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THE ANTISEPTIC

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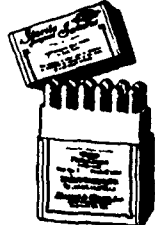
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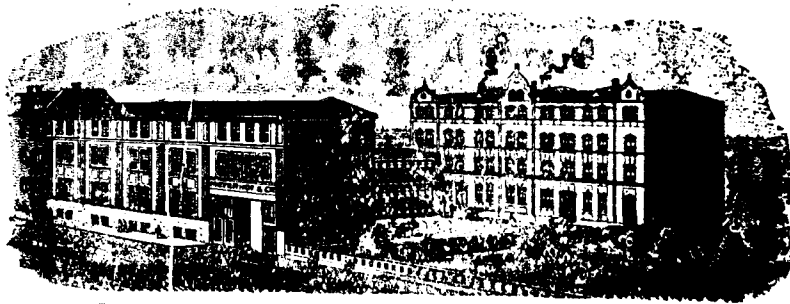
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Wishing you good success and requesting you to give your usual kind attention to our order sent by this Mail through your Agents, we beg to remain.

Yours Faithfully,
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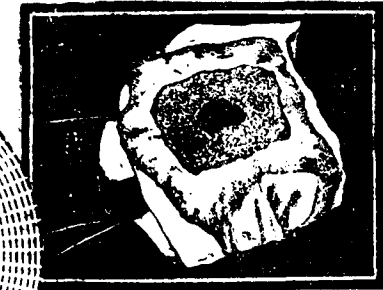
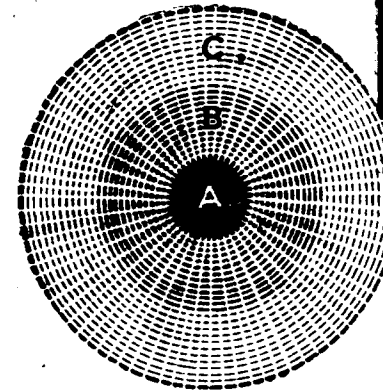
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This chart shows the Osmotic action of Antiphlogistine

DIAGRAM represents inflamed area. In zone "C" blood is flowing freely through underlying vessels. This forms a current away from the Antiphlogistine whose liquid contents therefore, follow the line of least resistance and enter the circulation through the physical process of endosmosis.

In zone "A" there is stasis, no current tending to overcome Antiphlogistine's hygroscopic property. The line of least resistance for the liquid exudate is therefore in the direction of the Antiphlogistine. In obedience to the same law, exosmosis is going on in this zone, and the excess of moisture is thus accounted for.

c. p. glycerine of Antiphlogistine and the water of the tissues, Antiphlogistine keeps up a steady heat generation.

This sustained heat is invaluable; relieving congestion by increasing superficial circulation, stimulating the cutaneous reflexes, and causing contraction of the deep-seated blood vessels.

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Antiphlogistine stands alone as a non-toxic, non-irritant abstractor of fluid exudates in superficial inflammations. It relieves deep-seated congestion by inducing superficial hyperemia, through its inherent hygroscopic property, and without irritation.

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Antiphlogistine generates and retains heat upwards to 24 hours

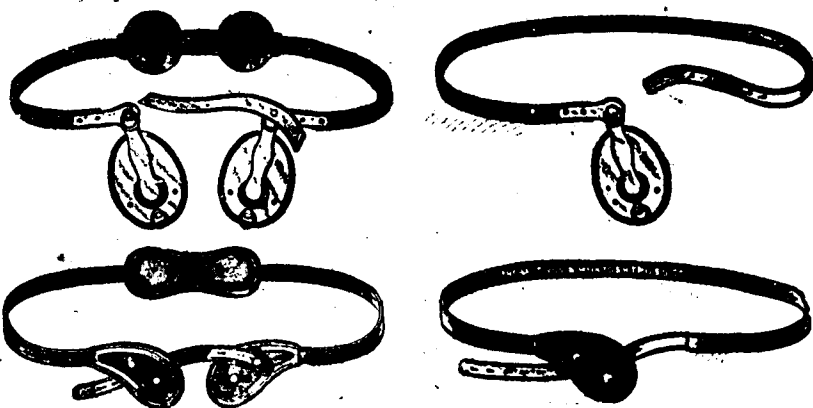
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As an analgesic in gynecological cases
Ergoapiol (Smith) is superior to opium
or coal-tar derivatives in that, besides
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patient to the danger of drug addiction,
it also offers a tonic and restorative
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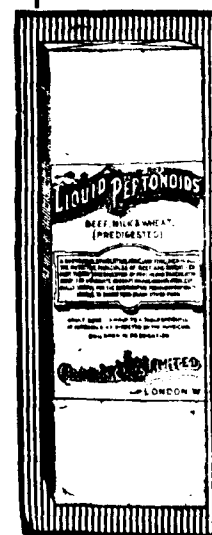
It is a uterine and ovarian sedative
of unsurpassed value and is especially
serviceable in the treatment of congestive
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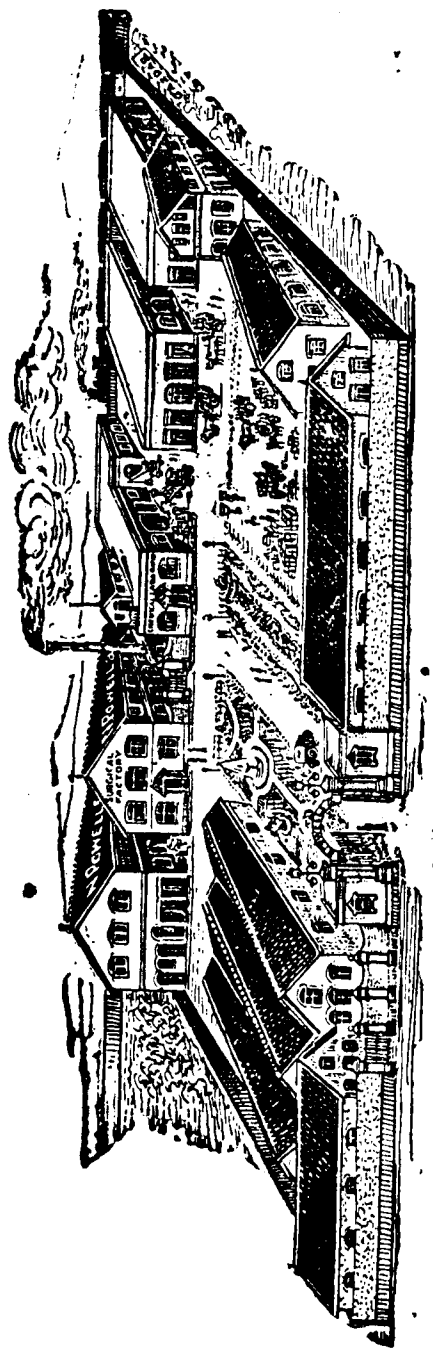
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Supplement to this issue.



Dr. F. B. BANTING,
DISCOVERER OF "INSULIN"

The Antiseptic

VOL. XXI
No. 1.

JANUARY, 1924.

ANNUAL
SUBSCRIPTION
Rs. 5

***HYPERGLYCÆMIA AND ITS SIGNIFICANCE**

By

J. P. BOSE, M. B., F.C.S.,

Mitra Research Scholar on Diabetes, Calcutta School of Tropical Medicine.

Hyperglycæmia is the condition in which the blood contains an abnormal amount of sugar. The normal blood-sugar content varies to a certain extent amongst different races, probably depending on the nature of the diet and a few other causes. Thus, the European living mostly on a diet rich in proteins has a lower blood-sugar content than the average Indian. The normal blood-sugar content of a healthy European has been found on an average to vary from 0.08 to 0.11%.

The blood-sugar content of a healthy Bengalee, on the other hand, varies rather widely. As a result of my investigations, I find that the blood-sugar content of Bengalees varies from 0.08 to 0.15%. It is interesting to note that an average Bengalee living on a diet which is very rich in carbohydrate and which is often taken in much larger quantities than is required by the system and, at the same time, leading a sedentary life, invariably shows a blood-sugar on the high normal side i.e., varying from 0.12 to 0.15%. By this statement, it must not be understood that all Bengalees give a blood-sugar figure higher than that obtained in the case of Europeans. I have in my record blood-sugar figures of a few Bengalees in whom they varied from 0.08 to 0.11% which is quite comparable with the figures for Europeans. These were individuals with active habits and accustomed to take mixed diet in moderate quantities.

The technique for blood-sugar determination has considerably advanced within the last few years. It is not my object to describe in this paper the history of its origin and the various methods which were adopted from time

* Specially contributed to "The Antiseptic."

to time for its determination. It will suffice to say that in all the old methods, the technique was tiresome and the results not quite accurate, and the amount of blood required for the test was large. This was the reason why blood-sugar determinations could not be done as often as one would have liked to do for investigation of a case, or for following the effects of treatment. With the rapid march of Bio-Chemistry and with the development of the micro-chemical methods of analysis, the clinical investigation has become very much simplified. With the introduction of the various instruments, such as the *Torsion Balance*, by which we can weigh quite accurately a few drops of blood, and the new type Colorimeters by which solutions of the same colour but of different strengths could be compared with the greatest of ease, the blood-sugar estimation has become as simple as the *rule of three* in arithmetic.

The modern method of blood-sugar analysis has done a valuable service to practical medicine. The possibility of repeating the blood sugar estimations at frequent intervals has not only given us an opportunity to investigate a case thoroughly or to follow the effects of treatment, but has helped to develop the sugar tolerance tests materially. (1) Thus by giving a person a known amount of glucose, levulose or galactose by the mouth and estimating his blood sugar at short intervals afterwards, we can accurately study how he is responding to the ingested sugar. This test would not only differentiate a non-diabetic individual who, for some reason or other, is passing sugar in the urine, from a true diabetic but would often help to establish diagnosis of the disease in the pre-diabetic stage.

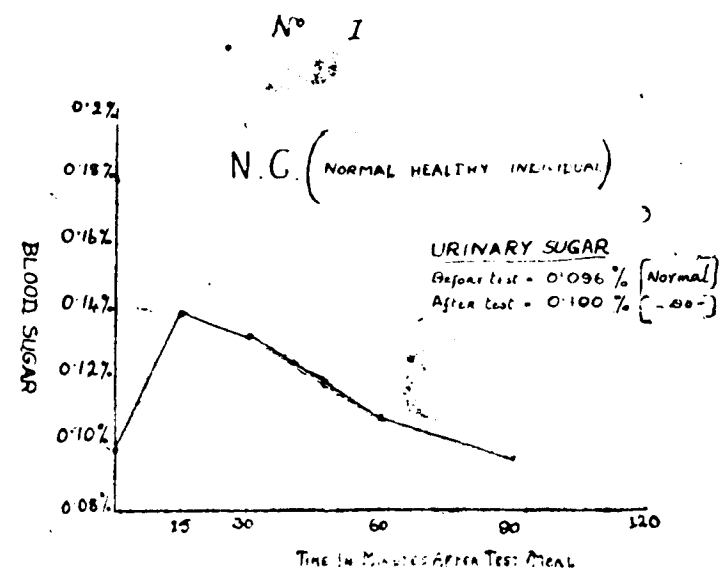
To do the Glucose Tolerance Test—

- (1) The patient must fast for at least 3 hours before the test.
- (2) A single blood-sugar estimation is done before the test is started (Fasting blood-sugar level).—
- (3) A test-meal of 50 grammes of glucose dissolved in about 100 c. c. of water is given to the patient to drink.
- (4) Blood-sugar estimation is done exactly at the end of 15 minutes. This process is repeated at the end of $\frac{1}{2}$ hour, 1 hour, $1\frac{1}{2}$ hours, and 2 hours after the test-meal is given.
- (5) The urines before and after the test are collected and the amounts of sugar present (normal or abnormal) estimated.

Appended below are two charts which illustrate "The Glucose Tolerance Test curves" in—(1) Normal healthy individual and
(2) Potential Diabetic (Pre-diabetic stage)

(1) J. P. Bose.—Glucose Tolerance Tests and their interpretations. I. M. G. Vol. LVIII, No. 6, June, 1923.

GLUCOSE TOLERANCE TEST

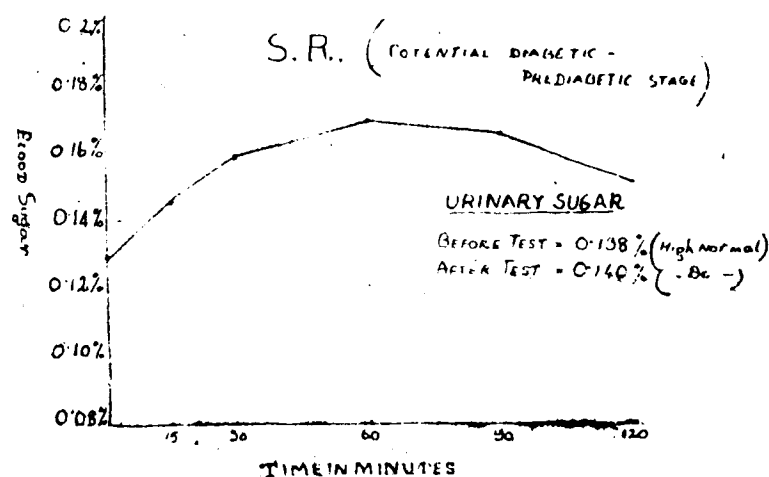


Note that—

- (1) The blood-sugar before the test is normal and so is the urinary sugar.
- (2) The maximum rise is in a quarter of an-hour's time and it is much below the *leak point* (about 0.18%).
- (3) The drop to normal is in 1 hour's time.
- (4) The urinary-sugar at the end of 2 hours is normal.

GLUCOSE TOLERANCE TEST

N° II



Note that—

- (1) The blood-sugar is "high-normal" to begin with and so is the urinary sugar.
- (2) The blood-sugar continues to rise after the test meal of glucose and the maximum rise (which is quite near the *leak point*) takes place at the end of 1 hour instead of a quarter of an hour.
- (3) The fall of the blood-sugar level is very gradual (*unlike Curve No. 1.*)
- (4) The blood sugar at the end of 2 hours is still raised above the normal. The long drawn-out curve suggesting delayed carbohydrate assimilation is quite striking.

THE MICRO-CHEMICAL METHOD OF ESTIMATION OF SUGAR IN THE BLOOD. (2)

The principle of the method is that an estimation of the sugar-content of the blood is made upon a few drops of blood obtained by a small prick of the finger. The hæmoglobin and proteins are precipitated from a known weight of blood by means of tungstic acid, and a crystal clear—watery filtrate obtained. This filtrate, when heated on a water-bath with an alkaline solution of copper tartrate, produces a reduction of the copper according to the amount of sugar present. The addition of a solution of phospho molybdic acid of such a strength that equal quantities will cause the

(2) Wallis and Bosc—Journal of Obstetrics and Gynaecology of the British Empire, Vol. 29, No. 2, Summer 1922.

copper solution to lose its blue colour, results in the production of a deep blue coloured solution. This blue colour is compared in a colorimeter with the colour obtained by treating a known standard sugar solution in the same way.

RE-AGENTS AND APPARATUS REQUIRED.

- (1) A sharp needle for pricking the finger.
- (2) Starch-free absorbing papers of sufficient size to absorb 120 to 150 milligrams of blood. The usual size supplied for this purpose is 16×28 mm, and the paper is specially prepared.
- (3) Steel-wire clips to hold the papers, and suspend them on the hook of the torsion balance.
- (4) A good torsion balance capable of weighing from 1 to 500 milligrams. This torsion balance, if well constructed, will give constant weights irrespective of temperature or humidity.
- (5) Small test-tubes of such a size that the special absorbing papers slip in, and can be completely submerged in the extracting medium.
- (6) Starch free filter papers (No. 1 Whatman).
- (7) Two burettes of 2 cc. capacity graduated in one-fiftieths with double bore stopcocks and mechanical fillers, to hold the two precipitating solutions, viz :—
 - (a) A 10 per cent. solution of sodium tungstate (pure crystals).
 - (b) A two-thirds normal solution of sulphuric acid.
- (8) A burette of 10 cc. capacity for distilled water.
- (9) A boiling water-bath with holes of a suitable size to hold the special boiling tubes in an upright position.
- (10) Boiling tubes of resistance glass graduated to hold at least 40 cc. with graduation-marks at 12.5 cc. and 25 cc. The lower end of the boiling tube is provided with a bulb of 4 cc. capacity, and a narrow neck above, 2 cm. in length and not more than 8 mm. in diameter. The bulb is designed to hold the mixture of blood filtrate and copper solution, and the constricted neck serves to prevent re-oxidation of the copper oxide by reducing the volume of liquid exposed to the air in the tube above.
- (11) Oswald pipettes of 1 cc. and 2 cc. capacity.
- (12) Alkaline copper solution made up as follows :—
 - Anhydrous sodium carbonate, 40 grammes.
 - Tartaric acid, 7.5 grammes.
 - Crystalline copper sulphate (pure), 4.5 grammes.

