alleviate the distressful headache. After defervescence tonics were given for a fortnight.

(*b*) *Prophylactic.*—In view of the probable cause of the fever two courses suggested themselves: (1) To get rid of the sandflies in the infected barracks. (2) To vacate the barracks. (1) It was thought that fumes of SO_2 would kill the flies, but an examination of the barrack rooms showed that any attempt at fumigation was likely to be futile, as a gallery runs round each room, the planking of which left wide spaces through which the fumes (and the sandflies) could escape. Recourse was therefore turned to (2), *viz*, vacating the barracks. In view of the fact that when No. 2 Double Co., returned from Madoglasht to Drosh no less than 78 cases of fever were in hospital on the 9th day. After their arrival out of a total of 185, I recommended that when the remaining Companies returned early in September, they should go into camp outside the Fort. The result was most satisfactory as the following figures illustrate. No. 4 Double Co. arrived in Drosh on the 4th September and No. 1 D. C. on the 8th September. The admissions for fever subsequently were 16 for the former and nil for the latter Company for the whole month. This could not be attributed to a scarcity of sandflies, for they were everywhere abundant up to the end of the month. Nor could the small admission rate be ascribed to immunity from previous infection before going to Madoglasht, for only 125 men of 209 in No. 1 Double Co., and only 32 out of 175 in No. 4 Double Co., had been previously infected. It seems evident therefore that when the men were removed from the vicinity of the infected cases, though sandflies were abundant, and bit freely, very few cases of fever were contracted. Several cases admitted to hospital for other diseases contracted sandfly fever in the wards. Unfortunately the heat during July and August is such in Drosh, that it would not be advisable to put men (Gurkhas) under canvas at that season of the year.

Immunity derived from an attack.—Out of the 489 cases, 63 men suffered from two definite attacks of fever, but no man from three. From the 53 available records of these cases I find that only 4 men had a typical saddle-back type of fever on both occasions. In a few cases a saddleback type of fever was manifested on the first, or second occasion, and type (*a*) or (*b*) was seen on the other occasion. In most cases types (*a*) and (*b*) were seen in both attacks.

A SPECULATION ON DIABETES MELLITUS. PRELIMINARY NOTE.

By T. H. FOULKES, F.R.C.S.,

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WERE this paper to commence with a description of the treatment employed in the cases shown on the charts which follow, it is extremely probable that no one would read any further, consequently it is necessary to detail the line of reasoning which led to the adoption of this treatment.

To avoid misunderstanding it may be admitted at once that the reasoning is entirely speculative and, but for the startling results obtained from the treatment denoted, there would have been no object in making it public.

A brief review of the physiological beliefs relative to diabetes is necessary, as it is on those beliefs that the hypothesis is based. It is taught by the majority of physiologists that all working cells require dextrose for the output of energy, which dextrose is taken from the blood. A cell cannot immediately burn off the dextrose received, but incorporates it first as glycogen, in which state it is fixed in the cells and may be stored for a long period. During work this incorporated glycogen is used for energy. Von Noorden says, "For the natural fuel of the cells is not glucose but glycogen." (1)

The glycogen used is then replaced from the dextrose in the blood. The amount of dextrose in the blood has a maximum of ·2 per cent. Should the percentage rise higher than this. The excess is excreted by the kidneys and a clinical glycosuria results. Activity of cells then means a lowering of the sugar percentage in the blood.

As the sugar is so imperative to the needs of the cells, some mechanism for maintaining the minimum sugar percentage in the blood is essential. This is one of the functions of the liver, which acts as an accumulator of sugar between the intestine and the tissues, and prevents the periodical flooding by sugar during digestion. Dextrose, absorbed by the intestine, passes to the liver where it is temporarily fixed as glycogen, to be turned out again when the sugar content of the blood is lowered, when extra demands are made by the working cells. Thus the sugar balance in the blood is kept at the constant level; on the one hand it is withdrawn according to the wants of the cells, on the other hand it is renewed from the glycogen store in the liver. The output from the liver is regulated possibly by means of a stimulus through the nervous system, possibly by a stimulus due to paucity of sugar in the hepatic artery blood, or possibly by hormones sent by the cells when sugar is needed, such hormone perhaps being a remnant of the broken down sugar molecule, a "metabolic clinker" such as Dr. Woods Hutchinson describes uric acid to be.

The action of the liver in converting dextrose to glycogen and *vice versâ* may be due to a ferment with reversible action. The pancreas may be partly concerned in this (as is discussed later). Finally, the dextrose conveyed to the liver is the result of digestion of carbohydrate food, this being turned into maltose by the saliva and pancreatic juice, such maltose being eventually inverted to dextrose by the *succus entericus* and the protoplasm of the cells of the intestine.

Turning now to diabetes, and using the term to mean only metabolic diabetes (*i.e.*, excluding gross nervous system lesions and pancreatic cases), it is not proposed to enter into a destructive criticism of existing theories of the pathology of the disease, but one point must be touched upon. It is stated by some, that the essential of diabetes is that the tissues have lost the power of utilising sugar. The evidence seems to point to the contrary being the case. The respiratory quotient in diabetics according to some authorities works out exactly similar to that of healthy people; oxidation has been shown to go on as well in diabetics as in healthy people. (2) A diabetic, until a late stage, shows no signs of starvation, no emaciation and no loss of weight. In fact, the disease is, as often as not, discovered quite by accident and for a very considerable time produces no further obvious effects on the patient.

