inference; partly because the pregnancy is not sufficiently advanced in some cases and partly because the separation of the placenta causing profuse bleeding would have caused the death of the child. The profession should also realise the necessity of earlier recognition of cases of ante-partum hæmorrhages and if facilities are not available for efficient management at home cases must be with all rapidity transported to a well-

equipped hospital. This is not always done and the treatment is carried on in the house until the situation becomes well nigh uncontrollable. The prognosis to some extent depends upon the variety of ante-partum hæmorrhage and in concealed accidental and central placenta prævia, the two worst varieties of ante-partum hæmorrhage, the prognosis is invariably most grave.

TREATMENT.

Accidental.—In slight cases treatment is more or less akin to a case of abortion; rest in bed, sedative treatment and treatment of toxæmia, if any; in moderate cases, vaginal plug, Puncture of the membranes, tight abdominal binder and injection of $\frac{1}{2}$ to 1 c.c. of Pituitrin; a method of delivery suitable according to the condition of the os, as Forceps, Version or perforation. The success of the treatment depends upon the co-operation of the uterus in each manœuvre. In severe cases and especially in the concealed variety, cesarian hysterectomy and not cesarian section has to be considered.

Placenta prævia.—In the management of a case of placenta prævia the first point to be remembered is that the lower uterine segment where the placenta is implanted does not possess that rare but commendable commodity of retraction and contraction, as the fundus of the uterus has. The governing factors that should preliminarily be surveyed in one's mind are :—

- (1) The variety and the amount of,
- (2) The duration of the pregnancy,
- (3) The question of the mother's life,
- (4) Life of the child,
- (5) Surroundings of the patient and
- (6) Facilities available.

The main consideration no doubt in a case of serious hæmorrhage is to effect an immediate arrest of the bleeding and the efficiency of any conceivable method of controlling the bleeding depends upon how far and how well you will be able to secure good pressure on the bleeding vessels. And this might be gained by (1) the presenting part, (2) the bag and (3) the vaginal plug; and this pressure may also be maintained after rupture of the membranes by uterine contractions and by presenting part. This is a rare complication in pregnancy that nobody can lay claim to a wide experience. A point of considerable moment is to save the life of the mother by a sure method and the result of the treatment can be said to be satisfactory if this is achieved, as the death of the child is, in many cases, an anticipated event. Therefore it stands to reason and judgment that in every case the life of the mother is the predominant feature to be considered and no treatment can be countenanced or tolerated which reduces the chances of the mother. Everything else must be subordinated to the interests of the mother, and any treatment which ensures and guarantees the safety of the mother must be given the foremost consideration without fear of any discordant view. A noteworthy feature in placenta prævia is that the hæmorrhage is of a recurring nature which gives a clue, in trying to direct us along the proper channel of treatment. Therefore the method of temporising save in a few exceptional cases can be pronounced to be the worst crime conceivable. A dictum which always should find a place in one's memory in the treatment of these ante-partum hæmorrhages is the law forbidding rapid delivery; as to expedite delivery in a woman who is exsanguined is to whip a tired horse. A condition of shock is

superimposed and the woman dies with dramatic and horrible suddenness. Rapidity of delivery is unwarranted unless the method you have adopted for arresting the bleeding is found ineffective. If repetition is given any credit, I might profitably lay emphatic stress on the condemnation and disapproval of any method which aims at the possible reduction of foetal mortality at the risk of raising the maternal mortality. This is the real foundation for the framework of the management of placenta prævia and when one calmly surveys the statistics of children born in placenta prævia he inevitably finds that a large percentage of children born according to certain methods though for statistical purposes may be considered alive, must really be considered dead, as they invariably succumb within the first few days of their infant life. The welfare of the patient is in no way promoted nor is it in concord with the canons of established obstetric practice, by temporising. The watchwords in the treatment of placenta prævia which comprise in a small compass the result of vast obstetrical experience are—

- (1) Do not temporise.
- (2) Recognise complications early.
- (3) Arrest bleeding by the most efficient method.
- (4) Avoid rapid delivery.
- (5) Observe absolute Asepsis and Antisepsis.