The sodium carbonate is dissolved in about 400 cc. of distilled water contained in a litre flask, warming of the mixture on a water-bath being necessary. The copper sulphate is dissolved separately, and then added to the carbonate solution, and finally the tartaric acid is added to the mixture.

The solution is made up to 1 litre volume with distilled water, and then filtered.

(13) Phospho-molybdic acid solution.—This is prepared by dissolving 35 grammes of pure molybdic acid (free from ammonia) in 200 cc. of 10 per cent. sodium hydroxide. To this, add 200 cc. of distilled water, and heat until all traces of ammonia are expelled. Cool, and then add 125 cc. of phosphoric acid (85% strength), and finally make up in a 500 cc. flask to the mark with distilled water. This solution should be of such a strength that 2 cc. will completely discharge the blue colour from 2 cc. of the copper solution. It is advisable to test these reagents from time to time, and to do blank estimation with the reagents occasionally.

(14) Standard solution of glucose.—One gramme of pure glucose is dissolved in 100 cc. of distilled water, and preserved with toluol. Its strength is determined by the polarimeter and by quantitative analysis. In this strength, it can be kept in a refrigerator for a month or more and used as a stock solution. To make up the standard solution from this, 1 cc. of the stock solution is placed in a 50 cc. flask, and diluted to the mark with distilled water. Each cc. of this standard solution contains 2 milligrammes of glucose.

(15) A good colorimeter, preferably the Kober type. The plungers in this instrument are fixed, and the cups are movable. The sides of the plungers are of black glass as also the sides of the cups, and thus there is no space between the fluid and the prisms to allow of extraneous sources of light, which introduces errors into instruments like the Duboscq colorimeter. Another advantage of Kober instrument is that the standard solution can be fixed at any point, and moved in relation to the unknown solution, thus enabling both strong and weak-coloured solution to be compared with ease and accuracy. To ensure readings of the colorimeter, it is advisable to use the same volume of reagents in each case, and the same dilutions. Working with the standard solution and the usual weights of blood absorbed on the special papers, it is possible to obtain colour which match very well, and give readings close together on the colorimeter scale. Depth of colour is proportional to the amount of sugar present, and if the amount of sugar in one cup is known, then the amount of sugar in the unknown can easily be calculated.

METHOD OF ESTIMATION.

The finger of the patient is cleansed carefully with alcohol or ether, and the flow of blood assisted by pressure of a piece of soft rubber tubing. The portion of finger just below the bed of the nail is selected, and a sterile needle inserted. The paper and hook having been weighed, is now soaked with blood as evenly as possible, and rapidly weighed. With a good flow of blood from the prick and careful absorption of blood, about

120. milligrams of blood can be obtained on each paper, and this is quite sufficient for a single estimation. To ensure good results with the torsion balance, it is necessary to acquire speed in absorbing the blood and also weighing, and not to touch either the paper or hook with the fingers. All the manipulations of the paper and hook are done with a pair of fine-pointed forceps, preferably ivory-tipped. Using good absorbing paper, and quick weighing on the torsion balance, the loss of weight by evaporation is almost negligible. The method of collecting and weighing the blood has much to recommend it, as it is rapid, accurate, cleanly, and impressive. The patient suffers just the slight inconvenience of the prick of the finger, and thus the estimation can be carried out on persons of any age, sex or temperament, without any danger of emotional disturbance. Sufficient blood for an estimation has been obtained from a patient during sleep, and the manipulations have been so light that the sleep remained undisturbed.

The paper soaked with blood is now inserted into a small test tube and 3.6 cc. of distilled water added. This amount of fluid is sufficient to cover the paper, and extract the blood from it, the extraction being allowed to proceed for exactly half an hour at ordinary room temperature. At such temperatures, and with this interval of time, extraction is complete, and there is no danger of glycolysis occurring with consequent disappearance of the sugar. The extraction must not be allowed to proceed beyond one hour. The proteins, etc., are now removed by means of the tungstic acid precipitation method of Folin and Wu, 0.2 cc. of the 10% sodium tungstate solution being first added, and then 0.2 cc. of the two-thirds normal sulphuric acid. This makes a total volume of 4 cc. The mixture shows a reddish precipitate which rapidly turns brown with the formation of a thick viscous precipitate. The precipitation is complete when a note like shaking mercury in a glass vessel is emitted. The mixture is now ready for filtration through a starch-free filter paper into a small clean tube, and if precipitation has been complete, a clear watery filtrate is obtained. It is important to note that all the materials and reagents used are standardized to precipitate a given amount of blood. Care should, therefore, be taken to ensure that at least 100 milligrams and not more than 140 milligrams of blood are taken for each estimation. The sodium tungstate solution and sulphuric acid solution should be so balanced that, after precipitating a given weight of blood, the filtrate is neutral or only slightly acid in reaction.

For an estimation, 2 cc. of the clear filtrate are measured by means of an Oswald pipette, and placed in one of the special boiling tubes and 2 cc. of the alkaline copper solution added. In another boiling tube, place 1 cc. of the standard glucose solution and 2 cc. of copper solution, and make up to the neck with distilled water. The two tubes are now inserted

in a boiling water bath and allowed to remain exactly six minutes. They are then removed from the bath and 2cc. of the phospho-molybdic acid solution added to each. A brisk effervescence occurs, and in the presence of sugar, a deep blue colour results, the depth of colour depending on the amount of sugar present. The contents of both the standard and the unknown are now diluted to the 12.5 cc. mark and thoroughly mixed. The two solutions are now placed in the two black cups, and compared in the colorimeter. The standard sugar solution may be set at any position in the colorimeter, but it is advisable to obtain readings of the unknown as near as possible to that of the standard solution. The colour is of such a distinct blue that, after a little practice, it can be matched with ease and remarkable accuracy.

The calculation is made as follows:—

Weight of paper and hook = 250 mgms.

Weight of paper, hook and blood = 360 mgms.

Weight of blood taken = $360 - 250 = 110$ mgms.

With the standard sugar solution set at 20 mm. on the colorimeter scale, the reading of the unknown solution is 25 mm. 1cc. of the standard sugar solution contains 0.2 milligrams of glucose. The amount of sugar present in 2cc. of the tungstic acid filtrate =

$$\frac{\text{Reading of standard} = 20}{\text{Reading of unknown} = 25} \times 0.2 = 0.056.$$

The amount of blood taken = 110 milligrams, and the amount of extracting fluid used = 4 cc.

$$\text{The amount of blood in the 2cc. of filtrate} = \frac{110 \times 2}{4} = 55 \text{ mgms.}$$

$$\text{Percentage of sugar in the blood} = \frac{0.056 \times 100}{55} = 0.101.$$

This result indicates a normal blood sugar content, the normal average figures ranging from 0.08 to 0.110 gram per cent.

I would now pass on to the consideration of the various conditions under which glycosuria may supervene and then discuss what the advantages are of the determination of blood-sugar over the urinary sugar. In old days when the blood-sugar method was not perfected, any one passing sugar in the urine was taken to be a diabetic. We know now that this is not so and consequently the definitions given of Diabetes Mellitus in the old text-books has got to be modified at the present day. We know now that a persistently raised blood-sugar is a more valuable clinical sign of Diabetes Mellitus than glycosuria. Those who have some experience in examining cases for life insurance know that diabetics would often prac-

tise strict dieting for a few days before they come for their examination. Now, if one finds an absence of glycosuria in these cases and bases his diagnosis on that alone, he would often do a serious mistake and the company would never forgive him for that. If the blood-sugar in such cases were examined, it would have been found to be raised much above normal. The urine examination may also reveal the presence of acetone-bodies in many of these cases indicating that a fraud has been practised. In cases like these, it is always advisable to give the patient a test-meal of two ounces of sugar dissolved in water and examine the urine one hour later. A diabetic under such conditions would pass a large amount of sugar, whereas a normal person would still be sugar-free by the ordinary tests usually employed in such examinations.

INTERNAL SECRETION AND HYPERGLYCAEMIA.

Acceleration of internal secretions of the thyroid, pituitary and the supra-renal glands may produce hyperglycaemia and even glycosuria. Glycosuria in Graves disease is quite common; in the early stages of Acromegaly, glycosuria is often noticed and the injection of adrenalin produces both hyperglycaemia and glycosuria.

In this connection, I do not think it would be out of place to mention the experiment which I carried on rabbits sometime ago to study the control of the nervous system on hyperglycaemia. It is well known that rabbits have a very sensitive sympathetic system. If they are frightened, their sympathetic system gets stimulated and there is an increased secretion from the supra-renals which produces a temporary hyperglycaemic condition in them. I found that when the blood was drawn quickly without disturbing the animal, the blood-sugar figure ranged between 0.12 to 0.20% but if the animal was brightened before the blood was drawn, the results were much higher.

RENAL THRESHOLD.

It has been found by experiment that in a normal individual, the concentration of sugar in the blood and urine is nearly equal. Now, it has been found by experiments that following ingestion of glucose, the urinary sugar runs parallel with that of the blood till the latter gets at concentration of an average of 0.17%. As the sugar in the blood rises above this point, the kidneys begin to excrete sugar. This point of concentration of the blood sugar has been described as the "Renal threshold" or the *Leak point* of the kidney.

Opinions differ among different workers in the field as to the figure for this threshold point. Thus, Jacobson says that glycosuria does not occur till the blood-sugar rises beyond 0.17%, Hamman and Hitchman on a study of 50 cases gave the same figure. Foster's figure varies between 0.149 to 0.164%. Bailey's figures come to about 0.167%. As a

result of my experiments, the figure of *leak point* for Indians is on an average 0.18%

This *leak point*, however, is not a constant factor. It varies to a certain extent in different individuals and much more so in diabetic individuals. If you examine the blood and urine of these cases before you start treatment, you will find that they probably have a blood sugar concentration of over 0.3% and passing about 3 to 4% of sugar in the urine. Now, if you starve the patient for 2 or 3 days, you will find that glycosuria will disappear but the blood sugar would still be raised much above the *leak point*.

It has been observed that when diabetes is mild or quiescent, the renal threshold remains stationary at about the normal level but when the disease is progressive, the threshold begins to rise. In a few rare cases of diabetes, the renal threshold was found to reach great heights with little or no sugar appearing in the urine. According to many observers, a rising renal threshold in the face of a careful dietetic treatment is a serious prognostic sign and usually indicates some sort of complication.

In treating a diabetic case, therefore, it is always desirable to maintain the blood sugar level as nearly to normal as possible though severe restrictions in diet may be necessary to attain this point. In such cases, the higher renal threshold might permit of a much more liberal diet without the appearance of glycosuria but this is not a safe plan to follow. I think it is much wiser to follow the former plan in the dietetic treatment of diabetes than to permit food which is just sufficient to prevent glycosuria.

Now let us see what the significance of a high *leak point* for sugar is in a diabetic:—

(1) It may be physiological and may be regarded as nature's attempt to prevent loss by storing up food material.

(2) In severe diabetics where extremely low diets are tolerated, the high threshold is essential to take care of the seriously impaired Carbohydrate metabolism.

To summarize, a persistently low blood-sugar level in a diabetic may be regarded as an extremely favourable prognostic sign, whereas a persistently high blood-sugar, in spite of under-nutrition, is usually an unfavourable indication.

*THE PHYSIOLOGICAL PROBLEMS OF DIABETIC METABOLISM

By

SEBASTIAN RODRIGUES, B.A., M.B., B.S. (BOMBAY).

(1) *Diabetes Mellitus an aberration of normal metabolism*—

The problem of the vitiation or aberration of normal metabolism and of the normal functions of the tissues of the vertebrate animal that takes place in Diabetes Mellitus has long engaged the attention of physiologists and pathologists. The recent isolation of Insulin and the discovery of the phenomena effected by its administration both in normal and diabetic systems have probably given us data sufficient enough, if interpreted correctly, to lead us to understand the essential nature of this vitiation of normal metabolism. In this paper an attempt will be made to study the intrinsic nature of this vitiation of metabolism.

(2) *The nature of the functions of the physiologically active tissues.*—

As diabetic metabolism involves a vitiation of the normal metabolism and activities of various tissues, a consideration in some detail of the normal metabolic activities and functions of various tissues in the vertebrate system is necessary to be able to understand the essential nature of this vitiation of metabolism. The tissues of the vertebrate animal may be primarily divided into two physiological groups, (1) The passive skeletal tissues whose function is to form the frame work passively covering, supporting and protecting the more active tissues of the next group, and (2) The physiologically active tissues whose function is to adopt by their active metabolic activities and functional processes the internal system of the animal to its internal and external requirements. The skeletal tissues may further be classified as exo-skeletal and endo-skeletal. The exo-skeletal tissue consists of a layer of connective tissues called dermis covered by layers of stratified epithelium, the outermost of which undergo keratinization. The deeper layers of this epithelium may contain pigment granules which by absorbing the actinic rays protect the system from the toxic physico-chemical action of these rays which have powerful chemical powers and if allowed to act upon the substance of the deeper tissues would by causing chemical changes in them alter them in toxic substances. (These pigment granules also seem to have the function of activating under the influence of the actinic rays the fat soluble vitamins into substances stimulating the metabolism of the tissue in some way preventing rickets and facilitating development of anti-microbial immunity.)

The endo-skeletal tissues are bone, cartilage, ligaments, tendons, fascia areolar tissue, etc., whose function is to form the framework supporting

* Specially contributed to "the Antiseptic"

and containing the more active tissues. In the matrix of these connective tissues are found cells which have active specific synthetic functions with regard to these tissues. In the cytoplasm of these cells, synthetic ferments are elaborated which are then secreted extra cellularly and by their extra-cellular synthetic action synthesize in situ the substances of the matrix and fibrous elements of these connective tissues. The function of these connective tissue cells is merely local trophic synthesis with tissue hypertrophy and hyperplasia during growth, and the metabolic activities of these cells are extremely slow. In the physiologically active tissues, metabolism is however extremely rapid, these tissues taking the active effective part in adjusting the system of the animal to its perhaps rapidly changing external environmental relations and internal requirements. Diabetes Mellitus is a disturbance in the normal metabolism of sugars, fats and amino-acids in the system effected by the normal co-ordinated metabolism and functional activities of these tissues. A consideration of the nature of their normal metabolic activities and functions is necessary for a proper study of diabetic metabolism. The master tissue of these tissues is neuronic tissue which tissue has the specialized function of initiating and conducting nervous impulses which impulses play an important part in co-ordinating the metabolic activities and function of other active tissues. A typical neurone consists of dendrons, nucleated cell body and axon. Anastomosing strands of longitudinal fibrils pass from the dendrons into the nucleated cell body and from these into the axon, and represent the specific structure conducting the nervous impulse. Next in physiological importance of function are tissues having the function of contractility. There are four main types of contractile tissues, three of which have long been well known as voluntary striated muscle, involuntary cardiac, and involuntary unstriated muscle. The fourth type of contractile cell was described by Rouget nearly 40 years ago, though its physiological importance has only been recently generally recognized. It is a greatly modified form of involuntary unstriated muscle cell and consists of a flattened oval nucleated cell body closely applied to the external surface of the capillary blood-vessels with fine protoplasmic processes completely encircling at intervals the capillary vessel. The cytoplasm and protoplasmic processes have the specific function of active contractility, the cell regulating by this activity the size of the capillary lumen. Dale (British Medical Journal, June 9th and 16th, 1923,) supposes that Rouget's cell also regulates by its functional activity the permeability of the capillary wall to transudation of fluid from within the lumen of the capillary into the tissues without. It is,

however, difficult to understand how the fine protoplasmic processes of these cells, which encircle the capillary wall at intervals only, would physically prevent uncontrolled transudation through the interspaces between these processes, unless there is some structure in the same plane, as these processes completely filling up these interspaces which structure is absolutely impermeable to transudation in both directions. Such a structure has not yet been described, and unless we assume its presence we cannot ascribe to the contractile cell of Rouget, besides the function of contractility it undoubtedly possesses, the specific function of directly regulating the permeability of the capillary wall. This specific function we must rather ascribe to the capillary endothelium itself. These endothelial cells are apparently capable of altering their permeability to transudation, the permeability in one direction increasing as that in the reverse direction decreases, according to the needs of the system. This specific function of these cells which may be called variable differential permeability, is controlled by vaso-motor activity and regulates the amount of the fluid in the blood vessels in accordance with circulatory requirements. (*Vide Antiseptic*, March 1923, page 112.) A similar function of selective variable differential permeability for certain substances at least must be assigned to certain cells like the epithelial cells of the intestinal villi, the cell lining Bowman's capsule and the renal tubules etc., these cells forming a continuous layer lining the lumen of a cavity or tubule and by the exercise of this specific function absorbing from or excreting into the lumen of the cavity certain substances whose absorption or excretion is necessary for the requirements of the metabolism of the system. In the basement membrane of certain external glands like the salivary are found flattened endothelial-like cells with branching protoplasmic processes which anastomose with the process of other like cells, the intervals between these processes being completely filled with a thin homogeneous membrane. It is not impossible that these cells having a special function of variable selective differential permeability play through the exercise of this function the effective factor in the formation of the watery portions of the natural secretion of these glands, the homogeneous membrane between these processes playing a passive part being impermeable in both directions. To the gland-cells of certain externally and internally secreting glands must be assigned the specific function of synthesis and liberation or secretion of certain substances necessary in the economy of the system. This specific function may be called synthetico-secretory. Certain cells like fat-cells have the function assigned to them of storing reserve material and liberating this material according to the requirements and economy of the

system, this reserve material being in most cases of exogenous origin as far as the particular storage cell is concerned. There are other specific functions performed by the cells of the body but from the point of consideration of the problem of diabetic metabolism these functions are unimportant. From this point of view the consideration of the metabolic processes and activities and the conditions of their action and the causal nexus involved in the performance of these specific functions enumerated above is most important.