Let it now be assumed that the seat of the disease is in the intestine, principally of course in the duodenum, and that the essence of the disease consists in a block

in the *absorption* of dextrose, partial or complete, according to the degree reached.

This may be due to some inherent weakness in the intestinal cells possibly ; in certain cases, from overwork.

Assuming for simplicity's sake, a severe form of the disease when the block may be complete, it may be imagined that sugar from the intestines fails to reach the liver at all. But the liver all the time receives insistent demands from the cells for sugar. These demands the liver cannot comply with, having no stored glycogen to convert to sugar. Stimulated to a great pitch of activity, however, by the more and more imperative demands of the cells, it might, in default off sugar, turn out its ferment into the blood. The circulating ferment would seize on any likely material met with and convert it into dextrose. The cells take of the sugar in the blood, and convert it into glycogen as in the normal state ; they may have time to burn it off if working, but they cannot keep it stored for any length of time as the circulating ferment would very soon re convert it into sugar. The cells thus can use the sugar on a sort of cash system, but they cannot store it This would probably not be manifest but for the percentage in the blood rising too high and the consequent action of the kidneys in eliminating it. In this way a vicious circle obtains, the cells constantly crying for sugar and receiving only a ferment which, while giving them sugar, also snatches away any that they try to store ; the kidneys always excreting the excess circulating, thus reducing the percentage in the blood, and so causing fresh calls to be made on the liver which then turns out more ferment. The sugar for the use of the cells is always loose in the blood instead of being fixed in the cells. So one reaches, though in a different way, Von Noorden's conception of diabetes, *i e.*, that "the connecting link, namely, the fixation of glycogen, is wanting."

This extreme stage may never actually occur, but all degrees of it may be imagined. In an early stage only a small number of intestinal cells may fail to absorb sugar, the number increasing as the cause of the disease increases.

The dextrose resulting from salivary and pancreatic digestion then is assumed to remain in whole or part in the intestine. What becomes of it subsequently must depend on conditions prevailing in the intestine. It would not long remain as dextrose, but would undergo very soon either alcoholic, lactic acid, or butyric acid fermentation according to circumstances. The products of such fermentation would be absorbed, and the absorption of such oxidisable matter may account for the beneficial effects of adding carbo-hydrates to an over strict diet when acidosis has set in.

It is well known that in the early stages of diabetes glycosuria only occurs after meals and more so after excessive carbohydrate meals. It is also an old observation that in slight cases when the glycosuria, by careful dieting, has been brought to a clinical zero, a very slight indiscretion in diet, such as an apple or a glass of beer, brings on the glycosuria again. Not only this, but the sugar may re-appear in great excess and may continue to be excreted for a long time after.

Dr. Fagge has said, "Now the only hypothesis which seems capable of explaining such facts as these, is that the saccharine or amylaceous article of food exerts some directly injurious influence so as to cause the blood to contain an excess of sugar for long afterwards and, if we believe that the liver is the organ principally concerned in supplying the blood with sugar, we can hardly help inferring that it is the liver on which this injurious influence is exerted. It seems as if saccharine food were *a poison* to a patient that is affected in this way."

It will be observed that the hypothesis under consideration does not so far account for this action of carbohydrates. It is necessary to account for it and a further assumption is required. Whether the original cause of the disease is excess of dextrose in the intestine or not, cannot be asserted, but it is here conjectured that carbohydrate in the intestines normally causes the intestinal cells to send a hormone to the liver (and perhaps also to the pancreas) to prepare the way for the reception of the sugar to be presently absorbed. This may be considered an extravagant assumption, but it should be remembered that the liver and pancreas are really duodenal cells, differentiated at an early stage of development, but retaining an intimate physiological connection with the duodenum throughout life. The formation and action of secretion show how intimate this connection is. The hormone in question is assumed to pass to the liver and evoke the production of ferment. The argument at this stage is, no doubt, question-begging. The circle is, however, broken by two facts which appear later. No evidence in favour of the assumption of this hormone can be brought forward, but Herter quotes an experiment made by Tuckett of Cambridge (in another connection) which is suggestive. It was found that when thoracic lymph from a *fasting* dog was injected into the portal vein of a cat, neither hyperglycæmia nor glycosuria resulted ; but both of these occurred when the injected lymph was from a *digesting dog.*

The foregoing is the hypothesis on which the experiments which follow have been based, and, as previously stated, nowhere rises above guess work. Without facts it would probably be summed up by most critics in one very short word !

It, however, indicates certain points for investigation. In the first place, it is clear that if a ferment is turned out of the liver into the circulation as assumed, it should be possible to demonstrate its presence in the blood.

This is not only possible, but the presence of the ferment has been found constantly in every diabetic examined.