To send a patient to the hospital or to call in the aid of a consultant after the woman has bled profusely for several days is almost akin to the practice of seeing the stable closed after the horse has escaped. The woman under these circumstances is collapsed and in extremis and any

method which obstetrics can improvise or devise for her will only invoke and court instantaneous death although such measures are only of a very conservative nature. Placenta prævia must be viewed in some cases as a surgical emergency, and instances will not be wanting where the initial blood loss might be so violent that the preservation of the life of the mother forms the only consideration. The patient must be treated on the very spot and any attempt at removal might at times terminate tragically. The point that the patient is not only unsafe until the uterus is emptied, but also after, as the trouble does not end and may result in an uncontrollable post-partum hæmorrhage, should be well brought home to those who undertake the management of a case of placenta prævia. An additional catastrophe awaiting such women in the form of embolism during puerperium should be well borne in mind, and the patient should be forbidden to sit up in bed for several days after her delivery.

With the above general remarks let us survey the various established methods for successfully combating a case of placenta prævia. The use of any method depends upon the condition of the cervix. Efficient vaginal plugging with a small dose of morphia to lessen the shock incidental to such a procedure is the ideal treatment where the os is completely closed; although in such cases dilatation of the cervix with metallic dilators might be done to admit of the second procedure; (2) Os admitting two fingers which will invariably be the case where the bleeding is fairly profuse, allows us a choice between bipolar version and champetierede-ribes bag. Variety and contrariety of views and violent disagreement prevail in the selection of any one of the two methods. The appalling foetal mortality

associated with bipolar version naturally raises the question if that can be successful as the standard method. Statistics under the two operations might be helpful.

			Maternal mortality.	Fœtal mortality.
Version	..	..	7·8	73·7
Hydrostatic	..	..	6·5	43·4
Dilator	..	..		

A glance at the statistics shows that the Hydro-static dilator has not much in favour of the mother as compared with version. But the foetal mortality appreciably reduced by nearly one half under this method seems to suggest whether this method cannot be adopted in preference to version from the point of view of both the mother and the child. This is an illusory scene thrown to us. If we rightly understand that many of the children are *only* live still-births and do not survive the first few days of the infant's life; and in view of the disadvantages that inevitably are associated with the use of champetier-de-ribess bag such as the necessity for a special appliance, the unwholesome condition of the bag and that aid by forceps or version is needed, the moment the bag is expelled; and the greater chances of development of sepsis under this method, and in the light of the fact that there is a ready disposal at our hands, of our own hands for doing bi-polar version, the question really arises if version is not an unparalleled rival in the management of placenta prævia. It has stood the test of time and it must be acknowledged that in version we have a never failing friend, ever loyal in the worst emergency and in the worst surroundings of a case of placenta prævia. My advocacy of the use of version

need not be construed in the light of depreciating the use of champetier-de-ribess bag as I am not unconscious of its utility. In certain cases with a certain amount of practice, skill and dexterity and a knowledge of the technique, version might be performed with the conviction and encouraging assurance that one is practising a method which has the acceptance of most eminent obstetricians and which has an unsullied reputation. To put in a nut-shell the treatment of placenta prævia when the Os is two fingers dilated, is bipolar version without extraction with due emphasis on the latter, under a rigid aseptic ritual. I need not dwell here upon the art of performing bi-polar version. But the points that merit attraction in the performance of version are:—1. Ascertain the back of the child and push the child's head in the direction of the back. 2. Recognise the tuberosity of the tibia. 3. Catch hold of any foot. 4. Don't try to introduce more than two fingers. 5. Use cervix or bullet forceps to pull the foot out. 6. Don't attempt at immediate delivery after version. 7. After having brought down the half breech, to act as an efficient plug on the placenta, restore the patient's condition and fortify yourself to meet a serious post-partum hæmorrhage. A greater freedom is given in the management of the Os more than two fingers dilated. Internal version and application of forceps added by perforation in head presentation, when fixed might with advantage be done. In all these manœuvres the risk of laceration of cervix is a very genuine one and unless one takes extra precautions the occurrence of the catastrophe is inevitable and a traumatic post-partum hæmorrhage will result.