(3) *The Cytoplasm and Nucleus—Two distinct functional units; The Cytoplasm, the organ performing the specific function of the cell; The Specific necessity of D-glucose for functional energetic activity:—*

Histologically each individual cell consists of two distinct anatomical units, the cytoplasm and the nucleus. Physiologically as each of these histological units performs a specific function quite distinct from that of the other, each of these units must be considered to be distinct separate physiological functional units. The cytoplasm of the cell is the specific organ for the performance of the specific functional activity, the cell as a whole contributes to the economy of the system. The nucleus, on the other hand, has a specific synthetico-secretory trophic function with regard to the cytoplasmic substances. Thus with regard to cytoplasmic function in storage cells like fat cells, the site of the storage of the reserve material is the cytoplasm, this material being stored up and liberated according to the requirements of the system by the activity of certain cytoplasmic substances. In a cell with synthetico-secretory functions like the pepsin secreting cells of the gastric glands, pepsinogen the mother substance of pepsin is synthesized and deposited in the cytoplasm of the cell, and when required by the economy of the system is converted into pepsin and secreted into the gastric cavity either through the action of neuronic impulse or the hormonal stimulus of gastric secretin. So also in the neurone, the specific structure initiating and conducting the nervous impulse is the neurofibril, a cytoplasmic structure. Though the nerve fibre isolated from its cell body whether dendronic or axonic, (i.e., peripheral sensory or motor nerve fibre,) can conduct nervous impulses equally well in both directions, the complete neurone is endowed with the function of polarity; in a complete neurone a nervous impulse can only pass from the dendrons through the cell body into the axon. In the cell body and dendrons are Nissl's granules which are absent in the cell body in the neighbourhood of the axon hilum and in the axon also. It is probable that this peculiar distribution of Nissl's granules in relation to the dendron

cell body and axon is the physico-chemical basis of the polarity of the complete neurone, this peculiar cytoplasmic distribution of these granules facilitating the passage of impulses in one direction and blocking them in the other. Similarly in muscle fibres the specific contractile structures are the longitudinal fibrils or sarcofibrils found in the cytoplasm. Contraction of the sarcofibrils is attended with the conversion of potential chemical energy into kinetic energy. This kinetic energy liberated is obtained from the oxidation of d-glucose into carbon dioxide and water, and the cytoplasm of the muscle cell must be considered to be a specialized physico-chemical mechanism for the conversion of the kinetic thermal energy of the oxidation of d-glucose into mechanical energy. From the point of view of the study of diabetic metabolism the nature of the processes involved in this oxidation of d-glucose in the system is of importance and should therefore be considered at some length. D-glucose is the specific source of energy or fuel required by this physico-chemical mechanism of the muscle cell for its activity, for though a thousand substances on oxidation liberate energy, it is only through the oxidation of d-glucose and d-glucose alone, that the muscle can do mechanical work. The exact physico-chemical processes in this specific activity of the muscle cell requires elucidation. Lactic acid is the intermediate product in this oxidation of d-glucose, and perfusions of sarcolactic acid solution into a living muscle causes contracture of the muscle. Probably, therefore, the changes involved in contraction and relaxation of a muscle fibre are surface tension changes taking place in the substance of the sarcofibril or of portions of the sarcofibril: dependent upon the formation and oxidation of lactic acid, and the changes may be hypothetically worked out as follows. The first change is probably the conversion of one molecule of d-glucose into two molecules of lactic acid. This is not an oxidation but is a mere inter-molecular re-arrangement of atoms and is slightly exothermal and is probably effected by a special ferment present in the sarcoplasm which has the power of converting d-glucose, and d-glucose alone into lactic acid, and whose activity at least undoubtedly in cardiac muscle requires the presence of the calcium ion. The second change is the combination of lactic acid with the substance of the sarcofibril or of certain portions of the sarcofibril to form a molecular compound. Apparently the molecules of this new sarcofibril-lactic acid compound have special powers of molecular aggregation comparable for instance to that of the molecules of a stretched rubber strand and tend to come together with great energy producing shortening of the sarcofibril, and muscular contraction. This physical aggregation, the third change, is a powerful exothermal physical process comparable to that which occurs

when a stretched rubber strand shortens or when a crystal is formed in a supersaturated solution. If no resistance is offered to the contracture, the molecules come together with great kinetic energy, and a large amount of heat is liberated; but if resistance within physiological limits is offered, the molecules aggregate slowly in contracture and mechanical work is done. These three changes enumerated above associated with and causing contracture of the muscle are therefore attended with great liberation of energy. The succeeding changes are associated with and effect relaxation of the contraction. The fourth change is a reversal of the third and is consequently equally endo-thermal as the third is exo-thermal; the fifth change is a reversal of the second. The sixth and the last change is the oxidation of lactic acid in carbon dioxide and water, which change is probably effected by an oxidase ferment present in the sarcoplasm whose activity at least undoubtedly in cardiac muscle requires the presence of potassium ion. This last change is in itself powerfully exo-thermal and removes lactic acid from the sphere of the preceding reversible reactions and allows thus relaxation to be fully completed. But as the fourth change is strongly endothermal, the heat liberated in the three reactions associated with relaxation of the muscular contraction is very much less than that of simple oxidation of lactic acid. A considerable expenditure of mechanical energy also takes place when cells with the specific function of selective variable differential permeability perform their specific activity. Thus in salivary glands the fluid portion of the secretion is secreted at a greater pressure than the systolic pressure in the common carotid. The site of the performance of this function in these cells is also the cytoplasm, and the energy expended in this activity is also obtained from the oxidation of d-glucose, though the manner in which this energetic activity is effected requires elucidation. A sufficient supply of free d-glucose is an absolutely essential requisite condition for the functional activity of the cytoplasm of certain cells with energetic functions. If the requisite supply of this substance were to be cut off from the tissues, the muscles would be unable to contract, the capillary endothelium would allow the fluid portion of the blood to exude into the tissues, the kidneys would stop secreting urine and so on, and life would come to a standstill.

(4) *The Alternation in the cytoplasm of the phase of functional activity with the phase of reparative rest.*—

In the performance of specific cytoplasmic functions by these cells, the material of the cytoplasmic structures and substances necessary for the effecting of these functions becomes used up, and with the exhaustion

of the material of these substances and structures necessary for the performance of these functions the cytoplasm becomes physiologically exhausted and unable to perform its specific function until that material is reformed and renewed. Thus in a pepsin-secreting cell of the stomach after a period of continuous stimulation, all the pepsinogen granules would be converted into pepsin, and when this has taken place, the cell for some considerable time would be unable to perform its specific function, the secretion of pepsin, until it has reformed new granules of pepsinogen. So also in the cell body of a neurone after a prolonged period of activity, the Nissl's granules become chromatolyzed, and with this change the neurone becomes physiologically exhausted and unable to perform its specific function until the granules are reformed. So too in a muscle cell after a sufficiently long period of intensive activity, the sarcoplasmic ferments responsible for the conversion of glucose into lactic acid and of lactic acid into carbon dioxide and water would become used up, and in the absence of these necessary ferments the muscle would be incapable of activity becoming exhausted by prolonged activity. The more intensive and prolonged the period of activity, the quicker and more complete is such exhaustion. The physio-chemical basis of this type of physiological loss of function is quite distinct from that which occurs from accumulation of katabolites in the tissue or from anoxaemia or hypoglycaemia. Thus in the cytoplasm of all kinds of active functioning tissues, after a period of intense specific activity, comes naturally a phase of functional exhaustion. If the conditions of the nutrition of the system is normal, specific synthetic ferments originating as we shall see later from nuclear metabolism commence actively, as soon as specific cytoplasmic activity has ceased, synthesizing and depositing in the cytoplasm the material of the various active cytoplasmic substances and structures necessary for the manifestation of specific cytoplasmic function. Thus in peptic cells, pepsinogen granules are reformed and kept in readiness for the next meal. In nerve cells Nissl's granules are reformed, and so on in other cells. Thus in the cytoplasm of all active tissues, there is in normal conditions an alternation of a phase of functional activity attended with wear and tear and katabolism with a phase of exhaustion, rest and repair, during which second phase if nutritive conditions are normal active anabolic synthetic changes take place in the cytoplasm reforming the cytoplasmic substances necessary for cytoplasmic activity. This alternation of phase is most manifest in certain tissues whose activities are intermittent, being only required at certain times, for instance,

voluntary muscle and loco-motor nervous system. Certain tissues as the vaso-motor and respiratory-motor systems must however be continually active. In these systems specific cytoplasmic functional activity and cytoplasmic reparative changes normally go on simultaneously without exhaustion occurring; but under abnormal circumstances exhaustion may be induced as in other tissues. Thus if the vaso-motor system be kept continuously stimulated without rest by repeated maximal doses of adrenalin, dilatation of the heart and other signs of vaso-motor exhaustion will set in after some time. The current theory that in cardiac muscle the systole is the period of katabolism and the diastole the period of anabolism is apparently incorrect. Anabolic changes like the synthesis of sarcoplasmic ferments probably go on equally rapidly in systole as in diastole.

(5) *The nature of nuclear metabolism.*—

It must be supposed that the cytoplasmic substances originate through the synthetic action of specific synthetic ferments. It must be asked whence do these synthetic ferments themselves originate. It must be logically supposed that these synthetic ferments are formed by the synthetic action of other synthetics, ferment, and that these other synthetic ferments themselves perhaps originate through the synthetic action of a third genealogical group of synthetic ferments. It is impossible that such a genealogical chain of synthetic ferments is infinitely long. Finally, we must come to the head of this genealogical line of intracellular ferments and we must logically postulate in the cell, the existence of some synthetic ferments which are the prime origin of this chain of synthetic ferments, through the synthetic action of which chain of ferments finally the active cytoplasmic substances are formed. To these prime synthetic ferments we must also logically assign the property of auto-synthesis. These prime synthetic ferments must also be bodies common to and therefore found in all cells, and since obviously the integrity and continuity of the vital function depends on their integrity and continuity, these bodies must be fixed continuous and persistent throughout all the changes the cell undergoes, and we should expect to find in them changes of a peculiar nature well adapted to ensure their continuity and integrity in the formation of new cells and in the gametocytic generation of new organisms. It is within the nucleus that we must look for these prime synthetic ferments, as experimental facts show that the presence of the nucleus is an essential condition required for the completeness and continuity of the processes of cytoplasmic synthesis, and in the nucleus we find the substances of the chromoplasmic structures (the chromosomes and chromatin network) which undergo

peculiar changes in homotypical and heterotypical mitosis to be bodies fulfilling all the theoretical requirement postulated for bodies of the nature of cellular primal synthetic ferments. Nuclear metabolism must therefore be regarded as reactions involving the synthesis of a genealogical chain of synthetic ferments commencing with the nuclear chromoplasm and ending with the ferments synthesizing the chromoplasmic substances. These nuclear synthetic reactions are physiologically quite distinct from the cytoplasmic reactions. Just as the cell membrane has specific functions with regard to the cytoplasm separating the cytoplasmic substances and the sphere of their reactions from the substances and reactions of the extracellular medium and regulating the passage of substances between the cytoplasm and the extra-cellular medium in their proper occasion and quantity according to the requirements of the organism, so also the nuclear membrane, a structure probably also of lipoid constitution, has similar functions as regards the nucleoplasm separating the nuclear substances and the sphere of their interactions from the cytoplasm and its reactions and regulating the passage of synthetic ferments from the nucleus into the cytoplasm according to the metabolic needs of the cell. Such a membrane forms a cell within a cell, and by its presence the nucleus physiologically a functional unit quite distinct in specific function from the cytoplasm becomes a distinct anatomical unit also. These active nuclear synthetic ferments also go through the same alternation of phases of specific activity and of exhaustion with reparative processes as the active cytoplasmic substances, being used up in performing their specific synthetic activity and requiring therefore reformation and replacement. These processes are probably as follows: The first stage is the auto synthesis of the basi-chromatin or the chromoplasm of the so-called chromatin network, which chromoplasm as considered already is the prime synthetic ferment of the cell. At the end of this stage the chromoplasm is in maximum quantity and the nucleus consequently stains most intensely with basic dyes. The next stage is the synthesis by the chromoplasm of the substances of the next synthetic ferment in this genealogical chain of ferments. The substance of this ferment must be identified with the substance of the nuclear oxyphil karyoplasm. As the chromoplasm is called the primary synthetic ferment, this ferment may be called the secondary cellular synthetic ferment. The next stage in nuclear activity is the secretion or passage of this oxyphil karyoplasm from the nucleus into the cytoplasm through a gap in the nuclear membrane. After it has passed into the cytoplasm this secondary synthetic ferment takes on its specific synthetic activity, either synthesizing itself directly the substances of the cytoplasmic structures or more probably synthesizing an intermediary tertiary synthetic ferment

which then in turn synthesizes the specific cytoplasmic substances. We may thus understand how the cell consists of two distinct functional and anatomical units, the nucleus and the cytoplasm, the cytoplasm being the organ which performs the specific function of the cell to the general economy of the system, while the nucleus is a synthetico-secretory unit forming and secreting ferments synthesizing the cytoplasmic substances, having thus the same function in all cells.

(6) *The stimulant effect of cytoplasmic activity on nuclear function and cytoplasmic tone.*—

Whereas the specific function assigned to nuclear metabolism is the maintenance and repair of the functional efficiency and tone of the cytoplasm by synthesis of the active cytoplasmic substances and structures taking the effective part in performance of specific cytoplasmic function, the specific functional activity of the cytoplasm has in its turn in normal conditions, a stimulant trophic effect on nuclear function and through nuclear function on cytoplasmic functional efficiency and tone. Thus if a whole limb is immobilized by splints, the muscles atrophy. If, on the contrary, the muscles are well exercised with adequate rest and in normal conditions with a good supply of nutritive material the muscles undergo hypertrophy and hyperplasia and increase in functional efficiency and tone. Over stimulation without adequate rest causes exhaustion and loss of tone as long as it is kept up and if occurring in vital tissue means death. Just as we have seen that the cytoplasmic functional activity of muscle cells and other active functional energetic units require for efficient functioning a good supply of d-glucose as the source of their functional energy, so the nuclear and cytoplasmic synthetic ferments require for their functional efficiency a good supply of various amino acids as the material out of which to effect synthesis of the nuclear and cytoplasmic substances. In long continued starvation not only is the cytoplasm wanting in functional tone through deficient synthesis of cytoplasmic substances, but the cytoplasmic synthetic ferments originating from the nucleus take upon themselves lytic activities in the non-vital tissues, the vital tissues in this way obtaining nutriment from and at the expense of the non-vital tissues. Thus we see that intensive cytoplasmic functional activity, if followed by an adequate period of reparative rest and in conditions of good nutrition, results in increase of functional efficiency both of cytoplasm and nucleus. Under such conditions the active tissues not only increase in functional tone, but if necessary certain tissues respond to the increased demand on their

function by hypertrophy and hyperplasia. Disuse causes however hypotonus and atrophy, both of which also result from over stimulation without adequate rest and repair or from malnutrition.