The demonstration is simple, and consists in testing the diastatic action of diabetic blood on starch solution. In order to avoid the fallacy due to the presence of sugar in the blood, a very minute quantity only is taken from a finger prick

A five per cent. starch solution is taken and the test is always carried out with controls as follows :—

1. A test tube containing two drachms of starch solution alone
2. Two drachms of starch solution with drop of diabetic blood.
3. Two drachms starch solution with drop of normal blood.
4. Two drachms normal saline solution with drop of diabetic blood.

After 24 hours' incubation at about body temperature these four solutions are tested for sugar with Fehling's test and it is found that No. 2 is the only one which has a reducing power. The amount of reduction is, as one would expect, very slight, that is, the red precipitate does not come down on boiling, but after boiling when tube has been standing a minute or two. Somewhat the more exact tests have since been made, taking half a minim each (measured by pipette) of diabetic blood and normal saliva and incubating them separately with starch solution. It is found that the diastatic action of diabetic blood and normal saliva is about equal in ordinary cases. In two very severe cases the blood ferment was far stronger than saliva.

Diabetic saliva has a very much stronger diastatic action in all cases examined so far.

It is only fair to add, that when testing *one* case the blood of his son was taken as control. The blood of both father and son was found markedly amylolytic The son has no glycosuria and seems quite healthy. His blood and urine have been tested several times and at different times of the day, always with the same result. He will be kept under observation.

Amylolytic ferment in blood has been described (5) but the quantity must be very small because, as above stated, only one case of non-diabetic blood among very many has been found to have such action in the minute quantities used. Curiously enough Achard and Clere state that this ferment is diminished in diabetics.

The finding of this ferment was sufficient to encourage a further experiment on the lines indicated by the hypothesis.

It is arguable that if the hyperglycæmia in diabetes is due to the action of the liver as described, the symptoms of the disease should be allayed if the liver could be given its ordinary material, dextrose, to work upon.

So if sugar were injected into the portal vein, it might be expected that the ferment produced by the liver would remain there to react with the sugar instead of being turned loose on the body tissues. We cannot of course recommend injections into the portal vein as a matter of practice, but dextrose, fortunately, is so easily absorbed that it seemed likely if it were injected into the intestine low enough down to be beyond the seat of the disease, its absorption might be followed by beneficial results.

After some vicissitudes it was found that a solution containing ½-1 oz. dextrose in about six ounces of water when injected into the rectum and retained, would constantly diminish the glycosuria the same day; the withholding of the injection is followed immediately by a rise in the sugar excretion.

It has been found necessary to attend to two points.

1. Dextrose must be injected. This may seem unnecessary to emphasize, but much delay and annoyance has resulted from having received a consignment of dextrine on one occasion, and cane sugar on another, in response to orders for dextrose. These were received from two reputable European firms of Chemists in India.

2. The injection must be retained for at least two hours and preferably retained altogether.

3. The testing should always be done by the same man. There is a distinctly noticeable personal factor in Fehling's test.

EXPLANATORY NOTE FOR CHARTS.

The dotted lines and the inside row of figures show the grains of sugar passed daily.

The outside row of figures and black lines similarly show amount of urine in ounces.

Underneath the charts the black arrows and lines show the amounts of glucose injected on the dates indicated above.

A circle denotes that no injection was given that day.

A cross below means that the injection was not retained on the corresponding day.

Perusal of the charts shows very definitely the immediate fall in sugar excretion after injection, and the immediate rise on stopping the injection. The fall is greatest in the first two days becoming less subsequently. This seems to be due to the fact that the thirst diminishes with the fall in the sugar excretion, and therefore absorption from the rectum is more rapid and complete at first. (Patients are now being urged to limit their fluid intake as much as possible to encourage thirst.)

Up to date twenty-seven cases have been charted, but, only four are appended out of consideration for the Editor. They are more or less typical of all.

After many experiments the treatment now adopted is, to start with two injections of dextrose, each of one ounce on the first day, and one injection of half an ounce on the subsequent days. As a rule this causes no inconvenience, but some patients cannot tolerate stronger doses than a quarter of an ounce for any length of time. The absorptive powers of patients also vary greatly as is shown in the cases given. One exceptionally severe case was under treatment last August and September. His urine on admission ranged from 500 to 800 ounces and his sugar from 1,4000 to 18,000 grains per day. His age was thirty, and he gave a history of symptoms from only one year back. He was very weak and emaciated. Under treatment his urine came down to between 170 to 250 degrees fluctuation, and his sugar between 3,000 and 5,000 grains; unfortunately one morning he got a shivering fit and a temperature of 101° and he died the next day, probably from terminal pneumonia common in this kind of case, but no physical signs were detected and no post-mortem was allowed. His diet was as follows:

Milk	... ½ pint.	Vegetables	... 2 oz.
Rice	... 2 lbs.	Tamarind	... ¾ oz.
Bread	... 1 lb.	Condiments	...
Mutton	... 1¼ lbs.	Salt	... ½ oz.
Dhall	... 2 oz.	Oil	...