The treatment of placenta prævia cannot be said to be complete, unless SECTION is given the consideration

that it deserves. Of late no subject has excited greater divergence of views and keenness in controversy and disagreement in its utility than the choice of this method for placenta-prævia. I had no occasion to think of doing Cæsarean section for a case of placenta prævia; for cases, on the one hand, were admitted either in such a condition that the mere thought of Cæsarean section can be regarded as a crime, and on the other hand the condition of some cases did not warrant such a procedure; and they might be and as a matter of fact, were dealt with under other methods, that I came across, no suitable case, for Cæsarean section. The object of Cæsarean section though purely to save the woman from this dire condition has also behind it a further mission in reducing the foetal mortality which is noticeable to a marked extent under the hackneyed methods of the treatment of placenta prævia. In this connection the motto "A bird in the hand is worth two in the bush" may be one's guide before one launches the surgical boat upon the stormy sea of Cæsarean section. No method could be considered commendable if it tries to achieve a lowered death rate of the foetus at the increased risk of the mother. Cæsarean section therefore may be regarded as an operation limited in its application in the treatment of placenta prævia and the propriety of performing the operation only in cases of central placenta prævia when the mother's condition allows such a procedure and where the child is probably alive can in no way be impeached. It sounds irrational to think of doing Cæsarean section before the 36th week of pregnancy as the chances for the child are minimised. The objection to the Cæsarean section that the scar is liable to rupture can be easily met by a rigid adherence to the canons of asepsis.

STATISTICS.

Operation	Author	Maternal mortality.	Foetal mortality.
Cæsarian section .	Muhro-Kerr	11%	22%
Do. ..	Doderlin ..	8.9%	30
Do. ..	Hospitals in Gr. Britain .	6.6%	24
Version ..	Doderlin ..	7.8%	{73.7
Do. ..	Hospital in Gr. Britain	8.6%	95.3

Cæsarean section though considered and acknowledged as an operation of choice in central placenta prævia where the maternal and foetal mortality is thrice as much as in the other varieties, is distinctly contra-indicated, when the mother is in an exsanguined condition and the foetus is dying or dead. In the milder varieties of placenta prævia the results achieved by other methods as far as the mother is considered are not unfavourable and with a questionable chance of the child even if delivered alive, it taxes our thoughtful consideration regarding the propriety of performing cæsarean section and it rather prompts us to announce that the cæsarean section has no place under those circumstances.

HEART DISEASE

It is a never failing source of anxiety to a medical man in charge of a case of pregnancy which is complicated by valvular disease of the heart. Cases vary in their seriousness, the heart being damaged and diseased to an extent and degree not far from breaking down, and when such heart cases become the subject of pregnancy it is impossible to conceive that there will be any relief, partial or complete,

of the anxiety of the medical attendant. In the light of modern knowledge about the function of the heart in normal and abnormal conditions, the prognosis of a case of heart disease has not so much relation to the particular valvular lesions as it has to the existence or otherwise of compensation. Greater recognition and appreciation is given to the functional activity and integrity of the cardiac musculature than to the impairment of the valves. And this knowledge unfolds that the issue in cases of heart disease is an individual one and not a general one. However there are certain elements which exert an adverse influence in pregnancy on the heart muscle. And they are, obesity, anæmia, exhausting work and lack of compensation. The varieties of heart disease that might complicate pregnancy are.—

1. Acute endocarditis,
2. (a) Chronic valvular diseases,
 - (b) Mitral insufficiency,
 - (c) Mitral stenosis,
 - (d) Combination of *b* and *c*,
 - (e) Aortic regurgitation ; Aortic stenosis,
 - (f) Uncertain lesions,
 - (g) Myo-carditis.

GENERAL CLINICAL FEATURES

In diagnosing a case of heart disease in pregnancy it must be well remembered that certain murmurs such as rough blowing murmurs to the left of and over the sternum, (a systolic murmur of slight intensity at base and apex) are incidental to pregnancy. As a matter of fact only a seventh of heart cases are diagnosed, many passing without attracting the attention of the patient or the physician. The orthodox view is that mitral-stenosis is a very grave

complication in pregnancy. Many pregnant women pass through the pregnancy without actually becoming conscious of the existence of the damaged heart. It may escape detection if no routine examination is made. The non-existence of inefficiency of the heart muscle before pregnancy as a rule allows a woman to pass through pregnancy and labour unscathed. The optimistic attitude we can assume regarding her, in pregnancy and after is solely dependant upon how far she will be able to fortify herself with all her forces. The scale will oscillate in her favour if she can exercise proper control about her diet, rest and sleep. To many pregnant women the environments social and domestic are antagonistic to the attainment of such an end. Insufficient nourishment, exhausting work, want of sufficient rest and sleep have to be faced by many ; and it is no wonder if compensation breaks down. Sir James Mackenzie who takes a gloomy view reports six changes in circulatory conditions as invariably associated with pregnant condition and they are :

1. Restricted field of cardiac response occurring from the early period after the first and second month and much earlier before the growth of ovum inside the uterus calls for increased blood supply.
2. Changes in rate and rhythm ; these are not only increased on exertion but unusual variation more particularly influenced by respiratory movements. Occurrence of such irregularities as extra-systoles of ventricle and auricles.
3. Dilatation of the right side of the heart.
4. Tendency to Oedema of the lungs.
5. Tendency to over-filling the veins of the lungs.
6. Marked pulsation in the veins of the neck.