(7) *The effect of Toxic Cellular Degeneration on Cellular Functions.*—

Normal performance of the normal cellular function by the cytoplasm and nucleus presupposes a normal physico-chemical condition of the nucleoplasmic and cytoplasmic structures and substances. Certain toxic substances of inorganic and organic origin if allowed to act upon the cell substances may directly cause abnormal physico-chemical changes in them or alter the nature of the cell substances by causing toxic alteration of the normal processes of cellular synthesis. Thus arsenic causes fatty degeneration; bacterial toxins may cause lardaceous degeneration. In this cellular degeneration there is a loss of the normal functions and activities of the cell involving both specific cytoplasmic functional activity and cellular synthesis. The cell that has undergone degeneration will not respond to various physiological stimuli in the same manner as a normal cell. If any part of the nuclear chromoplasm has undergone complete degeneration, loss of vital function is permanent, a portion of the primary synthetic ferments absolutely necessary for the continuity and integrity of the vital function being destroyed. If the nuclear substances are unaffected and the degeneration is only confined to the cytoplasm the cell may be able to regain its normal function and activity in time by causing resolution of the degenerative changes and refashioning of its normal physico-chemical structures. Loss of function due to toxic cellular degeneration must be distinguished from that which occurs from physiological exhaustion, hypoglycaemia, anoxaemia, or accumulation of katabolites. In the last four conditions the cell substances are in a normal physico-chemical condition. In cellular degeneration there is abnormal physico-chemical alteration of the cell substances.

(8) *Division of Active Tissues in Two Systems, the Dynamic Locomotor and the Nutritive or Digestive; the Phase of Cytoplasmic Activity in any one System simultaneously associated with Inhibition in the Other.*—

Practically all the active tissues of a vertebrate system may be more or less accurately divided into two distinct systems, such that the phase of intense cytoplasmic functional activity in any one of these systems is contemporaneously associated with a phase of equally intense inhibition and rest from specific cytoplasmic function in the other. Thus in the struggle for life for the maintenance of its own individual existence the vertebrate animal is called upon at times to put forth a great expenditure of co-ordinated dynamic or muscular energy. Thus in chasing and killing its prey,

devouring its food, fighting against or escaping from its enemies, there is in the system of the vertebrate animal an intensive cytoplasmic activity of certain sensory and organs, certain portions of his central and autonomic nervous system, practically the whole of its voluntary muscular system, certain involuntary muscles and certain endothelial cells and certain internal and externally secreting glands. During such periods there is equally intense inhibition of the activities of the whole of the alimentary canal, associated glands, nervous, muscular, and other tissues from the cardiac sphincter downwards with strong contraction of certain sphincters from the incisura angularis of the stomach downwards. Again, when digestion is in progress, for instance after a heavy meal there is intensive activity of the glands of the stomach, small and large intestine, the muscle of the gastric and the intestinal wall, gall bladder and ducts, and of certain portions of the central and autonomic system, etc. During this period of digestion there is considerable inhibition of the functional activities of the first system of tissues causing lethargy and disinclination to intense activity of the voluntary locomotor system. If the meal is large enough, this inhibition may be intensive enough to cause complete suspension of the voluntary activities of this system resulting naturally in sleep. We may thus practically divide nearly all the active tissues into two distinct systems such that the phase of specific cytoplasmic activity in one system is reciprocally associated with the phase of rest and inhibition in the other. The function of such co-ordinated activities of the first system of tissues is to effect a suitable response in the system of the animal to external circumstances in which purposive response to the co-ordinated exhibition of dynamic or muscular force is the dominating feature. Hence this system may be called dynamic locomotor or simply dynamic for short. The function of the activity of the second system is not to bring the animal in response to its external environment but to effect digestion, absorption, and internal storage of food materials. Hence this system may be called digestive or nutritive. Inhibition of functional cytoplasmic activity in any one of these two systems associated with internal functional activity in the other must be distinguished from loss of functional tone and activity from exhaustion, such inhibition of cytoplasmic function being due not to exhaustion of the store of active cytoplasmic substances, but to diminished irritability of these functional structures. Nor does this inhibition of cytoplasmic function mean suspension of all metabolic activities in the cell; on the contrary, as shall be seen later, this inhibition of cytoplasmic activity is attended with strong stimulation of reparative cellulo-synthetic processes on the cytoplasmic functional structures by synthetic ferments derived from the nucleus.

(9) *The active phase of each of these two systems associated with the secretion and action of certain hormones, the action of the hormones of one system being antagonistic to that of the other.*—

The active phase of each of these two systems is associated with the secretion or liberation from certain synthetico-secretory cells and the action on the system of certain active substances called hormones. With dynamic activities the hormone associated is adrenalin from the chromaffine tissues; with activities of the nutritive system the hormones associated are gastric and duodenal secretions from the pyloric and duodenal mucous membranes and insulin from the pancreatic islets of Langerhans. The action of these hormones play a great part in effecting and co-ordinating the metabolic processes associated with activity and inhibition respectively of these two different systems of tissues. As should be expected, the action of the hormones of one system opposes the action of the hormones of the other system.

(10) *The Action of Adrenalin, the Hormone of the Dynamic System.*—

Adrenalin, the hormone of the dynamic system, has a powerful stimulant effect on the irritability to stimuli of the nervous, muscular, glandular and other tissues of the dynamic system and on the strength of their response to excitation. On the tissues of the nutritive system adrenalin has an inhibitory effect both as to their excitability and the strength of their response to stimuli. Adrenalin causes constriction of the blood vessels of the stomach, small and large intestine, gall bladder, pancreas, etc. diminishing the blood supply to these organs. It causes inhibition of the gastric intestinal, pancreatic and biliary secretions. It causes inhibition of the peristalsis of the stomach intestines, of all bladder and ducts with strong contraction of sphincters from the gastric incisura angularis downwards. There is no doubt that it has an inhibitory effect on the activity of the nervous tissues both central and autonomic whose activity is associated with and co-ordinates the activity of the digestive system. It has an inhibitory effect on the secretion of the nutritive system hormones, gastric and duodenal secretions and pancreatic insulin by their respective synthetico-secretory cells. On the tissues of the dynamic system adrenalin has the opposite effect as already noted. Inhibiting the contraction of the transverse muscular fibres in the bronchial mucous membrane during expiration, it gives freest ingress and egress to air which with deepening and quickening of respiration results in maximum oxygenation of the blood so necessary for the activities of the dynamic tissues. Acting on the liver cells, it stimulates them to greater liberation of d-glucose from stored up glycogen and fat and so increases the concentration of this substance in the blood. It stimulates the heart to beat more forcibly and rapidly, and by its conjoint action on the capillary endothelium

raises blood pressure and increases circulatory activity. On the blood vessels of the dynamic system, if adrenalin has no vaso-dilator effect, it has at least a vaso-constrictor effect much less powerful than the same effect on the intestinal and associated blood vessels, so that though, notwithstanding the higher blood pressure, the intestinal blood vessels are constricted and their blood flow reduced, with the higher blood pressure the dynamic tissue blood vessels are opened out and an increased blood supply with necessary oxygen and d-glucose sent through them.

(11) *The Action of gastric and duodenal Secretions.*

On the gastro-intestinal tract and associated tissues of the nutritive or digestive system, the action of the digestive system hormones gastric and duodenal secretions is the opposite of that adrenalin. These hormones cause hyperaemia of these tissues through vaso-dilation of their blood vessels. Through their stimulant effect on the nervous, muscular, glandular, and other tissues of this system there is increased secretion of various digestive secretions, and increased peristalsis, and as a result of these activities foodstuffs ingested are digested. After digestion absorption of the products of digestion takes place through the striated and bordered epithelium lining the villi and the crypts of Lieberkuhns and other factors come into play. On the tissues of the dynamic system these digestive hormones have apparently an inhibitory action as regards their specific cytoplasmic function which inhibition after a heavy meal may be so intense as to cause complete suspension of their activities in sleep. This inhibition is usually attributed to relative anaemia caused passively by drain of blood to the digestive system. Just as we have attributed the relative anaemia of the intestinal tissues during the action of adrenalin to vaso-constriction, so we may also ascribe this relative anaemia of the tissues of the dynamic system and during digestion to be partially due to active vaso-constriction probably effected by the action of the digestive hormones on those blood vessels. It is not improbable that the digestive hormones have also on the other tissues of dynamic system a similar inhibitory effect as adrenalin has on the tissues of the nutritive system.

(12) *Inhibition of Cytoplasmic Function Caused by these Hormones Attended with Stimulation of Reparative Cellulo-synthetic Processes in the Inhibited Tissues.—*

Inhibition of the functional activity of the tissues of one of these two systems caused by the hormone stimulating intense cytoplasmic activity in the other system does not imply total suspension of metabolic activity and cellular processes in the tissues of the inhibited system. The phase of rest from specific functional activity in the cytoplasm is, as seen already,

the period of reparative cytoplasmic synthesis. We should therefore expect that such inhibition of the cytoplasm from specific function is associated with the processes of reparative synthesis of cytoplasmic structures. There is even reason to believe that a hormone stimulating the tissues of one system to intensive cytoplasmic function and inhibiting the cytoplasmic activity of the other system actually stimulates intensively reparative cellulo-synthetic processes in the inhibited system. Thus in man after a period of intense muscular activity within physiological limits, not only is the appetite keener and the individual able to ingest a larger quantity of food, but the digestive capacity of the nutritive system is increased and the digestion of the larger meal is more complete than otherwise. The better digestion of a larger meal implies greater functional tone and activity of the tissues of the digestive system. This greater functional tone is, as already considered, caused by greater trophic cellulo-synthetic activity of the nuclear functional unit and its ferments. This intensive reparative cellulo-synthetic action in the digestive tissues takes place during the phase of intense functional cytoplasmic activity of the tissues of the dynamic system. Hence we may argue that the factor associated with intensive cytoplasmic activity in the dynamic tissues causes proportionally intensive cellulo-synthetic processes in the tissues of the nutritive system. This factor causing this intensive cellulo-synthetic effect can be none other than the dynamic hormone adrenalin. Reciprocally it seems that the digestive system hormones, the secretins have a similar cellulo-synthetic stimulant effect on the tissues of the dynamic system. After digestion of a good meal a man certainly not only feels more fit, but is actually more capable of doing active muscular work than in a starving condition. Further the functional tone and efficiency of the muscles of a sedentary individual with ample diet is certainly better than that of another sedentary individual who is able to obtain only a meagre diet. So too of two strenuous muscular workers, the one who has an ample supply of food will certainly be stronger than another with a sparse supply of nourishment. Arguing from the analogy of the stimulant effect of adrenalin on the cellulo-synthetic processes in the nutritive tissues, it is not improbable therefore that the digestive system hormones, the secretins have a stimulant effect on the activity of cellulo-synthetic processes in the tissues of the dynamic system. This cellulo-synthetic stimulant effect of these hormones probably explains why hypo-function of the islets of Langerhans is much more frequent in sedentary individuals than in individuals who lead a strenuous muscular life. For as insular tissue is part of the nutritive system a strenuous muscular life stimulates this tissue to greater reparative cellulo-synthetic

activity causing thereby greater functional tone and efficiency. We may therefore conclude that the dynamic and digestive system hormones which inhibit the cytoplasmic activity of the digestive and dynamic systems respectively, intensively stimulate reparative cellulo synthetic activity in the cells whose specific function they inhibit. It is perhaps important to note that hormones may have a stimulant action not only on the specific cytoplasmic functional activity of the cells but also on reparative cellulo synthetic processes. There can be no doubt that this later action of hormones helps greatly in maintaining the normal functional efficiency, tone and metabolism of the tissues.

(13) *The necessity for storage in the system of the products of digestion.*—

As a result of the activities effected by the digestive system hormones gastric and duodenal secretions, proteins are hydrolized in the alimentary canal to amino-acids, carbohydrates to hexoses, and fats to fatty acids and glycerine. These products are absorbed into the system through the epithelial lining of the intestines through the activity of its component cells. Amino-acids supply the material necessary for nuclear and cytoplasmic synthesis. Assimilable sugars like fructose, mannose and galactose are converted by intermolecular atomic re-arrangement into d-glucose. D-glucose whether originating from this atomic re-arrangement or absorbed directly from the alimentary canal is the absolutely necessary substance required by certain cells as the source of the energy they manifest in their functional activity. Fatty acid and glycerine are destined to be transformed later on in the liver in d-glucose and to be used up as such.

[A certain small portion of fat and d-glucose is required for cellulo-synthetic purposes, and amino-acids not required for cellulo-synthetic purposes are de-aminated and the de-aminated portion converted into d-glucose.]

Though a supply of amino-acids and d-glucose are required by the cell for its synthetic and energetic requirements respectively, these substances are not required continuously by the cell but only during its phases of cellular synthesis and specific functional activity respectively. Moreover these crystalloid substances are absorbed into the system in large quantities during digestion only and if allowed to remain in crystalloid solution in the blood and tissues would cause extreme irregularity of the osmoticity of the blood and tissue fluids which would be highly unphysiological as it would interfere with the regularity of the physico-chemical conditions required for the uniform working out of the tissue metabolic physico-chemical changes. Further when these crystalloids are present in the blood in strong crystalloid solution concentration, the kidney becomes excessively permeable to them, and by passing into the urine they would be lost to the system almost

as rapidly as they are absorbed. Some mechanism is therefore needed to prevent these substances remaining in the blood and tissue fluids in strong crystalloid solution and to conserve them for the needs of the system and to present them to the tissues in crystalloid solution when the tissues need them and in quantities according to their metabolic requirements. This mechanism is provided by the reversible action of certain ferments present in certain cells and tissue fluids (blood and lymph) which have the power of converting amino-acids into proteins, and glucose into glycogen and *vice versa*. Fatty acid and glycerine are required by the liver cells only when there is a need for endogenous glucose formation, that is, as shall be seen later, when the hepatic glycogen store becomes depleted. The same arguments against the pressure of amino-acids and glucose in strong solution in tissue fluids apply against the presence of free glycerine and fatty acid in the blood and tissue fluids in strong crystalloid solution. Moreover both free glycerine and fatty acids, if present in strong crystalloid solution in the blood, would act as toxic bodies. Glycerine is an organic solvent; fatty acids are acidic. Hence after digestive absorption there is an absolute necessity that these two substances should be prevented from remaining in the system in free solution. This prevention is effected by the action of ferments present in certain cells and in the blood and lymph which have the power of converting fatty acid and glycerine into fats. By their reversible action fat is transported to the liver when required for endogenous glucose formation. The activities of these ferments effecting the conservation of amino-acids, d-glucose and fat are controlled by the action of certain hormones in accordance with the metabolic requirements of the system.

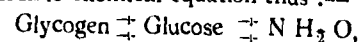
(14) *The Various Processes Effecting Alteration in the Concentration of free Glucose in the System.*—

A molecule of d-glucose found at any time free in the blood and tissue fluids may originate in a twofold primary manner. It may originate from the absorption of carbohydrates digested in the alimentary canal, other assimilable sugars being converted by intermolecular re-arrangement into d-glucose. Glucose originating in this manner may be called glucose of exogenous origin. The liver has the function of converting fats chiefly, and amino-acids also after de-amination, into d-glucose; glucose originating in this manner may be called glucose of endogenous origin. There is a third source of an intermediate nature, the liberation of d-glucose from glycogen stored up in the liver and other cells, which glycogen itself originated from the storage of glucose of primary endogenous or exogenous origin. Whatever its origin, a molecule of d-glucose present free in the system may meet immediately with four fates. It may be oxidized in the

muscles and other cells to carbon dioxide and water and thus be destroyed in the system. It may pass out into the urine and thus be totally lost to the system. It may be transformed by dehydrolysis in the liver and other cells into glycogen, or it may be converted in the liver cells into fat. Three processes occurring in the system increase, and four processes decrease the amount of free glucose present in the system, the total amount and concentration of free glucose in the tissues at any time being determined by the relative action of those seven processes. Three of these processes are determined by conditions not directly under the internal metabolic control of the system. The amount of endogenous glucose absorbed during digestion is determined by the quantity and quality of food the animal is able to obtain. The amount of sugar oxidised is regulated by the energetic and thermic requirements of the system in response to its external environment and its other necessities. The amount of glycosuria taking place is primarily determined chiefly by pathological lesions and alteration of normal function in insular tissue, or the pituitary and other tissues. Provided the liver cells are in a normal physiological physico-chemical and are not in a condition of toxic cellular degeneration, the system has in normal conditions a perfect complete automatic control over four intrahepatic processes, the transformations of fat into glucose and of glucose into fats and the hydrolysis of glycogen into glucose and the dehydrolysis of glucose into glycogen, and by regulating the activity of these processes determines the concentration of free d-glucose in the blood and tissues in accordance with the activity of the uncontrolled processes.