Daily testing is necessary, as the rectum seems occasionally to fail to absorb, and it is then necessary to stop the injections for a day or two for rest and usually a wash out with a saline is given. It is not yet known how long injections will be necessary to bring any case to zero, it being impossible to get patients of this class to keep on regularly with the treatment. One mild case shown actually came to zero, but he has not come back, so that it is impossible to say if this condition was permanent.

In nearly every case treated the result has been the same and there has been a marked improvement in the general health Some cases do not, however, react, when there is severe illness of any kind; for instance, one case of chronic malaria showed no improvement at all. Intestinal troubles naturally prevent improvement. One other case gave no results, but as he was a policeman applying for medical certificate he may be regarded with suspicion. Late stages of the disease with carbuncles require immediate surgery, as no improvement takes place until the carbuncle has been dealt with. It is in these cases that one finds diacetic acid in the urine which also calls for early treatment. The excretion of sugar usually falls when carbuncles appear, so such cases are not good tests.

It is especially to be noted that no cure is being attempted here, the treatment at this stage being solely employed in order to test the hypothesis. No drugs have been administered and the patients have all been on their usual diets, which, as is well known, are almost entirely carbo-hydrate. No case has been noted as giving a positive reaction for diacetic acid. This is the usual experience here, probably on account of the large amount of carbo-hydrates consumed. Some of the cases as may be seen from the notes are well advanced.

The severity of diabetes depends on the long continued poisoning of the tissues by dextrose (or by the ferment.) This poisoning can at any rate be reduced if not entirely eliminated and though this is an early stage to say so, it does not seem too much to prophesy that no one need in future die of diabetes.

The ferment in the blood should decrease as the patient improves. This, however, requires somewhat delicate testing which is not possible with the means available here.

Whether the hypothesis is proved or not by these cases remains for discussion. The writer, biassed by preconceived ideas, is probably the last man likely to reach a correct conclusion. It seems very difficult to account for the reduction of glycosuria and the general improvement of the patient by dextrose injection on any other grounds than those assumed. There are several weak points, not the least being the presence of ferment in the blood of the son of the case noted above.

The general idea of diabetes then, as reached by this theory, is as follows:—

1. There are two causes to consider.

First, the cause of the disease, which is assumed to lie in the upper part of the intestine, mainly, no doubt, in the duodenum, preventing the absorption of sugar.

B. G. Rao. Age 55.

			Urine, oz.	Sugar, grains.
April	27		122	2,200
,,	28		110	2,000
,,	29		99	2,500
,,	30		104	3,000
May	1	½ ounce	110	1,900
,,	2	,,	100	550
,,	3	,,	90	100
,,	4	,,	90	100
,,	5	,,	90	90
,,	6	,,	99	90
,,	7	,,	90	85
,,	8	,,	92	85
,,	9	,,	70	60
,,	10	,,	70	60
,,	11	,,	70	60
,,	12	,,	70	55
,,	13	,,	65	50
,,	14	,,	65	50
		Discontinued		
May	20		60	1,800
,,	21		68	2,000
,,	22		65	1,750
,,	23	1 oz. bd.	65	1,500
,,	24	,,	62	720
,,	25	,,	33	260
,,	26	,,	33	260
,,	27	,,	34	170
,,	28	,,	32	150
,,	29	,,	28	120
,,	30	½ oz. bd.	30	100
,,	31	,,	32	90
June	1	,,	32	100
,,	2	,,	34	150
,,	3	,,	35	100
,,	4	(Not retained)	35	265
,,	5	,,	35	280
,,	6	,,	37	200
,,	7	,,	35	220
,,	8	,,	35	200
,,	9	,,	34	180
,,	10	,,	35	180
,,	11	,,	35	150
,,	12	,,	...	...
,,	13	,,		...
		Discontinued		
October	12		95	1,300
,,	13		96	1,700
,,	14		100	2,000
,,	15		100	1,700
,,	16		106	1,500
,,	17		110	2,500
,,	18		110	2,300
,,	19		110	2,300
,,	20	1 oz. bd.	115	2,500
,,	21	½ oz. once	90	900
,,	22	,,	80	480
,,	23	,,	66	340
,,	24	,,	68	340
,,	25	,,	62	320
,,	26	,,	62	280
,,	27	,,	62	280
,,	28	,,	62	280
,,	29	,,	62	300
,,	30	,,	60	300
,,	31	,,	56	300

A SPECULATION ON DIABETES MELLITUS : PRELIMINARY NOTE.