It is a matter of common knowledge that the onset of dyspnoea and the appearance of Oedema in a heart case calls for a thorough revision of the tests of the heart's efficiency and a proper course of treatment should be imperatively instituted if the evidence is unfavourable. The response successfully or otherwise to the treatment will be a criterion for allowing or interrupting pregnancy. It cannot be again said that pregnancy exerts an adverse influence on a woman whose heart efficiency is questionable. In the latter months of pregnancy the heart has to labour under displaced conditions and under conditions of increased blood pressure; and this naturally summons into being exaggerated heart-rate. There is also the additional impediment to the circulation of lower limbs and return of venous blood and under these circumstances it is a surprise why all the heart cases do not fail. Even if worst conditions set in it is not impossible to re-establish efficiency as evidenced hereby.—

1. One case of mitral-stenosis which showed failure of compensation at the time of labour but passed through the labour successfully.

2. By another case of mitral-stenosis showing signs of failure of compensation to an intense degree ending in premature labour at the 7th month uninjured under a violent orthodox treatment of digitalis orally and hypodermically which I consider a very good weapon in establishing not only satisfactory compensation but also in bringing about the desideratum of premature labour in those cases. I consider that digitalis is a potent drug and can with advantage be utilised as a method for inducing premature labour. This opinion, I base, on the experience gained by me in treating some of these heart

cases and cases of Anæmia where acute dilatation of the heart was an outstanding feature. I can record here another case of mitral stenosis admitted into the hospital with failure of compensation at the seventh month returning home with compensation fully established and coming back at nearly full term, with complete failure of compensation to undergo labour with compensation established and to pass through puerperium without serious troubles arising in the same. In the name of the fear of compensation, I must narrate to your surprise that a case of double mitral with compensation failing at no time in pregnancy passed through labour successfully and puerperium uneventfully. The first case mentioned by me sought admission while in labour with the heart condition at the worst and she was not able to lie down even for delivery and she miraculously escaped the jaws of death. I may not be taken amiss when I say that I should bring home to you the fact that during labour it is the oscillation of the blood pressure that exerts a baneful influence on the damaged heart and it is the most dangerous period when it may truly be said "the sword of Democles hangs heavily on the woman's head." Despite the miraculous survival of some of these cases after labour they are likely to terminate tragically during any day in the puerperium from syncope. The additional dangers in puerperium are endocarditis, embolism, and oedema of the lungs. Post-partum hæmorrhage is a dire complication that might victimise some of these cases.

MANAGEMENT DURING PREGNANCY.

The management of a case where the heart muscle is efficient and the field of cardiac response is in no way abnormal does not differ from a case where there is no

pregnancy. When signs of failure are noticeable the case should be considered and managed as one of cardiac emergency. Rest, Digitalis, Camphor, Valerian, Trinitrini, intravenous injections of strophanthus, oxygen inhalations, paral-de-hyde for inducing sleep, morphia cautiously in certain cases constitute the list of remedies. The dictum or the golden rule, take care of the stomach by at no time subjecting it to heavy food, and the heart will take care of itself, should be well remembered. The alarm or the signal of failure should be promptly responded to by instituting a campaign of suitable treatment in which no detail should be overlooked. Rest, to the hard, working labouring classes, exercise short of fatigue in the well-to-do who having obtained some knowledge of their condition are culpable by taking superfluous rest), such as walking, driving, motoring should be enjoined. Some of the minor symptoms such as heartburn acidity and constipation should receive prompt attention. Induction of labour as a treatment is a disputed one. That the question does not arise except in cases of serious heart failure, there can be no two opinions about, and when the symptoms are serious, it is no doubt doubtful, if any benefit will accrue from this procedure. There can, however, be no disagreement to the view that in cases which do not respond to treatment there is a legitimate demand upon this procedure, notwithstanding the chances of small success.

The periods of main risk are during the oscillations of the blood pressure and during the puerperium. Sir James Mackenzie advises to terminate pregnancy

(1) When there is orthopnoea.