(15) *Conditions controlling the conversion of glucose into glycogen or of glycogen into glucose in the hepatic cells and the blood glucose concentration.*—

The conversion of free glucose into glycogen and the liberation of free glucose from stored up glycogen in the liver cells is effected by the action of an amylase ferment present in the hepatic cells, the action of this ferment being reversible. By dehydrolizing glucose it stores up glycogen, and by hydrolizing glycogen it liberates glucose. Like all reversible reactions, the action of this ferment has its equilibrium point which varies under different conditions of action. These reversible reactions may be represented by a reversible chemical equation thus :—



and in a particular condition of action with a certain concentration of free glucose in the liver cells, these two reversible actions would balance one another. This concentration in which the two reactions, glucose into glycogen, and glycogen into glucose, balance one another is the equilibrium concentration and will be called the hepatic glucose—

glycogen equilibrium concentration or simply the hepatic equilibrium concentration for short. The action of adrenalin is to change the speeds of these two interactions, slowing the glucose into glycogen interaction and hastening the glycogen into glucose interaction, thus raising the hepatic equilibrium concentration. The action of insulin on the speed of these interactions is the reverse of that of adrenalin, thus lowering the hepatic equilibrium concentration. The action of these hormones on the hepatic glucose equilibrium concentration is antagonistic and the intensity of their effect is proportional to their respective concentration in the system. Whatever the hepatic equilibrium concentration, if the concentration of d glucose in the liver cells is greater than the hepatic equilibrium concentrations, glucose is converted into glycogen until the hepatic glucose concentration becomes equal to the equilibrium concentration. If the concentration of free glucose in the hepatic cells is less than the hepatic equilibrium concentration, glycogen is converted into free glucose until the free glucose concentration in the liver cells becomes equal to the hepatic equilibrium concentration. The concentration of free glucose in the liver cell naturally therefore tends to approximate as close as possible to the hepatic equilibrium concentration at a particular time and is thus determined by it. The hepatic glucose equilibrium concentration also controls and determines the blood glucose concentration. Blood brought to the liver by the portal vein and hepatic artery comes in intimate contact with the hepatic cells with the natural result that there is freest interchange by osmotic diffusion between the hepatic blood and the liver cells. If the concentration of d-glucose in the hepatic advected blood (that is the blood brought to the liver cells by the hepatic artery and portal vein) is greater than the hepatic equilibrium concentration glucose would naturally diffuse from the hepatic blood into the liver cells thereby raising the glucose concentration in the liver cells. Rise in the hepatic glucose concentration above the hepatic glucose equilibrium concentration would mean increased conversion of glucose into glycogen. In certain conditions it would also mean transformation of glucose into fat. Both (in certain conditions only one) of these processes would continue till the blood glucose concentration has become equal to the hepatic equilibrium concentration. If the glucose concentration in the hepatic blood is less than the hepatic glucose equilibrium concentration, sugar would naturally diffuse from the hepatic cells into the blood. This diffusion would cause diminution in the glucose concentration which diminution would cause conversion of glycogen into glucose. In certain conditions it would also cause transformation of fat into glucose. Both (in certain conditions only one) of these processes would continue till the glucose concentration in the

hepatic advected blood is equal to the hepatic glucose equilibrium concentration. The hepatic glucose equilibrium concentration thus regulates and controls the blood glucose concentration tending to approximate the blood concentration as close as possible to the hepatic equilibrium concentration. The hormones adrenalin and insulin by regulating the hepatic glucose equilibrium concentration control the blood glucose concentration.

(16) *The Hormonic Position of Insulin; the Regulation of the Secretion of Insulin.*—

During digestion of carbohydrates a large amount of glucose is absorbed into the intestinal vessels and carried in strong concentration by the portal vein to the liver where it is stored up as glycogen (and in some conditions converted into fat.) During this period of digestion the secretion of adrenalin is inhibited, and this inhibition aids in effecting this storage of glucose. It is also probable that there is during this period of absorption of intestinal glucose a natural physiological increased secretion and action of insulin, for it is just at this period there is maximum need of the action of insulin to prevent the flooding of the system with a concentrated solution of free d-glucose. The work of Bensley, Lane, Homan and Allen has shown that diabetic hyperglycaemia is attended with disappearance of the water-fixed granules from the insular cells. This disappearance is interpreted to mean that hyperglycaemia stimulates the insular cells to greater secretion of insulin and thus causes functional exhaustion in these cells. Presumably therefore during alimentary hyperglycaemia there is increased secretion and action of insulin, and as far as their phase of functional activity is concerned, the period of intensive cytoplasmic functional activity of the insular cells is contemporaneous with that of the tissues of the nutritive system. The following other reasons induce us to include insular tissue and insulin among the tissues and hormones of the nutritive system. The cells of the islets of Langerhans have a common developmental origin from the hypoblast as the externally secreting acinous pancreatic cells. Developmentally insular tissue belongs to the nutritive system. Painting the pancreas with a dilute solution of adrenalin causes hyperglycaemia presumably by inhibiting the secretion of insulin. Presumably therefore intensive activity of the dynamic system associated with increased secretion and action of adrenalin has an inhibitory effect on the specific secretory function of insular tissue just as it has on other tissues on the nutritive system. The action of insulin and adrenalin are antagonistic just as the actions of adrenalin and the secretions are. For these reasons we may associate insular tissue and insulin from the point of view of functional activity with the nutritive system tissues and hormones. Unlike the digestive hormones, the secretions,

whose period of secretion and activity is limited to the phase of active digestion, insulin must exert its influence on the hepatic cell continuously for instance even during starvation when the formation and action of the digestive hormones are suspended. In this respect insulin resembles adrenalin whose secretion and action though most intensive during the period of intense functional activity of the dynamic system, still continues, though in much lesser degrees at other times. Unlike adrenalin which is quickly destroyed in the system and whose action develops quickly and then ceases after a short while from the destruction of the drug, the effect of a maximum physiological dose of adrenalin developing in one or two minutes and lasting for twenty to thirty minutes, insulin is very slowly destroyed or excreted and is by far much more prolonged in its action. The effect of a single physiological dose of insulin after developing in half an hour may last even from twelve to twenty four hours. Once secreted the action of insulin may last for a considerable length of time. Presumably therefore, as regards their specific function, the hormones of the nutritive system may be classified into two groups, the digestive and the conservative. The digestive hormones, gastric and duodenal secretions, effect digestion of food materials in the intestinal canal, and their period of activity is discontinuous being confined only to the period of digestion. The activity of the conservative group comes into action after absorption of the products of digestion. Their function is to prevent flooding of the system with the crystalloid products of digestion by synthesizing them into reverse materials of high molecular weight and low osmotic diffusibility, inhibiting thus the untoward effects of this flooding and to conserve the products of digestion after absorption for the needs of the system. Their activity though maximal during the period of digestion and absorption must continue also throughout the period during which digestion and the activity of the digestive hormones is in abeyance. Insulin belongs to this class of conservative hormones of which possibly there may be others with functions affecting other food-stuffs. The function of insulin is to conserve in the liver chiefly, glucose whether of exogenous or endogenous origin as glycogen for the systemic requirements, and its secretion though maximal during digestion and absorption of carbohydrates continues also at other times. More certain knowledge is needed of the factors stimulating secretion of insulin from the insular tissue and inhibiting this secretion and stimulating cellulose-synthetic processes in these cells. The condition causing increased secretion of insulin has been surmised by Homans, Allen and others on experimental data to be hyperglycaemia. It is not nervous impulse, for severance of nervous connection by excision of the whole of the pancreas is not followed by hyperglycaemia if a sufficient portion of the tail is implanted in the subcutaneous fat. Presumably it is

hormonic or hormonal-like, though it is not duodenal secretion as shown by Bensely and Homens. Neither presumably is there increased secretion of insulin in digestion and absorption of a meal in which carbohydrates are absent, as such an increased secretion in this condition would cause pathological hypoglycaemia. Presumably therefore, as already conjectured, it is the increased quantity of d-glucose in the systemic blood that occurs during the absorption of digested carbohydrates or that would automatically follow any lessening, through excretion or destruction of the action of the insulin previously secreted on the liver, the stimulus to the secretion of insulin being proportional to the degree of hyperglycaemia. Presumably also, as soon as a certain degree of hypoglycaemia is reached, the further secretion of insulin is inhibited, and the action of adrenalin on insular tissue even though attended with hyperglycaemia is not only to inhibit the secretion of insulin, but to stimulate actively reparative cellulo-synthetic processes in these cells. Further light must be thrown on these points by our experimental physiologists.

(17) *The nature of the effects produced by artificial hyperinsulinaemia.*—

Though it is easy to produce by artificial administration of excessive doses of adrenalin or insulin hyperadrenalinaemia or hyperinsulinaemia in such degrees as to be acutely incompatible with life, physiologically the liberation of these hormones by their respective synthetico secretory cells never reaches such pathological intensive degrees. An over dose of adrenalin would cause death by rupture of the cardiac muscle or of the arteriolar wall. The respiratory vaso-motor, thermotactic and other vital systems must, if life is to be maintained, continue to maintain a certain minimum degree of activity at all times even when the activities of both dynamic and digestive systems are suspended, for instance in sleep during starvation. The maintenance of this minimum degree of activity by these vital systems means the continuous oxidation of a certain amount of glucose and therefore requires the maintenance of a certain minimum concentration of d-glucose in the blood by a continuous liberation from the liver. If the concentration of d-glucose falls below this level, the vital tissues are unable to perform their specific function and death results. Artificial administration of insulin in doses large enough, unless attended with suitable corrective measures, would cause such lethal degrees of hypoglycaemia. The symptoms of such hypoglycaemia are caused partly by direct hypo-function of various tissues and partly by the irritative and other effects of exudate from the blood vessels forming in various organs through hypo-function of the capillary endothelium. The symptoms are therefore muscular weakness, feeble circulation and respiration, falling temperature, restlessness, convulsions, stupor coma etc. Such hypoglycaemia

can be prevented or cured by timely injections of adrenalin or by intravenous injections of suitable quantities of d-glucose or by oral administration of large quantities of carbohydrates in an easily absorbable form. Normally in life neither of these hormones is ever secreted in such intensive amounts as to cause such effects as there are natural physiological mechanisms automatically preventing such secretion. The mechanism preventing oversecretion of adrenalin is probably as follows: Increased secretion of adrenalin raises the blood pressure in the intracranial arteries; rise in intracranial pressure resulting from this rise in intracranial arterial blood pressure stimulates the vaso-depressive medullary centres which probably send down impulses to the adrenals inhibiting further secretion of adrenalin as soon as the hyperadrenalinaemia begins to reach the danger point. The mechanism preventing pathological hyperinsulinaemia is probably as follows: Increased systemic hyperglycaemia when not attended or caused by increased adrenalin secretion probably directly stimulates secretion of insulin, the intensity of the stimulus to secretion of insulin being proportional to the degree of systemic hyperglycaemia, and when the systemic glycaemia falls to a certain physiological level further secretion of insulin is inhibited and the danger of fatal hyperinsulinaemia averted. This physiological level must be some what higher than the minimum glucose blood concentration compatible with the maintenance of life.

(18) *The storage of glycogen in other tissues.*

Besides the liver, glucose is stored up as glycogen in small amounts in muscle cells, leucocytes, bone, skin, etc. In functionally active, energetic cells this small store of glycogen serves no doubt as a source of glucose for its specific energetic activity, being presumably converted into glucose under conditions that obtain in the cell during specific activity of the cell; but this store being small will not serve for prolonged intensive activity. In insular diabetes mellitus with attendant hyperglycaemia although the hepatic glycogen store is very low, that in the muscles is more than normal. Until we determine the conditions regulating muscular glycogen storage and liberation, it is impossible for us to explain this phenomenon, but presumably the storage of glycogen in the muscles is not controlled by various factors at work in insular diabetes in exactly the same manner as the storage of glycogen in the liver. Perfusion of an isolated muscle with glucose solution containing besides insulin causes greater increase in the muscular glycogen store with diminution of the glucose in the perfusion fluid than perfusion with glucose solution not containing insulin. This greater diminution of glucose in the perfusion fluid has been taken by some authorities to point that insulin causes a greater specific oxidative use of glucose presumably by stimulating the functional activity of the muscle. Insulin, however, causes a hypoglycaemia, and it must be pointed

out that it would be absolutely unphysiological for a hormone stimulating greater functional activity and oxidative use of d-glucose in the muscles and other tissues to diminish at the same time the supply of glucose absolutely necessary for the performance of that activity.

(19) *The Liver the Organ for the endogenous formation of glucose and fat; the necessity for the continual maintenance of hepatic glycogen store; Depletion of the hepatic glycogen store the condition stimulating endogenous glucose formation from fats; Saturation of the hepatic glycogen the condition stimulating endogenous fat formation from glucose.*—

The liver is not only the main organ in the body for the storage of glucose as glycogen, but it is the sole organ in the system having the glycosynthetic and liposynthetic functions, i. e., of the functions of converting fats and amino-acids into glucose and of converting glucose into fats. It is through exercise of this glyco-synthetic function by the liver that the glucose continually circulating in the blood originates when carbohydrates are cut off in the diet as in starvation, or in diabetes mellitus when exogenous glucose absorbed from the alimentary canal passes in a short time into the urine. If the liver is wholly excised, the haemic glucose concentration rapidly falls and death ensues. Not only must the liver supply to the tissues continually a certain quantity of glucose at all times even when carbohydrates are completely cut off in the diet, but it seems from a study of the conditions of life of the vertebrate animal in the natural struggle for existence, that it is an absolutely essential function of the liver that it should continuously keep up its store of glycogen to a certain physiological level. In the struggle for life the vertebrate animal may be called upon at any time to put forth a prolonged intensive activity of its dynamic tissues to maintain its individual existence. Thus a herbivorous animal, for instance, a deer, may be required at any time to save its life by a prolonged rapid flight from a pack of wolves; or a pack of starving wolves required to obtain their food by a prolonged chase of a herd of deer. Such a prolonged intensive activity of the dynamic tissues of the animal requires a prolonged intensive supply of glucose from the liver under the most favourable conditions possible. Though the liver can liberate into the system a continuously intensive supply of d-glucose from the transformation of fat into glucose, as long as the animal's store of fat lasts or till the animal dies of acidosis, such a supply would be attended with intense acidosis which would be a considerable handicap to the animal in the struggle for existence. Hence it is necessary that at all times the liver should keep up a sufficient stock of glycogen stored ready to supply the dynamic system tissues with the glucose they need in such emergencies under the most advantageous conditions possible. Thus it is that in the

mammalian and other vertebrate systems even in conditions when carbohydrates are completely cut off from the diet, in condition of normal non-diabetic metabolism the liver has always a considerable store of glycogen kept in reserve. Unless the system of an animal keeps such a reserve of glycogen, that particular animal would be considerably handicapped in the struggle for existence as compared with other animals and would soon be eliminated. Hence it becomes the imperative duty of the liver to maintain at all times its glycogen store to a certain necessary physiological level. Should at any time the glycogen store of the liver for any reason falls below this level, it becomes the imperative duty of the liver to replenish this store as soon as possible. The greater the depletion below this level, the more urgent is the call upon the liver to replenish this store. If exogenous glucose is presented to the liver, and the hepatic glucose equilibrium concentration is less than the haemic glucose concentration, the liver replenishes this store mainly by absorbing and converting this exogenous glucose into glycogen. If this is impossible, for instance, when carbohydrates are cut off in the diet, the liver must replenish this store by converting either fats or aminoacids into endogenous glucose and storing this endogenous glucose as glycogen. In these later conditions the greater the depletion of the hepatic glycogen store the more urgent and the greater is the need for endogenous glucose to replenish this store. Thus we see that when the hepatic glycogen store becomes depleted there is a natural physiological imperative demand on the liver to replenish that store by endogenous glucose formation, the greater the depletion the more urgent the necessity for the formation of endogenous glucose. The study of the condition of the hepatic glycogen store found in glycosuria shows us that the liver responds to this urgent necessity by an equally intensive endogenous glucose formation, the production of endogenous glucose in the liver being directly proportional to the degree of depletion of the glycogen store. As the liver is the main organ for storage of glycogen and the sole organ for endogenous glucose formation, in a case of continuous glycosuria during a period during which no absorption of exogenous glucose takes place from the alimentary canal, practically all the glucose passing into the urine originates entirely from the liver either through hydrolysis of glycogen into glucose or from endogenous glucose formation or from both. In such a condition the continuous glycosuria is rendered possible and takes place because the liver by hydrolysis of glycogen and by endogenous glucose formation from either fat or proteins continuously generates and liberates into the blood as much glucose as is simultaneously excreted into the urine and is oxidized in the system. The greater the degree of the continuous glycosuria therefore, the more equally intense becomes the simultaneous liberation of