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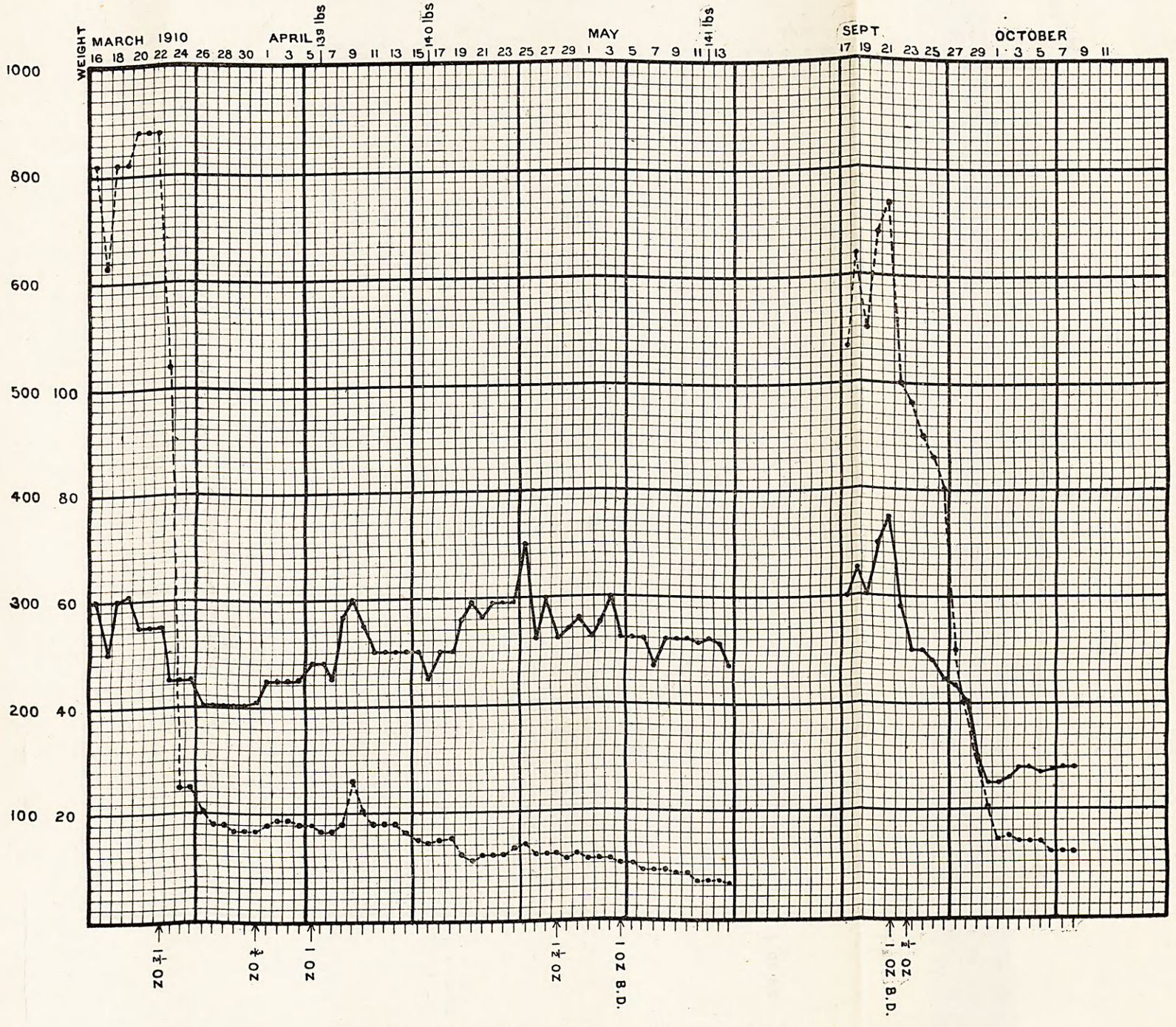
Civil Surgeon, Waltair.

G. APPALA NAIDU, AGE 55.



The outside row of figures show grains of sugar passed daily.