(2) In mitral stenosis with oedema of lungs or with a heart rate over 100, with violent palpitation on the slightest exertion.

(3) In aortic regurgitation with a corrigan pulse or with a distinct forcible apex beat outside the nipple line.

MANAGEMENT OF LABOUR.

The main risk arises from alterations of blood pressure and from fatigue. Post-partum hemorrhage is an added risk. To avert these risks, Rupture of the membranes can with advantage be made, as this procedure puts an earlier limit to the rise of blood pressure. Delivery may be expedited with forceps, when the passages are dilated as this curbs and limits to a great extent the oscillations of blood pressure and removes fatigue. The application of a sand bag weighing about 5 to 7 lb over the abdomen in the second and third stages merits an important consideration, as by its means pressure is maintained over the large vessels and also it prevents the fundus from relaxing. Rectification of abnormal presentations is also another means of avoiding undue exertion and fatigue to the mother and thus must be done as early as possible before the patient goes into labour. The use of anaesthesia and its beneficial effect on a heart case demands a legitimate consideration in these cases, for, by its judicious use the woman might be relieved to a great extent of the sufferings and agony of labour pains which otherwise would act adversely on the mother. Chloroform, aether and hyoscine all have their use in suitable cases.

MANAGEMENT DURING PUERPERIUM.

We cannot enjoy a clear conscience until the puerperium is safely passed through. The effects of physical exertion of labour, changes in the quality of the blood and alterations in the intra-abdominal pressure incidental to the cessation of placental circulation, present

unaccustomed problems to a damaged heart and it won't be a matter of surprise if the heart fails in the puerperium. Our reliance has to be placed on full doses of Digitalis, Camphor, Valerian, the use of sand bag of two to four pounds on the abdomen, regulated exercise in bed and when allowed to get up restricting her exertion to short periods, with a liberal allowance of rest periods; these are the actual collapse averting agencies. The treatment cannot be considered complete until a word is said about lactation. This is an individual one and in cases of severe heart failure it must be interdicted but in milder cases the exertion in nursing must be controlled as far as possible by the child being supported by an additional agent. The question is very often asked whether a heart patient can marry and bear children. The laws of Peter—maid don't marry, wife don't carry, mother don't baby—are now-a-days violated. As a working rule it may be declared that a woman with a heart disease where compensation has been fully established may enjoy the social and matrimonial amenities and face labour without fear as her neighbour with an undamaged heart but those who exhibit signs of frequent failure should be shown the danger flag "avoid pregnancy" Sir James McKenzie enunciates that pregnancy should be forbidden.

1. If the cardiac response is limited and palpitation is readily induced by exertion and no improvement noticed under treatment.
2. Dilatation of the heart and oedema of the lungs
3. Nodal rhythm
4. Mitral stenosis with diastolic murmur
5. Aortic lesions unless the muscle is in good condition and intact

Pregnancy may be allowed

1. When the field of cardiac response is fair and there is no enlargement of the heart
2. If there is good recovery from cardiac failure
3. Mitral stenosis with good efficient muscle.

SUMMARY OF TREATMENT.

(Pregnancy)

1. Have a close observation on the patient
2. Allow pregnancy if there is no failure of compensation
3. Avoid cardiac strain
4. Treat signs of slight failure (oedema, irregular pulse), albuminuria, cardiac dilatation, by rest, cardiac tonics and careful diet
5. Terminate pregnancy if serious signs of failure are present by throwing as little strain as possible on the cardiac muscle
6. Don't interfere with the patient, if seen at or near term as it heralds more danger
7. Improve patient's condition.

LABOUR.

First stage :—Rest in reclining position

Puncture membranes to relieve strain

Use Hyoscine if necessary.

Second stage :—Conduct labour in the reclining position

Shorten labour,

Avoid bearing down efforts,

Anaesthetise and use forceps,

Avoid post partum hæmorrhage and collapse by sand bags and saline,

Cæsarean section under spinal anaesthesia, may be considered.

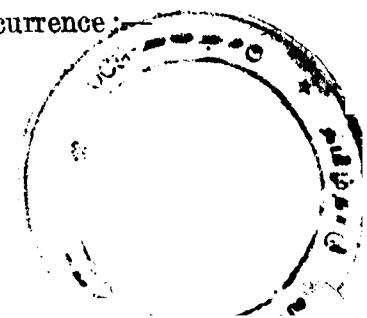
ANAEMIA.