glucose from endogenous glucose formation and from glycogen by the liver into the blood. If this sugar liberated continuously by the liver were only obtained by hydrolysis of glycogen, there would be a continuous process of depletion of the hepatic glycogen store which would continue till the glycogen store became completely exhausted and when further liberation of glucose from the liver would naturally stop. Such an event never takes place in glycosuria. In a case of continuous glycosuria, after the depletion of the hepatic glycogen store has reached a certain level, the depletion comes to a standstill and no further fall in the level of the glycogen store takes place, the glycosuria continuing as intensely as before. The degree to which the level of the glycogen store falls down before the process of depletion comes to a standstill varies directly as the intensity of the glycosuria. If the matter is carefully considered, only one explanation can be found to explain how the continuous glycosuria and liberation of glucose from the liver takes place when the depletion of the glycogen store comes to a stop, taking into consideration the fact that the degree of depletion reached is directly proportional to the intensity of the glycosuria; and that is, that depletion of the hepatic glycogen store stimulates the liver to endogenous glucose formation either from fat or proteins, the intensity of the stimulus to and of the process of endogenous glucose formation being directly proportional to the degree of the depletion of the glycogen store. What happens in these cases is this. The continuous glycosuria and oxidation of glucose in the system causes depletion of the hepatic glycogen reserve and as the process of depletion continues to increase, the intensity of endogenous glucose formation also increases till the depletion has reached such a degree that endogenous glucose formation is stimulated to such a degree as to supply all the glucose that is both being excreted in the urine and is being oxidized in the system. When this takes place, no further conversion of glycogen into glucose is required and the depletion of the hepatic glycogen reserve comes to a standstill. It must therefore be recognized that depletion of the hepatic glycogen reserve below a certain physiological level stimulates the liver to intensive endogenous glucose formation, the intensity of the stimulus being proportional to the degree of depletion below this level. Now when the liver is stimulated by depletion of its glycogen store to intensive endogenous glucose formation, the material transformed into glucose may be either amino-acids or fats. Amino-acids have to be de-aminated before transformation to glucose, and the ammonia liberated in this de-amination would induce a tendency to ammonaemia and alkalosis. Depletion of the glycogen store and the attendant endogenous glucose formation is always accompanied by acidosis which acidosis is proportional to the degree of depletion of the glycogen store and to the intensity of the endogenous glucose formation. Hence we are compelled to argue that when

the liver is stimulated by depletion of its glycogen store to intensive endogenous glucose formation, the material it is stimulated to transform into glucose cannot be amino-acids but must be mainly fats. More consideration will be given to this topic when we come to consider the genesis of diabetic acidosis and the conditions in which endogenous conversion of amino-acids into glucose takes place, but for the present we may take it that we have sufficient reason to assume that depletion of the hepatic glycogen reserve below a certain physiological level stimulates the liver to intensive endogenous transformation of fats into glucose, the intensity of the stimulus being directly proportional to the degree of depletion below this level. In the reverse condition of saturation of the hepatic glycogen store, the reverse phenomenon takes place, the liver being stimulated to the endogenous formation of fat from glucose and the intensity of the stimulus to endogenous fat formation being directly proportional to the degree of saturation of the glycogen store above the physiological level. Were it not for this transformation of glucose into fat when the system has an excessive supply of exogenous glucose to deal with as in the marmot just before hibernation the store of glycogen in the liver cells would become so great as to distend and perhaps mechanically rupture the liver cells. A good supply of excess of exogenous glucose absorbed from the alimentary canal is required for this transformation of glucose into fats. Depletion or saturation of the hepatic glycogen store below or above a certain physiological level therefore stimulates the liver to endogenous formation of glucose from fats and of fats from glucose respectively, the intensity of the stimulus being proportional to the degree of depletion or saturation. The recognition of this relation of the hepatic transformation of fat into glucose to the condition of the hepatic glycogen store is most important as it is the clue that leads us to understand the essential nature of the vitiation of metabolism in diabetes mellitus.

(20) *The intestinal absorptions of fat and the transformation from the intestinal canal to fat cells.—*

Fat must therefore be recognized as the main source of endogenous glucose in the body. A fat molecule present in the system may originate in two ways. It may be exogenous fat originating directly from absorption from the intestinal canal, or it may be endogenous fat derived from the transformation of glucose into fat in the liver. Whatever its origin, its purpose in the system is mainly to be transformed into glucose in the liver and so oxidized in the system as glucose for the functional energetic requirements of the cells. For this purpose fat is required in the liver cells only when endogenous glucose formation is taking place. At other times fat must be stored in reserve in the fat cells of lipid tissue. Intestinal absorption

and storage of fat is effected in the following manner. In the intestinal canal fat is hydrolized by pancreatic lipase into glycerine and fatty acid which are then absorbed by the epithelial cells of the small intestine. Within these cells these two substances meet another lipase which synthesizes them into fat. The action of this epithelial lipase is reversible, and fatty acid and glycerine resulting from the reversal of this synthetic action diffuses into the lacteals where they meet another lipase which resynthesizes them into globules of fat which are carried through the thoracic duct into the blood stream. The lipase present in the blood stream has also a reversible lytic action and fatty acid and glycerine resulting of the lytic action of this lipase passes into the fat cells of lipoid tissue where the lipase present in the fat cells synthesizes them into fat. This transportation of fat from the intestinal canal to the fat cells during digestion and absorption of fats is rendered possible by the fact that the concentration of free fatty acid and glycerine necessary for equilibrium of these two reversible reactions, fat into fatty acid and glycerine, and fatty acid and glycerine into fat which are effected by these lipase ferments diminishes progressively as we pass from the intestinal canal to the fat cells.

(21). *Transportation of fat from the fat cells to the liver or from the liver to the fat cells, the respective accompaniment of the endogenous formation of glucose from fat or of fat from glucose in the liver in the respective conditions of depletion or saturation of the hepatic glycogen store.*—

When either through oxidation of d-glucose in the body or through glycosuria the hepatic glycogen store is depleted beyond the physiological storage level, and exogenous glucose is not available or if available, cannot be converted into glycogen as the amount absorbed from the intestinal canal is not sufficient to raise the haemic glucose concentration above the hepatic glucose equilibrium concentration, the liver commences forming endogenous glucose in an endeavour to replenish the hepatic glycogen store to the physiological level. As considered in the last paragraph fat must be held to be the main source for endogenous glucose formed for this purpose. It is essential therefore that in these conditions a sufficient quantity of fat is supplied to the liver cells for transformation into glucose. Fat is therefore transported from the fat cells through the blood into the liver cells. The hepatic cells which contain a lipase enzyme, store up this fat and commence at once transforming it into glucose. The greater and more continuous the depletion of the glycogen reserve in the liver, the more intensive and continuous would be the necessity for hepatic endogenous glucose formation, and the more intensive and prolonged would be the transportation from the fat cells to the liver. Such transportation of fat, otherwise called "mobilization

of fat" is seen in its most typical form. In complete experimental extirpation of the pancreas. In this condition on account of the abolition of the physiological action of insulin on the liver cells, the hepatic glucose equilibrium concentration rises very high and the blood becomes extremely hyperglycaemic. As a result of this hyperglycaemia the kidney cells become excessively permeable to passage of glucose into the urine and large quantities of glucose pass continuously into the urine. This passage of glucose into the urine depletes the concentration of free glucose in the blood; the depletion of the concentration of free glucose in the blood in turn causes diminution in the concentration of free glucose in the liver cells which therefore falls very much below the hepatic glucose equilibrium concentration. This marked fall of the free glucose concentration in the hepatic cells markedly disturbs the equilibrium of the reversible reactions of the glucose-glycogen interactions and thus causes conversion of glycogen into glucose and depletion of the glycogen store. Depletion of the glycogen store continues until it reaches such a degree that the endogenous glucose formation from fat stimulated by the depletion is equal to the quantity of glucose simultaneously excreted into the urine oxidized in the tissues. When this degree of depletion is reached, the free endogenous glucose generated in the liver cell is equal to the glucose liberated by the liver cells into the blood and the concentration of free glucose in the hepatic cells automatically becomes equal to the hepatic glucose equilibrium concentration and no further depletion of the hepatic glycogen store takes place. Whereas the normal physiological hepatic glycogen store of the liver is about twenty per cent of the mass of the liver, in experimental excision of the pancreas, the hepatic glycogen becomes depleted to half per cent of the liver weight, the liver cells being infiltrated with fat as a result of the transportation of fat to the liver. The reverse processes of the endogenous formation of fat from glucose and the transportation of fat from the liver to the fat cells take place when, as for instance, in the mormot prior to hibernation, the hepatic glycogen store is saturated and there is excess of exogenous glucose absorbed from the alimentary canal, the intensity of these two coincident processes being jointly proportional to the degree of saturation of the liver with glycogen above the physiological level and the supply of exogenous glucose. The transportation of fat from the fat cells to the liver and in the reverse direction involves and is effected by alterations in the equilibrium concentrations of free fatty acid and glycerine in the reversible reactions effected by the lipases present in the fat cells, blood and hepatic cells. In the transportation of fat from the fat cells into the liver, this equilibrium concentration must progressively diminish as we pass from the fat cell to the liver. In transportation of fat in the opposite direction, the differences in

these equilibrium concentrations must be quite the reverse. These alterations in these equilibrium concentrations must be hormonal in causation, the hormone or hormones being probably formed by the liver cells. In a case of continuous glycosuria the degree of infiltration of the liver cells with fat is always proportional to the degree of depletion of the hepatic glycogen store and the intensity of the glycosuria. The exact manner in which depletion or saturation of the hepatic glycogen store stimulates endogenous transformation of glucose from fat and of fat from glucose with these coincident alterations in these equilibrium concentrations is a physiological problem requiring further experimental study.

(22) *The nature of the chemical changes involved in the endogenous conversion of fat into glucose and of glucose into fat.*—

The chemical changes involved in the endogenous conversion of glucose into fat is the exact reversal of the chemical changes involved in the endogenous conversion of fat into glucose. The latter changes are oxidative in nature and require a large supply of free oxygen. Fat is probably first hydrolyzed into glycerine and fatty acid. Two molecules of glycerine by oxidation with one molecule of oxygen are converted into two molecules of glyceric aldehyde which by condensation form d-glucose. A very large quantity of oxygen is required to oxidise the fatty acids of higher fats to glucose. Almost as many atoms of oxygen are required as there are atoms of carbon in the fatty acid if the whole of the fatty acid is to be converted completely to the final end products of this physiological conversion of fats into d-glucose, either d-glucose or d-glucose carbon dioxide or water whatever these final products of this hepatic transformation, if this transformation is to take place without any liberation of any of the intermediate products formed naturally in this endogenous conversion in the liver cells of fatty acid into glucose. It is probable that the same hepatic ferments that oxidise fat into glucose by a reversal of this oxidizing action reduce glucose to fat.

(23) *The Genesis of Diabetic Acido-Ketosis.*—

In proportion to its size and the intensity and importance of its metabolic processes and functions, the arterial supply of blood to the liver is comparatively small. Thus it is that when intensive endogenous conversion of fats into glucose takes place in the liver, the supply of oxygen to the liver cells becomes insufficient to oxidize the whole of the fat molecule to the final products of this hepatic conversion (either glucose, or glucose, carbon dioxide and water), and some of the intermediate bodies formed naturally in this transformation of fat into glucose are set free into the system. These intermediate substances are B-oxy-butyric acid, aceto-acetic acid and acetone. If these substances are produced in large amounts and

for prolonged periods and allowed to act upon the tissues, they produce toxic effects on the system. Acetone is a lipoid solvent and probably acts in the same manner as ether and chloroform first stimulating and then narcotizing the tissues, and perhaps also causes toxic cellular degeneration. B-oxybutyric and aceto-acetic acids being acids cause toxic effects by lowering the alkalinity of the blood and tissues acting in the same way as other mineral and organic acids. The intensity and duration of the liberation of these toxic substances is therefore proportional to the intensity and duration of the process of the endogenous transformation of fat into glucose which in turn depends upon the duration and degree of depletion of the hepatic glycogen store below the physiological storage level. Such depletion is caused by and depends on the intensity of two processes in the system, intensive oxidation of glucose in the system and the passage of glucose into the urine during such periods as when exogenous glucose is not being absorbed into the system in sufficient quantities. In the normal system especially under normal conditions of diet, the production of these intermediate bodies, if it at all occurs intensively, never continues for such periods as to cause marked toxic effects. As in the normal system glycosuria does not occur, the only factor causing acute depletion of the hepatic glycogen reserve would be intensive activity of the dynamic tissues causing intensive oxidation of d-glucose. Intensive activity of the dynamic tissues would demand an intense supply of d-glucose from the liver and if continued for sufficient time would cause intense depletion of the hepatic glycogen reserve. This depletion would stimulate intensive endogenous transformation of fat into glucose with accompanying intense acidosis. But this acute condition of depletion of the hepatic glycogen store with acidosis is only temporary. Sooner or later the intense activity of the dynamic tissues must cease from exhaustion, and when this happens there is no longer any demand or necessity for the liver to liberate into the blood stream as large quantities of sugar as before, and endogenous glucose formed in the liver as a result of the depletion of the glycogen store becomes available for transformation into glycogen, in this way raising the level of the glycogen store. As the level of the glycogen store rises, the stimulus to endogenous glucose formation from fat diminishes with consequent diminution in the formation and liberation of these intermediate toxic bodies, until finally when the hepatic glycogen store reaches the physiological storage level, (or rather more correctly reaches the physiological storage level as close as possible as allowed by the continuous need of the tissues for d-glucose) the stimulus to endogenous glucose formation from fat and the liberation of these intermediate substances is reduced to a minimum. In normal system, acute acidosis is only a temporary pheno-

menon occurring only during intense activity of the tissues of the dynamic system. Mild degrees of acidosis may be however observed in individuals who are not receiving in or absorbing from the diet a sufficient quantity of carbohydrates or amino acids. Such mild acidosis occurs in complete starvation resulting either from privation of food or from excessive vomiting or when kept on a diet consisting mainly of fat with insufficient quantities either of carbohydrates or of proteins. When sufficient quantities of exogenous glucose are not absorbed from the alimentary canal, the liver has to obtain the glucose which it liberates into the blood stream mainly from fat. Hence mild acidosis occurs in privation of carbohydrates from the diet. This acidosis can be rapidly checked by administration of suitable quantities of carbohydrates by the mouth or by intravenous infusions of d-glucose solution. So also in a starving individual or in a person whose diet consists entirely of fats, the liberation of these intermediate acidic and ketonic substances is greatly reduced if large amounts of exogenous amino-acids are absorbed from the alimentary canal. How this is effected we shall enter into later when we come to consider the metabolism of amino-acids. This mild degree of acidosis can be aggravated by any factor causing increased oxidation of d-glucose in the system, such as muscular exercise, administration of adrenalin, alcohol, strychnine in toxic dose, etc., fever, microbic and other toxæmias, etc. When glycosuria however occurs during a period when no exogenous glucose is being absorbed from the alimentary canal, the glucose passing into the urine is obtained, as already considered, from endogenous conversion of fats into sugar taking place in the liver, the glycogen store of the liver being depleted to such an extent, that the endogenous glucose formed and liberated by the liver into the blood stream, is equal to the amount continuously passing into the urine and being oxidized in the system. In these conditions a degree of acidosis proportional to the intensity of the glycosuria would ensue. In such a case of glycosuria if a sufficient quantity of exogenous glucose were administered, there would also be diminution in the production of these intermediate acidic substances, but as the glycosuria is continuous, this diminution would only be temporary being confined only to the period of administration of exogenous glucose. When in such cases of glycosuria exogenous glucose is absorbed into the system, it becomes at once available for four purposes, oxidation in the tissues, glycosuria, conversion into glycogen or transformation into fat according to the nature of the various processes taking place according to the condition of the system. If the rapidity of absorption of exogenous glucose into the system is intensive enough, it becomes available to supply part or the whole of the sugar

passing into the urine, and there is no longer the same drain of glucose from the liver into the blood. Endogenous glucose formed in the liver remains in the liver and becomes available for conversion into glycogen thus raising the level of the hepatic glycogen store and diminishing the intensity of the production of these intermediate substances. But this effect is only temporary in a case of continuous glycosuria lasting as long as the intensive absorption of exogenous glucose is taking place. Both in the severe acidosis resulting from continuous glycosuria and in the mild acidosis of carbohydrate privation the toxic effects of the intermediate acidic substance B-oxybutyric acid and aceto-acetic acid can be prevented by administration of suitable quantities of alkalies which act by neutralizing their acidity. In alimentary glycosuria which occurs in some cases during the period when large amounts of carbohydrates are being absorbed, as all the sugar passing into the urine is directly exogenous in origin, no acidosis takes place.