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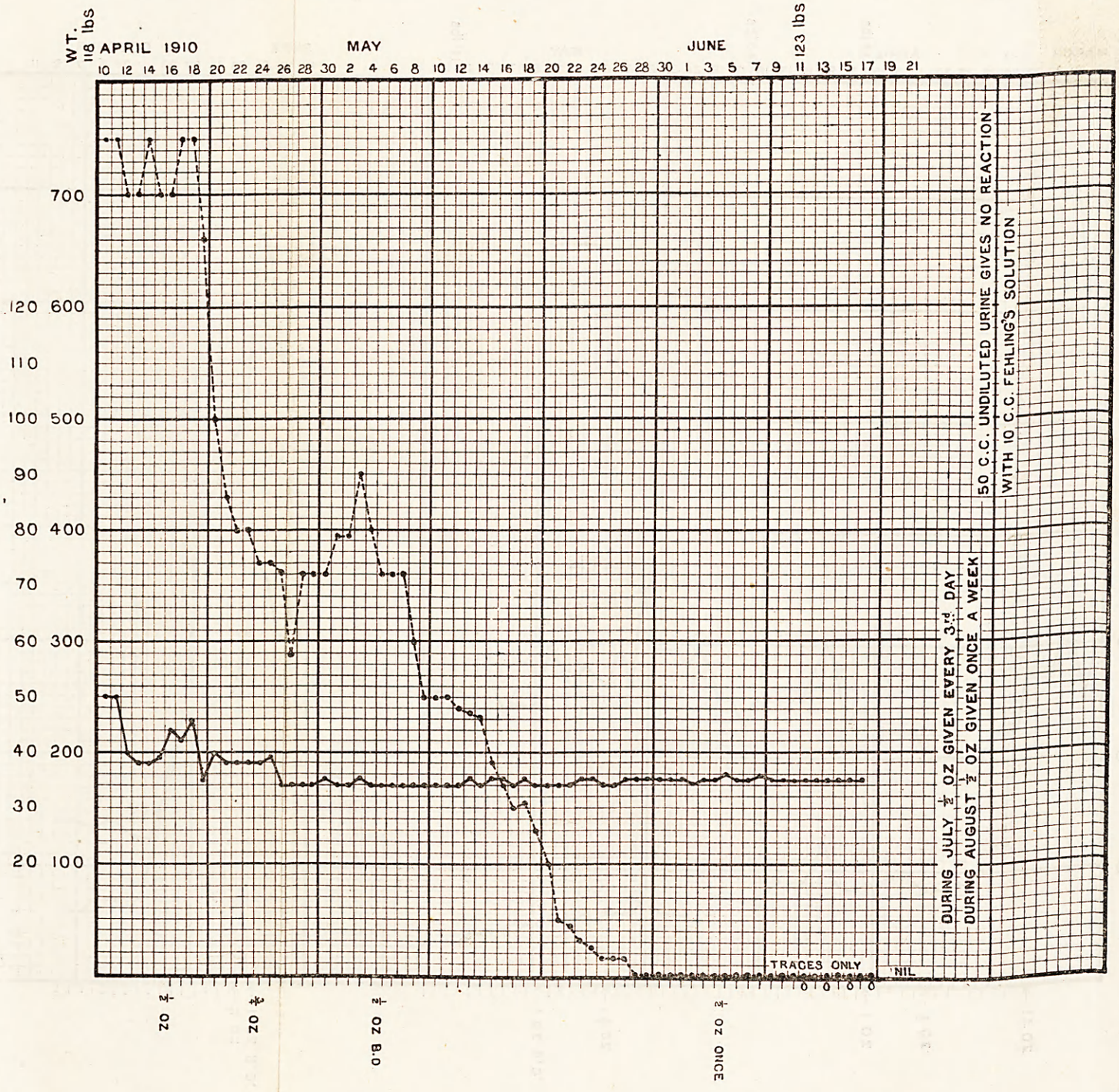
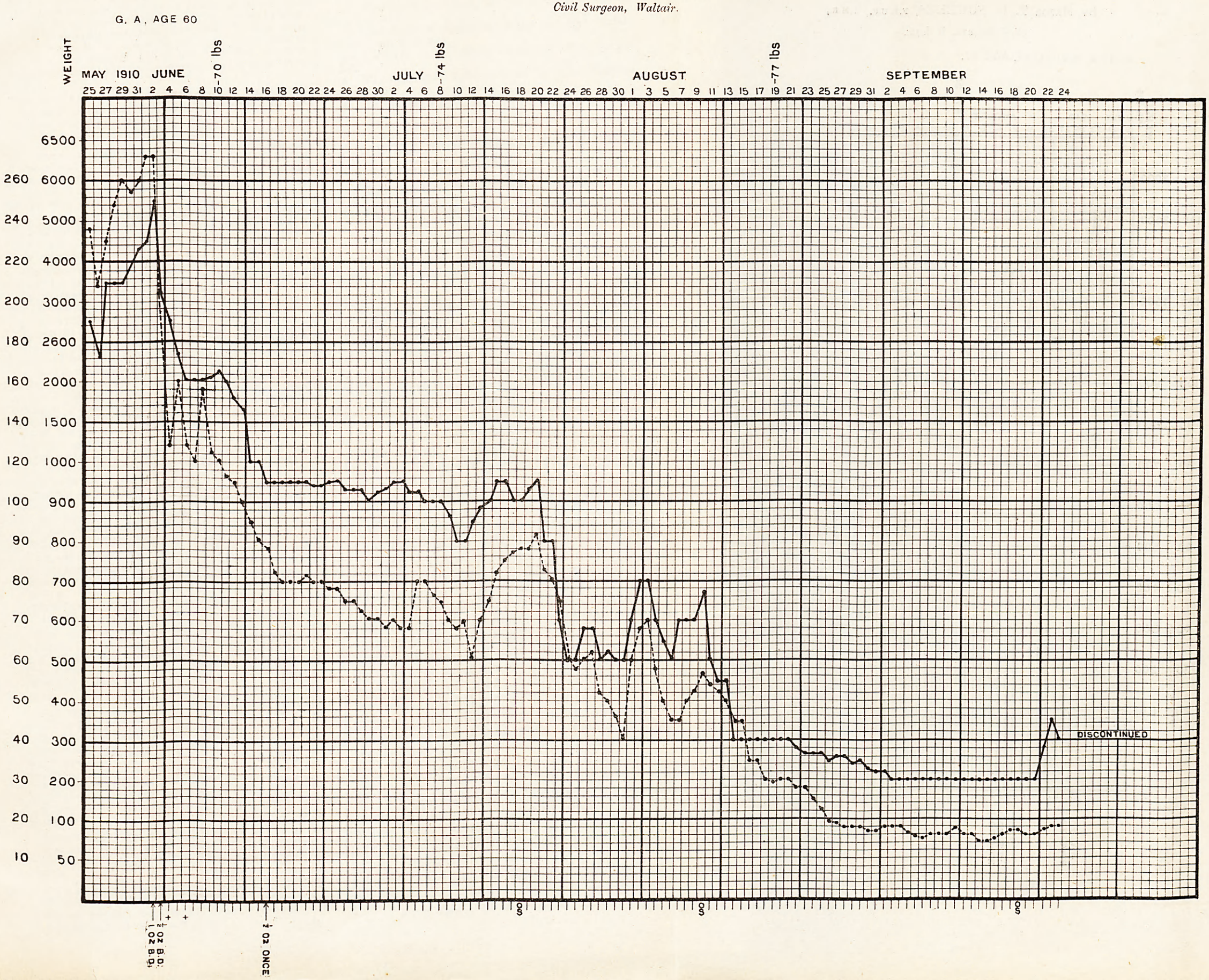
K. S. NAIDU, AGE 39.