The literature about Anaemias of pregnancy is meagre and not in my view satisfactorily traceable anywhere. It has not in my opinion received enough attention as it should at our hands. I dare say that it will be within the experience of all of us to observe some cases which markedly draw our attention on account of their pallor and impoverished condition of the blood. Whatever may be the underlying cause, the astounding feature and noteworthy character of the patient is intense anaemia. In order to have a comprehensive idea of Anaemia of pregnancy it is desirable and profitable to have a knowledge of the condition during pregnancy. Investigations into this subject have no doubt resulted in keen and violent controversies but it is admitted by all and agreed to by many that Hydroemia is a natural alteration in the blood incident to pregnancy. Our knowledge of the investigation still further unfolds that there is an increase in the total quantity of blood with decreased intensity. The number of red cells shows full range of high and low values and it depends upon the nutrition, and this rule holds good for hæmoglobin as well. A diminution of red cells and hæmoglobin is discernible after confinement. This is only transitory as rapid restoration to normal condition takes place. The white cells especially of the poly nuclear variety are noticeably increased in pregnancy and much more in labour returning to normal condition during the puerperium. Neither variation in the coagulative power nor uniformity of change in sodium chloride content is evidenced. An increase in Cal. Salts and phosphates and a decrease in iron content are features physiological to pregnancy. Additional features notice-

able in pregnancy are excess of fat and Cholesterolin and increase in antitryptic power of the serum returning to normal condition only on the tenth day of the puerperium. Pernicious anaemia is the only variety made mention of in many of the text books. Many cases are not of this type and to me it looks like that the classification of the anaemia as one of physiological and pathological might not be inappropriate. A number of cases showing pallor of a mild degree, slight puffiness of the face and slight oedema of lower extremities, do come out unscathed in labour although when they enter the arena of labour they do so with considerable risk behind, and these cases passing through puerperium also uncomplicated may be rightly labelled as belonging to physiological anaemia. There are yet a noticeable number of cases in whom the symptoms are more pronounced and intense such as marked pallor and puffiness of the face to a great degree, extreme oedema of the upper and lower extremities, oedema of the abdominal wall and in some cases ascites with acute dilatation of the heart and hæmic murmurs. It is this class that must be styled as pathological anaemia. The frontier between physiological and pathological is at times so ill-defined that the physiological might become pathological at any moment. These women are invariably admitted in the first stage of labour and very seldom a few days or weeks before labour. Under these circumstances it is a matter for surprise that the result of labour strikes a funeral note in only a few.

Causes.—An analysis of 23 cases shows that the following are formidable causes of the pathological anaemias and they are arranged in the order of their occurrence:

1. Ankylostomiasis.



2. Defective and damaged function of the kidneys.
3. Latent Syphilis.
4. Malaria.
5. Dysentery.
6. Tuberculosis rarely.

SYMPTOMS.

Symptoms are marked pallor, general anasarca, glossy and shiny skin, puffiness of the face (more marked in the kidney variety and ankylostomiasis and its absence in some syphilitic cases), ascites, dilated heart, rapid pulse, scanty urine, presence of albumin and casts, deficiency in R.B.C. and hæmoglobin, absence of fits, although albumin might be present (which I believe is due to natural Vene section by the hook worms, in ankylostomiasis cases thereby decreasing the B.P.), irregular pyrexia before labour and becoming worse after and lowered blood pressure. The chief dangers these patients are liable to are:

1. Heart failure during pregnancy, labour and puerperium.
2. Post-partum hæmorrhage.
3. Embolism.
4. Sepsis.
5. Intercurrent affections especially pneumonia and diarrhoea.
6. Premature labour ending in death of foetus in some and in some, foetus surviving a few days after delivery.

The particulars of about 23 cases are given below:—

Of those 23, 19 were multiparæ and four primiparæ. Albumin was present in the urine in nine cases. Albumin and casts in five cases; Hæmoglobin ranged from 25 to

50 per cent; and the total number of red corpuscles varied from 1 to 4 millions. Nucleated red-cells were not found; Poikilocytosis in one case. In 13, labour terminated prematurely. In 2, labour was difficult, there were still births of children. Maternal mortality 1 death, and three discharged otherwise about dying. In two, Wassermann reaction was positive.

TREATMENT.