(24) *The Vicious Cycle of Diabetic Acidosis ; the Deviation of Amino acid from cellulose synthetic to antacid purposes caused by diabetic acidosis.*—

In these case of contiguous glycosuria when an intensive degree of acidosis is caused by the prolonged depletion of the hepatic glycogen store, a vicious cycle is generated leading to still greater acidosis. Intense acidosis stimulates the respiratory and vasomotor and other centres in the medulla and spinal cord which send down impulses to the adrenal glands, heart and blood vessels and skeletal muscles stimulating them to greater functional activity thus causing greater oxidative consumption of glucose. The action of the increased amount of adrenalin liberated by this stimulation is to raise the hepatic glucose equilibrium concentration and therefore the blood glucose concentration also. Rise in the blood glucose concentration may cause greater glycosuria than before. In these two ways by causing greater glycosuria and greater oxidative consumption of glucose, diabetic acidosis put an increased demand on the liver for greater liberation of sugar than before, in this way further depleting the hepatic glycogen reserve and stimulating still more intensively endogenous glucose formation from fat, which increased endogenous glucose formation would mean still greater production and liberation of the intermediate acidic and ketonic bodies. In conditions of continuous glycosuria, diabetic acidosis therefore creates a vicious cycle leading to still further acidosis. (A. the oxidative consumption of d-glucose stimulated by acidosis caused by depletion of the hepatic glycogen store is much less than the endogenous glucose formation stimulated by the depletion, in normal systems with no glycosuria, the level of the hepatic glycogen store rises to normal after depletion in spite of the acidosis.) The system endeavours to

neutralise the effect of these acid bodies on the alkalinity of the blood and tissues by de-aminating amino-acids and using the resulting ammonia to restore the alkalinity. In long continued intensive diabetic acidosis large quantities of amino acids needed for cellular synthesis are thus deviated for this antacid purpose. All tissues, except the liver, atrophy as amino acids required for cellular synthesis are not available. The liver in response to the extra demand on its functions undergoes physiological hypertrophy and hyperplasia. Loss of functional tone of the tissues in diabetes mellitus is partly due to atrophy from deficient cellular synthesis and partly to toxic cellular degeneration caused by ketosis.

(25) *The conservation of amino-acids in the plasma protein; the metabolism of amino-acids.*

If the function of glucose and of fat through its transformation into glucose is to serve through oxidation as the specific source of energy for the functional energetic activities of the tissues, the specific function played by amino-acids in metabolism is to serve as the material required for the synthesis of the active substances and structures of the active functional units of the tissues, through the activity and mechanism of which active substances and structures these functional units of the system perform their specific function. The main source of amino-acids is exogenous in origin and is from the intestinal absorption of amino-acids obtained from hydrolysis of proteins in the alimentary canal. Just as glucose and fat are stored up and conserved for the needs of the system in liver and fat cells respectively, so also there is reason to believe that amino-acids after intestinal absorption are not allowed to flood the system in strong crystalloid solution and to cause irregularity of the osmotic toxicity of the tissue fluids and be lost in the urine, but are synthesized immediately after absorption into compounds incapable of affecting the osmotic toxicity of the system and impermeable through the kidney and conserved in this form for their special cellulo-synthetic function for the benefit of the system, being given out to tissues according to their cellulo-synthetic requirements. Though the view current among physiologists at present is that amino-acids absorbed from the intestinal canal during digestion circulate in the blood streams in crystalloid form only, such amino-acids as are required by the cells for cellulo-synthetic purpose being removed from the blood stream and synthesized by the cell into the cell substances, any superfluous amino-acids not required for this cellulo-synthetic purpose being de-aminated soon after intestinal absorption and the de-aminated portion being converted into d-glucose, and that further when amino-acids are required for cellulo-synthetic purposes during a period when amino-acids are not being absorbed from the

intestinal canal, these amino-acids are obtained by lysis of cell-proteins (as undoubtedly occurs in long continued starvation), and though the theory of Abderhalden that amino-acids absorbed from the intestinal canal are synthesized into the protein or proteins of the blood plasma in the intestinal wall (or rather, more correctly in the intestinal blood vessels) is not yet generally accepted, yet, for the following reasons, Abderhalden's theory must be regarded as substantially correct. The plasma protein or proteins cannot be regarded to be spontaneous in origin; like other proteins of the tissues and cells it must be regarded as originating through the synthetic action of certain synthetic ferments, the ferments synthesizing the plasma proteins being present in the blood. Further it must originate directly or indirectly from synthesis of amino-acids absorbed originally from the alimentary canal. Ferment action being reversible, the synthetic action of the ferments synthesising the plasma protein from amino-acids must continually be balanced by a small concentration of free amino-acids in the blood, however small this concentration of free amino-acids may be. These free amino-acids present in the blood would always be available for absorption by the cells and use by them for their cellulo-synthetic purposes. Moreover the protein substances of the cells are all of them (with the exception of the nucleoli, plasmosome and karyosome) functionally active substances, these active cell substances taking an active part in and therefore being absolutely necessary for the functional activity of the cell and the functional efficiency of the whole system. The plasma proteins are comparatively inert substances; no such active functional changes, as for instance take place during contraction in the proteins of a muscle cell, can be said to take place in the substance of the plasma protein in the adjustment of the system to its normal requirement. From the point of view of physiological economy it would be more advantageous to hydrolyse this inert plasma protein and to use the resulting amino-acids for cellulo-synthetic purposes than to hydrolyse the active proteins of the cells, as hydrolysis of the cell proteins would be followed by loss of functional efficiency of the cells and of the whole system consequently. It is only in abnormal conditions of starvation that hydrolysis of cell proteins is necessary for cellular synthesis. For these reasons we must subscribe to the theory that amino-acids absorbed during digestion are synthesized directly in the intestinal capillaries into the plasma protein and that the plasma protein represents a reserve of amino-acids, just as glycogen in the liver cells and fat in lipid cells represent reserves of glucose and of fatty acid and glycerine respectively. Reuter has shown that before amino-acids from the intestinal canal pass into the intestinal blood vessels they are synthesized

into and stored up as protein in the epithelial cells of the villi near the basement membrane. This temporary storage delays the passage of amino-acids into the blood, and this delay is doubtless of some physiological importance. The synthesis of amino-acids into the plasma protein in the intestinal capillaries is effected by special proteo-synthetic ferments present free in the blood; this synthetic action is reversible and is being continuously balanced by a small concentration of amino-acids free in the blood. In the tissues when cellular synthesis is active either in the nucleus or cytoplasm, these free amino-acids are absorbed by the cells and synthesized into the nuclear and cytoplasmic substances. Though the intestinal epithelial cells having the function of temporarily storing up amino-acids as protein in the intestinal absorption of amino-acids, quite unlike the storage of special cells for the special storage of glucose and of fatty acid and glycerine, no special cells are set apart in the system which have the special function of storing amino-acids in the reserve. A small quantity of amino-acids is, however, stored up in reserve in the nuclei of all cells as the basophile and acidophile nucleoli (karyosome and plasmosome.) These bodies must be recognized to be representing only a reserve of amino-acids stored up to be used later in nuclear synthesis. By a reversal of the process involved in de-amination of amino-acids the system is able to synthesize endogenously at least certain amino-acids. Nucleic acids, substances chemically resembling amino-acids, being complex compounds of ammonia with carbon groups, are wholly endogenous in origin. Synthesis of other amino-acids is perhaps most marked in starvation, and there is no doubt that these endogenously formed amino-acids as well as the intermediately derived amino-acids resulting from hydrolysis of the cell proteins of the non-vital tissues in starvation are synthesized and stored up in the plasma protein before being given up to the vital tissues for their cellular synthesis. Whatever the origin of amino acids, the essential specific function played by amino-acids and the allied nucleic acids in the economy of the system is to serve as material for the purpose of the cellular synthesis of the active substances and structures of the cell. The plasma protein or the plasma proteins are bodies of specific chemical composition having each definite quantitative compositions of various amino-acids. The various amounts of different amino-acids absorbed into the intestinal blood vessels during degestion will not necessarily have the same quantitative proportion to one another, as the definite quantitative proportion of these amino acids in the plasma protein. Hence in intestinal absorption only such quantities of various amino-acids as are exactly in proportion to the quantitative ratio of these amino-acids in the plasma protein will be synthesized in the intestinal capillaries into the plasma protein, and any portion of a particular amino

acid in excess of such proportionate quantity will be left free in the blood. So, too, various cell and tissue protein substances have not the same quantitative composition of amino-acids as the plasma protein. In synthesizing the substances of the cell and tissue structures from the amino-acids resulting from haemic proteolysis of the plasma protein the tissues will synthesize into these substances various amino-acids only in proportion to the quantitative chemical composition of the substance synthesized, and quantities of various amino-acids in excess of the proportion required will be set free in the tissues and the blood. Thus when either absorption of amino-acids from the intestinal canal or cellular synthesis in the tissues is actively taking place, not inconsiderable quantities of amino-acids incapable of being immediately synthesized either into the plasma protein or the proteins of the cells are set free in the blood and tissues. These superfluous amino-acids are not allowed to remain free in the tissue fluids but are de-aminated chiefly in the liver, but also in the intestinal wall, blood spleen and other organs into ammonia and the corresponding hydroxyacid. The hydroxyacid is further transformed in the liver into d-glucose, proportionately much less oxygen being required to convert the hydroxyacid into d-glucose than the higher fatty acid. When amino-acids are de-aminated in this way the ammonia obtained from this de-amination before being converted into urea in the liver exerts its specific stimulant effect on the system, the dynamic action on general metabolism of amino-acids not used for cellulose-synthetic purposes and not de-aminated for antacid purposes being due to the stimulant action of this ammonia liberated, which ammonia has a powerful stimulant action on all kinds of functional units, exciting them to greater functional activity and greater oxidative consumption of d-glucose. On account of this stimulant effect of ammonia on the oxidative consumption of d-glucose, though the hydroxyacid portion of the de-aminated amino-acid is converted in the liver into d-glucose, yet after the stimulant effect of this ammonia has passed, the result of such de-amination and endogenous conversion of amino-acid into glucose is not to add very much to the quantity of glucose in the body and consequently of glycogen or of fat either. For this reason it would be unphysiological on the part of the system, when the hepatic glycogen store is depleted, to endeavour to replenish this depleted store by endogenous conversion of amino-acids into glucose. Moreover, the total quantity of amino-acids ingested, especially in non-carnivorous animals, even if the whole were converted into glucose at the expense of cellulose-synthetic requirements, would supply only a small portion of the glucose requirements of the system. The endogenous conver-

sion of fats into glucose has no similar dynamic effect on metabolism unless it is so intensive as to cause marked acidosis in which case the acids liberated stimulate the central nervous system. These reasons and other reasons mentioned in paragraphs 19, 21 and 23, that endogenous glucose formation in the liver is attended with acidosis and not ammoniæmia and alkalosis, and with transportation of fat to the liver cells and infiltration of the liver cells with fat, we are justified in adhering to the theory of Pflüger that fats are the main endogenous source of glucose in the body, though the general opinion among physiologists is that amino-acids and not fats are the main endogenous source of glucose in the body. In normal conditions with diet containing normal quantities of carbohydrate, amino-acids are needed and specially conserved for cellular synthesis, and it is only those free amino-acids that are rejected by the tissues as being in excess of their cellulose-synthetic requirements that are transformed into glucose, and this is apparently more to prevent the harmful effects of the unphysiological accumulation of these crystalloids in the tissues than for their physico-chemical energetic value. When acidosis occurs, amino-acids are, however, deviated from their cellulose-synthetic function to antacid purposes, being de-aminated in the liver chiefly and the ammonia obtained by de-amination being used to restore the alkalinity of the blood and tissue fluids, the hydroxyacid portion being as before converted in the liver into d-glucose. When ammonia obtained by de-amination is used for antacid purposes, being neutralized it fails to exert its specific stimulant effect on metabolism and does not cause greater oxidative consumption of d-glucose in the system as it does when de-aminated spontaneously and not used for antacid purposes. D-glucose formed in this condition from the de-aminated portion of the amino-acid being not used up remains in the system and becomes available to raise the quantity of d-glucose in the system and, if required, to be converted into glycogen. Thus it is that when a sufficient quantity of protein is administered to a starving animal, the quantity of intermediate acidic and ketonic bodies liberated by the liver is found to have diminished. What happens is this. In a starving animal d-glucose liberated by the liver into the blood stream originates practically entirely from fats and is therefore attended with the liberation of a corresponding quantity of these intermediate bodies. When proteins is given to the animal, a not-inconsiderable quantity of superfluous amino acids are de-aminated in the liver and the ammonia obtained from this de-amination is immediately neutralized by the intermediate acidic bodies formed in the liver from the transformation of fat into glucose. Being immediately neutralized, ammonia obtained

from these amino-acids fails to exert its stimulant effect on metabolism and the d-glucose obtained from the hydroxyacid part of the amino-acids goes to raise the concentration of d-glucose in the liver. Part of this d-glucose obtained from these amino-acids is converted into glycogen, raising the level of the hepatic glycogen store and diminishing the endogenous formation of glucose from fat and the liberation of these intermediate acidic and ketonic bodies.

(26) *Various conditions affecting the blood glucose concentrations.*—

Adrenalin, as we have already seen, causes hyperglycaemia by raising the hepatic glucose equilibrium concentration. Insulin has the opposite effect, causing hypoglycaemia by lowering the hepatic glucose equilibrium concentration. In Addison's disease in which the adrenals are diseased and we have consequently hypofunction of the adrenal medulla we have therefore hypoglycaemia. In conditions where insular tissue is in an abnormal condition of cellular degeneration, interstitial fibrosis or of vasculo-obstructive changes, or is in a condition of physiological cytoplasmic functional exhaustion through excessive stimulation, we have in these conditions hypofunction of insular tissue causing hyperglycaemia. Hypofunction of insular tissue caused by exhaustion through excessive stimulation gives rise to a vicious cycle as the hyperglycaemia resulting from hypofunction further stimulates these cells in this way not allowing them sufficient time and opportunity to recuperate and thus causing greater cytoplasmic functional exhaustion and hypofunction. Extract of the posterior lobe of the pituitary which has sometimes the effect of stimulating the involuntary muscles to greater functional activity, when it has that effect on the involuntary muscles, also causes a physiological hyperglycaemia necessary for this functional activity probably by raising the hepatic glucose equilibrium concentration in the same manner as adrenalin does. In the digestive absorption of carbohydrates in spite of the increased action of insulin in normal conditions the systemic blood is hyperglycaemic though much less than the portal blood. This is so because the circulation carries the portal blood into the systemic area before the liver cells have time enough to absorb the glucose it contains. Hyperthyroidism is associated with hyperglycaemia, hypothyroidism is associated with hypoglycaemia. Frolick's syndrome (*dystrophia adipose genitalis*), a condition associated with disease of the pituitary is attended with hypoglycaemia. How the secretion or absence of secretion of these glands, thyroid and pituitary, affects the blood glucose concentration, whether through acting directly on the liver and affecting the hepatic glucose equilibrium concentration or indirectly through action on insular and adrenal tissues, is doubtful.