A SPECULATION ON DIABETES MELLITUS: PRELIMINARY NOTE.

By Major T. H. FOULKES, F.R.C.S., I.M.S.,

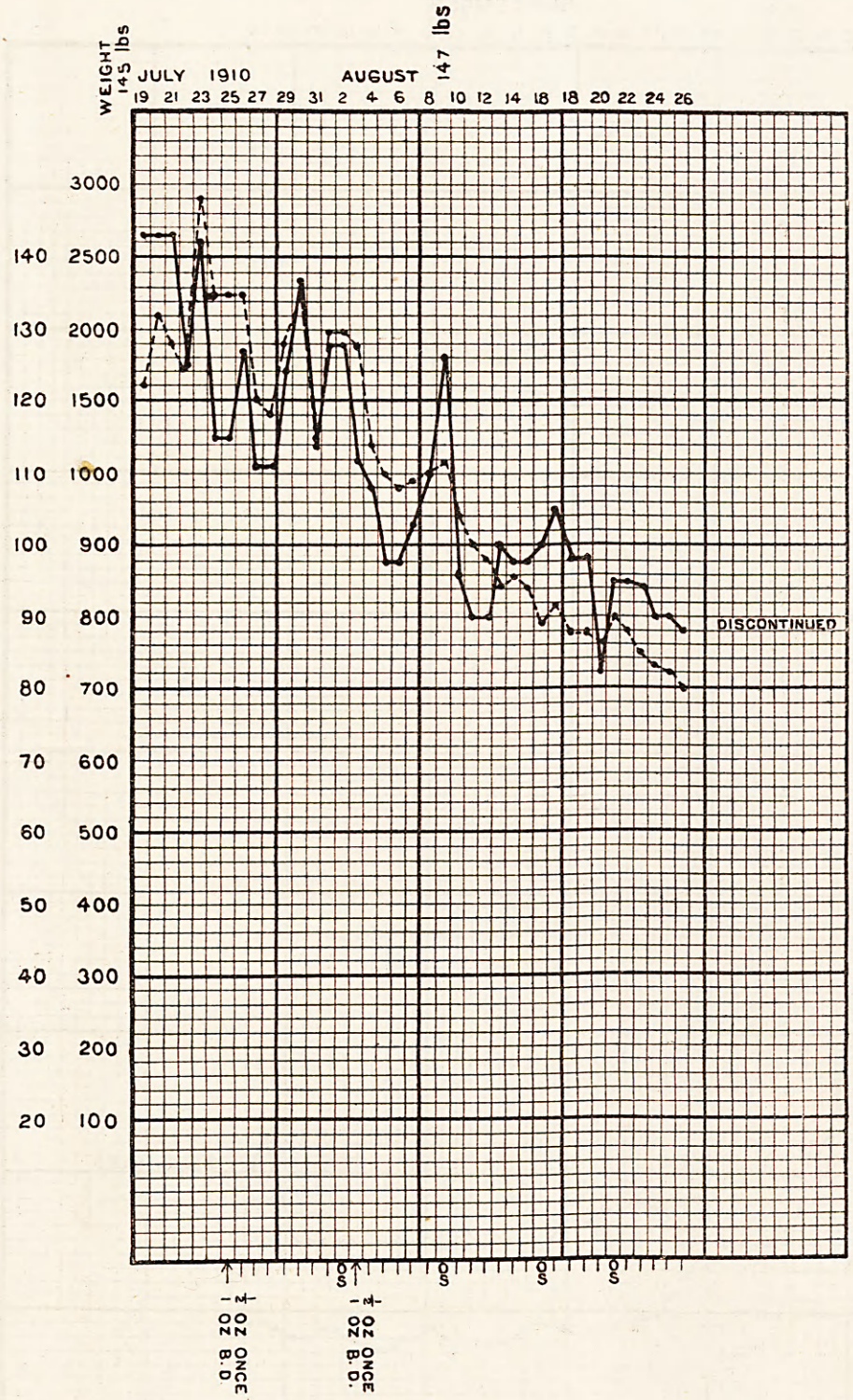
Civil Surgeon, Waltair.