One remarkable feature that has struck me in the treatment is the quickness and rapidity with which such cases respond to treatment. The woman becomes almost unidentifiable within a week or so after the treatment has begun and more especially after confinement. In cases of hook-worms, treatment need not be necessarily aimed at the expulsion of the worms. Treatment directed towards improving the blood was found satisfactory. Subsequent to delivery, treatment of the worms by thymol or any suitable drug may be carried out. A case in which thymol was given terminated in premature labour. In syphilitic cases, anti-syphilitic treatment should be started with as little delay as possible. The following routine treatment was adopted by me and was found successful. In all these cases patients were subjected to an orthodox regimen of Bland's pills. In cases where kidney difficulty was disclosed, a powder consisting of 1 grain of Calomel and 30 grains of Caffein Citras and 30 grains of diuretin into six powders, one powder four-hourly, was given with decided advantage, in cases of general anasarca. In cases where the dilatation of the heart is prominent, digitalis and strychnine injections, the former invariably producing the salutary effect of bringing about premature labour, were found useful. During labour the use of a sand bag of

5 lb. and sub-mammary or occasionally intravenous injections of saline formed important methods of counter-acting heart-failure. Post-partum hæmorrhage will be a real uncontrollable catastrophe if precautions against the occurrence are not taken. Judicious management of the third stage of labour is essential lest, a fragment of placenta left inside the uterus may foster violent sepsis which may run riot. Not the least important of all precautions, it is especially needful to adhere closely to the rituals of asepsis; and without proper attention to this from the very commencement of contact with the case in labour no knowledge of the art of obstetrics nor skill and dexterity in its management will avail to avert the most serious, the most distressing, the most anxious and the least pardonable of all complications in midwifery practice, septic infection. It is therefore important that every detail of asepsis must be rigidly and punctiliously carried out. The appearance on the scientific horizon of blood transfusion as a remedy in many of the cases of loss of blood throws some light as to whether this can become an established procedure of efficiently treating these anæmic cases also by the same method.

In fine, ladies and gentlemen, I have with the literature available and with my humble and small experience, outlined a method of treatment for placenta prævia practically available under all circumstances, a method capable of yielding results but little inferior to any other method as far as the maternal mortality is concerned and it can be pronounced to be decidedly superior to those methods which aim at the reduction of foetal mortality at the expense of the mother. In the course of this lecture I have submitted my views here and there for your

professional consideration and judgment. And I should like my experience placed before you to be regarded as incentive for further investigation rather than as any presentation of a definitely shaped obstetrical practice.

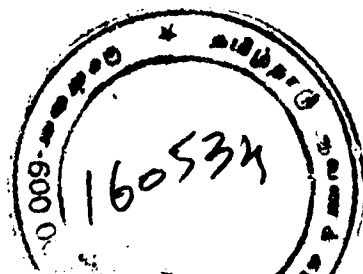
MOLAR PREGNANCY.

An observation of six cases of molar pregnancy makes me emphasise the following points:—

1. Of the six cases three were multiparæ and three primiparæ.
2. The causation of these cases is as obscure as it was when midwifery was born. I am inclined to believe that Endometritis more or less of a specific origin is a fruitful factor in the production of the mole.
3. The condition can be readily missed in one's diagnosis and one might be deluded to believe it to be an ordinary abortion. A proper organised method of examination bearing in one's mind the probability of hydatid mole as one of the probable causes of hæmorrhage in the earlier months of pregnancy, together with the classical and characteristic abdominal signs will seldom land one into error.
4. The text-book description of the appearance of cysts in the discharge as a diagnostic factor is hardly helpful, as cysts were not found in them, commonly.
5. Immediate evacuation is essential and imperative and unless this is done the risk to the woman is grave beyond measure. In this connection I must not fail to mention an

instance of a case where the husband, though very literate did not welcome the suggestion of immediate evacuation and evacuation was postponed to the fatal issue of the woman. Evacuation must be done boldly but cautiously after dilating the cervix with metallic dilators. Fortify yourself with all measures to combat or to prevent a serious post-partum hæmorrhage.

6. Strict adherence to the canons of Asepsis will be the trumpet of your triumph.
7. Recurrence was noticeable in one case but the malignant transformation alarmingly described in books was not noticeable. The three cases traceable so far have not developed malignancy although a year or more has elapsed, and two of them became pregnant one delivering a still-born child and the other continues in her pregnancy.
8. Evacuation must be complete and done cautiously without perforating the uterus, a likely liability.



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