A SPECULATION ON DIABETES MELLITUS: PRELIMINARY NOTE.

By Major T. H. FOULKES, F.R.C.S., I.M.S.,
Civil Surgeon, Waltair.

A. T. S. MUDALIAR, AGE 42.



This cause the present treatment does not touch, and of it we know nothing. Whether it is bacterial, hereditary, inherent weakness in the cells, or poisoning by dextrose, can only be guessed at present. It is treated by dieting and, in suitable cases, may be cured in this way, but such a cure is not available for high caste Hindus who form the bulk of our patients in this country. (Madras Hindus of high caste may not eat meat or even eggs.)

Second, the cause of the symptoms, which is the result of the above, and is the one directly attacked by the treatment described.

2. The sugar in the urine is entirely endogenous.

3. The sugar formed from food is not utilized but acts as poison on the intestinal cells. It may be absorbed as alcohol, which would be a grim irony of fate in high caste men who are professedly among the most teetotal men on earth.

4. It seems likely that a diabetic who developed a fistulous opening between the urinary tract and the intestine would become partially self-regulating, like a shunt wound dynamo.

There are many side issues connected with the subject of diabetes on which speculation might be carried to great lengths. As far as possible in this paper such speculation has been kept within the bounds necessary to make a reasonable working hypothesis.

It will be observed that little mention has been made of the pancreas. It is assumed that the pancreas is not concerned in diabetes. It is true that if the pancreas is removed in animals or, if in man, the pancreas is so grossly diseased as to amount to removal, a mimicry of diabetes occurs. Taking this fact together with the results shown of glucose injection in diabetes, it may well be assumed that the normal action of the internal secretion of the pancreas is to re-act with dextrose *in the liver* acting there as a restrainer of the ferment action and allowing the liver to fix the dextrose as glycogen. The effect of removal or gross disease of the pancreas would be to interfere with glycogen storage in the liver, so that the circulation would be flooded with sugar after every meal with consequent glycosuria and starvation. It may be remembered that all the venous blood of the pancreas passes straight to the liver.

The changes described by pathologists in some cases of diabetes might sufficiently be accounted for by the chronic poisoning this organ, in common with all other organs in the body, has undergone in cases dying of diabetes. Many organs show far more change and especially characteristic and constant are the changes in the stomach and duodenum.

The cases brought forward are few. The results however, in every case are so constant and similar that it is believed they are sufficient to exclude coincidence. The difficulties of getting together a large number of cases and treating them regularly in one place are great.

The experiments mentioned in this paper do not pretend to absolute accuracy. They are done in a corner of the out-patient department. It must be added that this investigation has been done in the spare moments of the usual very busy day's work, and, contrary to the recent statement of Mr. MacVeagh in the House of Commons, the duties of a Civil Surgeon in this country are exceedingly heavy, and they are not done under the most advantageous circumstances.

It is thought that the results shown are of sufficient importance to be made public so that further investigations may be carried on by more competent hands.

I must express my very great obligations to Hospital assistant Appalanarasayya Naidu (retired) and Sub-assistant surgeon Sighamony Pillay who have both rendered me invaluable assistance.

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GLEANINGS FROM THE CALCUTTA *POST-MORTEM* RECORDS.

IV. CIRRHOSIS OF THE LIVER. *

BY LEONARD ROGERS, M.D., F.R.C.P., B.S., F.R.C.S., I.M.S.,

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The Frequency of Cirrhosis of the Liver in India.—Cases of cirrhosis of the liver are very frequently seen in the wards of Calcutta hospitals. Yet common as they now are, some 30 years ago they were nearly twice as frequent in the *post-mortem* records as during more recent years. In view of this fact it is surprising to find Norman Chevers in his Commentary on Indian Diseases (1886), remarking: "I believe, this condition is rarer in Calcutta than it is in London." In the limited literature at hand I have not been able to find any figures illustrating the frequency of the disease in English *post-mortems*, but McFarland in his *Pathology* quotes Forster as having met with cirrhosis of the liver 31 times in 3,200 *post-mortems*, in Berlin or just under one per cent. On the other hand, in 4,809 medical *post-mortem* records in Calcutta, cirrhosis of the liver was met with in no less than 6·9 per cent. of the subjects, or 7 times as frequently as in Europe, if the extensive Berlin statistics can be taken as a fair sample.

This remarkable frequency of cirrhosis of the liver in India—it has been reported to be very common in Lahore by D. W. Sutherland and in Bombay by Gordon Tucker—makes a study of the disease of great interest and importance from several points of view. Firstly, with regard to the rôle of alcohol in the etiology of the disease, medical opinion is in a somewhat fluid state at the present time. Thus, while Hawkins in the last edition of Clifford Allbutt's *System* states that "It is beyond question that the excessive use of alcohol is by far the most common cause of cirrhosis, and that if alcohol did not exist portal cirrhosis would be a rare disease." On the other hand Rolleston in his work on *Diseases of the Liver* and Kelly in Osler's *System of Medicine*, both consider that the rôle of alcohol has been overestimated in the past, and that it acts as a

* A paper read at Medical Section, Asiatic Society of Bengal.