(27) *A review of the nature and conditions of the normal hepatic functions with regard to the metabolism of carbohydrate fats and amino-acids.*—

We are now in a position to summarize as a whole our knowledge of the normal hepatic functions regarding the normal metabolism of glucose fats and amino-acids as far as it concerns the problem of diabetic metabolism. The basic factor regulating this metabolism, and therefore the cardinal concept on which depends its proper understanding, is the hepatic glucose equilibrium concentration. This, as already defined, is the concentration of free glucose in the liver cells at which the glucose-into-glycogen and the glycogen-into-glucose interactions exactly balance one another. The concentration of free glucose in the liver cells always tends to approximate as close as possible to this hepatic glucose equilibrium concentration. Adrenalin raises and insulin lowers this hepatic glucose equilibrium concentration, the effect of these drugs being directly proportional to the algebraic sum of the doses administered. If the blood brought to the liver cells conjointly by the hepatic artery and portal vein has a greater free glucose concentration than the hepatic glucose equilibrium concentration, glucose is withdrawn from the blood into the liver cells and converted either into glycogen or fat. If, however, the hepatic blood glucose concentration is less than the hepatic glucose equilibrium concentration, glucose obtained either from fat or glycogen passes from the liver cells into the blood. The concentration of free glucose in the circulating blood always tends to approximate as close as possible to the hepatic glucose equilibrium concentration. If the hepatic glycogen store is depleted below a certain physiological level, the liver is stimulated to endogenous glucose formation from fat. If the hepatic glycogen store is saturated above the physiological storage level, the liver is stimulated to endogenous fat formation from glucose. The intensity of the stimulus to endogenous glucose or fat formation is proportional to the degree of depletion below or saturation above the physiological storage level. Superfluous free amino-acids in the blood in excess of the small normal physiological hæmic concentration of free amino-acids are de-aminated by the liver and the de-aminated portion converted into d-glucose. In conditions of acidosis, the liver de-aminates amino-acids and uses the resulting ammonia to neutralize the acidosis.

(28) *Alteration of normal hepatic metabolic functions in abnormal hepatic circulatory and cellular conditions.*—

It must, however, be remembered that these normal hepatic physiological functions are performed in this normal manner only when the liver cells are in a normal physiological condition as regards the physico-chemical composition and action of their active cellular substances and in a normal

condition of circulation and nutrition. We cannot expect the liver cells when in condition of æræmia, blood stasis, venous obstruction, fibrosis etc., or in condition of toxic cellular degeneration, to perform their normal metabolic functions in the normal physiological manner as normal liver cells do in normal conditions. As an example of how abnormal circulatory and nutritive conditions affect the normal hepatic cells, we may take the effect of insulin when artificially administered and allowed to produce hypoglycæmic collapse. If a sufficient dose of insulin with a good supply of exogenous glucose is administered to a depancreatized animal, the hepatic glycogen store rises from half per cent to the normal twenty per cent of the liver weight. This represents the normal action of insulin on the normal liver in normal conditions. If however a perfectly normal animal with a perfectly normal hepatic glycogen store is administered a large dose of insulin and is allowed to go into a condition of hypoglycæmic collapse, when examined after it has been in a condition of hypoglycæmic collapse for some time, the hepatic glycogen store will be found to be greatly depleted. There can be no doubt that in this latter condition we are observing not the direct physiological effect of insulin on the hepatic metabolism, but the direct effects of an artificial hypoglycæmic collapse caused by an unphysiological administration of an over dose of insulin. On account of hypoglycæmic collapse, respiration and circulation would be feeble; the blood supply to the liver cells would therefore be deficient both in fats and oxygen and normal hepatic functions would suffer from anoxemia. The normal hepatic function of endogenous glucose formation from fats would fail; and if the tissues are to be supplied with the d-glucose they metabolise even in this condition of hypoglycæmic collapse, the glycogen store of the liver must undergo continuous depletion to supply this glucose. So too in various toxic degenerative conditions of the hepatic cells, we have various abnormalities of hepatic functions with regard to the metabolism of carbohydrates, fat and amino-acids. Thus in chloroform poisoning we have fatty infiltration of the liver cells with acidosis without glycosuria, the acidosis being apparently due partly to failure of the deaminating function of the liver. The acidic and ketonic substances of diabetic acidosis evidently also seem to cause toxic cellular degeneration in the liver and other organs. Thus in a chronic case of diabetes due to insular hypofunction, administration of insulin does not check the liberation of the acidic and ketonic bodies so rapidly as it checks the glycosuria.

(29) *The Renal threshold; various kinds of glycosuria.*—

In the normal individual, with normal concentrations of d-glucose in the blood, the renal tubules are very slightly permeable to the passage of d-glucose into the urine, the total daily quantity of sugar passing into the urine being in normal conditions a very small fraction of a gramme.

If, however, the blood glucose concentration is progressively raised above normal concentrations, the proportional permeability of the tubules to passage of glucose into the urine rises very considerably and rapidly. The minimum concentration of d-glucose in the blood at which glucose passes into the urine in such large amounts as to be detected by the usual gross chemical tests (Fehling's, Benedict's etc) is by convention called the renal glucose threshold. Passage of glucose into the urine in such large amounts is called glycosuria. The percentage of glucose in the blood of a normal individual who is fasting (*i.e.*, not digesting and absorbing a carbohydrate meal) and is at rest is 0.10%. During active exertion attended naturally with hyperadrenalinæmia, the blood-glucose concentration may rise up to 0.20%. Symptoms of hyperglycaemic collapse commence when the blood sugar percentage falls to 0.06% and below. During absorption of a carbohydrate meal, there is an alimentary hyperglycaemia, the degree and period of which varies according to the rapidity of the absorption and the amount of carbohydrate ingested. The renal threshold for a normal man is 0.23%. When there is hypofunction of insular tissue due either to physiological exhaustion through over stimulation, disease or extirpation, the blood glucose percentage rises according to the degree of insular hypofunction. In severe conditions in proportion to the degree of hypofunction, the blood glucose concentration may rise, even when carbohydrates are not being absorbed intestinally and when the person is absolutely at rest, above the normal renal threshold 0.23% to 0.30% or even more. In other abnormal conditions, the pathology of which is not properly understood, the renal glucose threshold may fall even as low as 0.04%, that is below the normal blood glucose concentration in starvation and rest. A condition similar to this last abnormal condition may be artificially induced by administering phloridzin, a drug which has the effect of making the kidney tubules excessively permeable to d-glucose, lowering the renal threshold below the normal blood glucose percentage in starvation and rest even. When the renal epithelium is diseased as a result of chronic nephritis the renal glucose threshold may rise up to 0.33%, and in some of these cases of chronic nephritis we may have as a result of disease, etc., hypofunction of insular tissue causing hyperglycaemia much above the normal renal threshold (0.23%) but not so high as this abnormal renal threshold (0.38%). In such case in spite of hypofunction of insular tissue and hyperglycaemia, we have no glycosuria. We may have therefore three varieties of conditions associated either with hypofunction of insular tissue or abnormal renal thresholds or with both. In the first condition there is hypofunction of insular tissue causing hyperglycaemia above the normal renal glucose threshold, but owing to chronic nephritic changes the renal threshold is still

higher than this abnormal hyperglycaemia thus preventing glycosuria. In the second condition there is hypofunction of insular tissue causing hyperglycaemia above the normal renal threshold, the renal glucose threshold in this case remaining normal. In the third condition, the insular function and therefore the blood glucose concentration, is normal, but the renal glucose threshold is through disease abnormally low, even lower than the blood glucose concentration in a condition of starvation and rest. In the first condition we have no glycosuria, but as a result of hypofunction of insular tissue the hepatic glucose equilibrium concentrations are much higher than in an individual with normal insular function. The blood glucose concentration adjusts itself to these higher hepatic glucose equilibrium concentration, and as there is no continuous glycosuria, except for the effects of chronic nephritis, the metabolism of carbohydrates fats and amino-acids in the liver and tissues proceed quite as normally as in a normal individual. Thus in absorption of carbohydrates from the intestine, the absorbed glucose will raise the blood glucose concentration above the hepatic glucose equilibrium concentration and thus will cause the liver to transform the excess of exogenous glucose into glycogen and fat. In spite of the hyperglycaemia, the energetic tissues will oxidize only as much glucose as they require for their energetic requirement. If as a result of a period of intense activity of the energetic tissues, the hepatic glycogen store was acutely depleted, the endogenous glucose formed from fat as the result of the stimulation caused by the depletion of the hepatic glycogen store would remain in the system and be available for conversion into glycogen, thus raising the level of the hepatic glycogen store to the physiological level. Thus, except for the effects of the chronic nephritis, the metabolism of carbohydrates fats and amino-acids would proceed quite normally in cases affected by the first condition. Actually in these cases we find no signs of diabetic metabolism as depletion of the hepatic glycogen store, acidosis, wasting of the tissues, etc. In the second and third conditions, we get marked glycosuria proportionate to the difference in the blood glucose concentration and the renal glucose threshold. In the second condition the abnormality causing glycosuria is insular hypofunction; in the third, the low renal glucose threshold. The d-glycosuria and diabetes of the second condition may therefore be called insular in origin; that of the third condition renal in origin. Whether the glycosuria be of renal or insular origin, the concentration of d-glucose in the blood will fall in proportion to the intensity of the passage of d-glucose into the urine. This fall in the blood glucose concentration would cause depletion of the hepatic glycogen reserve, the liver attempting to restore the blood glucose concentration to the level of its hepatic glucose equilibrium concentration first by converting glycogen into glucose. The depletion of the hepatic glycogen store stimulates endogenous

formation of glucose from fat, and continues till the depletion reaches such a degree that the endogenous glucose formed continuously as the result of the stimulus of the depletion is equal to the amount of glucose simultaneously passing into the urine and oxidized in the tissues. In proportion to the intensity of endogenous glucose formation we have liberation of B-oxybutyric and aceto-acetic acids and acetone, the intermediate products of the transformation of fats into glucose. These intermediary acidic and ketonic bodies cause secondary effects already considered. Whether the glycosuria be of insular or renal origin, secondary to the glycosuria we have the same sequence of pathological causes and effects to which syndrome of symptoms and signs and sequence of causes and effects we give the name Diabetes Mellitus. In both renal and insular types of diabetes mellitus we have exactly the same vitiation in the metabolism of sugars, fats and amino-acids in the system, and clinically it is impossible to distinguish between these two types of diabetes mellitus as they show the same signs and symptoms unless we determine the blood glucose concentration. In paragraph No. 18 we argued from the fact that administration of insulin causes hypoglycaemia, that it is impossible that insulin is a hormone specifically stimulating the energetic tissues to greater oxidative metabolism of d-glucose, for such a stimulant hormone should coincidentally cause a hyperglycaemia physiologically necessary if this specific stimulation is to be effective. A study of these three types of combinations of abnormalities of insular function and renal glucose thresholds confirms us in this opinion and conclusively demonstrates that normal metabolism or abnormal diabetic metabolism of sugars, fats and amino-acids does not at all depend upon whether insular function is normal or not but upon whether glycosuria is absent or present. If glycosuria is absent, the normal metabolism of sugars, fats and amino-acids proceeds quite normally even in the absence of insular secretion. If glycosuria occurs, even if insular function is normal, we have the same diabetic aberration of metabolism as in cases of glycosuria with deficient insular function. Diabetic alteration of metabolism essentially results not from insular hypofunction but from glycosuria. In the normal man, normal insular function effects normal metabolism not by any direct action on energetic tissues but through preventing glycosuria by keeping the hepatic glucose equilibrium concentration and the blood glucose concentration below the normal renal glucose threshold, thus conserving the hepatic glycogen store at the physiological level and preventing the harmful effects secondary to continuous depletion of the hepatic glycogen store. The capacity of the normal liver cells to keep up their glycogen reserve to the physiological level depends conjointly on the renal glucose threshold and on insular function which determines the hepatic glucose equilibrium concentration. When the hepatic glucose equilibrium concentration, whether

normal or abnormal, rises above the renal glucose threshold, whether normal or abnormal, glycosuria and its resultant pathogenic effects causing diabetic vitiation of metabolism takes place.

(30) *The aetiology and treatment of diabetes mellitus of renal origin.*—

We may now come to the question of the aetiology and the problem of the treatment of these two types of diabetes mellitus. Of the aetiology and pathology of the factors and conditions causing an abnormally low renal glucose threshold not much is known (at least to the writer). Various facts point out that the posterior lobe of the pituitary has some hormonal control over renal secretion. Thus injury or lesion to the pituitary and various parts of the brain chiefly the infundibular region are reported to cause diabetes insipidus, which can be controlled by administration of posterior lobe extract. Hypodermic administration of this extract is also reported to reduce the amount of urinary sugar in both types of diabetes mellitus. Probably therefore in cases of renal glycosuria, there is some lesion traumatic or otherwise in the apparatus controlling urinary secretion either in the pituitary posterior lobe or in the nervous centres either controlling or controlled by its secretion. Administration of posterior lobe extract has therefore been advised and may be tried in renal glycosuria.

(31) *The aetiology and treatment of insular diabetes mellitus.*—

Thanks to the original work of eminent physiologists as Claude Bernard, Minowski and Mering, Homans, Allen, Scott, Bunting and others, we are now in a position to understand how insular hypofunction gives rise to insular type of diabetes mellitus. Hypofunction of insular tissue itself may be caused by several aetiological factors. In most cases of diabetes mellitus, the insular cells, as regards their cell substances, blood supply etc., are in a normal physiological physico-chemical condition, but are deficient in normal functional tone and efficiency as a result of physiological exhaustion caused by excessive continuous stimulation of cytoplasmic functional activity and by deficient stimulation of reparative cellulo-synthetic processes. Physiological exhaustion is a normal phase in the cycle of cellular processes of all active tissues, and is normally followed by reparative cellulo-synthetic processes. When not followed by reparative cellulo-synthetic processes, physiological exhaustion becomes abnormal in duration of its phase and then causes pathogenic effects. In other cases insular hypofunction may be caused by abnormal pathological changes either in the insular cells their blood supply, interstitial connective tissue, etc., by various pathogenic agents and pathological conditions. Various microbic infections such as syphilis, tubercle, cancer, etc., either of the insular parenchyma blood vessels, or interstitial connective tissue may have a pathological

action on insular tissue causing hypofunction. Various toxins and toxic substances, such as the toxins of microbic infection in other parts of the body, the toxins of intestinal and other sapraemias, the toxins of pregnancy, the toxins of dietetic origin, the toxins or toxic substances of gout, and various extraneous organic and inorganic poisons as phosphorous, arsenic, chloroform etc., etc., may cause pathogenic cellulo-degenerative changes either in insular cells or its blood vessels and interstitial connective tissue causing hypofunction. Airtaminosis and endocrine disturbances may also have a pathogenic action on insular tissue causing hypofunction. Inflammatory round cell infiltration, fibrosis, calcification of insular connective tissue resulting either from direct microbic action or toxæmia would mechanically prevent hypertrophy and hyperplasia and would be an aetiological factor tending to perpetuate hypofunction. Arteriosclerosis, capillary sclerosis, venous stasis and other vascular changes resulting from high blood pressure, portal obstruction, etc. by causing anaemia or interfering with the normal blood supply would also cause hypofunction of insular tissue. In the treatment of insular diabetes we have in insulin a symptomatic therapeutic agent of great potency and utility by the administration of which we can rapidly check symptoms and effects resulting from insular hypofunction. By administering insulin in suitable doses in any case of insular diabetes even in extreme conditions of diabetic coma we can rapidly lower the hepatic glucose equilibrium concentration and the blood glucose concentration below the renal glucose threshold and thus prevent glycosuria. When glycosuria is inhibited glucose whether of exogenous or endogenous origin remains in the system and is able to be utilized to raise the level of the hepatic glycogen store. The level of the hepatic glycogen store rises to the normal physiological level. With this rise to the normal physiological level the stimulus to endogenous glucose formation is diminished to the minimum and acidosis and its pathological effects inhibited. When insulin alone is administered, there is danger of hypoglycaemic collapse which danger may be averted by administering sufficient quantities of easily absorbable carbohydrate, or counteracted by timely injections of adrenalin or by intravenous glucose solutions. By administering insulin to a case of insular diabetes we may thus restore to normal the metabolism of carbohydrates, fats and amino-acids as long as the effects of insulin last. Administration of insulin has only a temporary symptomatic effect. Our aim in the treatment of this disease should therefore rather be to cure hypofunction of insular tissue by restoring insular tissue to full normal physiological tone and efficiency and maximum histological hypertrophy and hyperplasia. The action therefore of various pathogenic aetiological factors causing pathological changes either in insular cells, its blood vessels and blood supply and connective tissue must be inhibited, and their pathogenic

effect undone. Mallanah and Raman Pillai (Antiseptic, May and July, 1923) have made in this respect an important contribution to the treatment of insular diabetes mellitus by demonstrating that cases of diabetes mellitus associated with some bacterial infection (carbuncle, cystitis, septicaemia, etc) could be cured of diabetic symptoms by administration of auto-vaccine. In these cases there is no doubt that the diabetes was secondary to the degenerative action of the toxins of the bacterial infection on insular cells. The action of the auto-vaccine aborted the degenerative effect of the toxins on insular tissue by developing greater immunity to the bacteria and their toxins and curing the infection, thus allowing insular cells to regain their normal function. Specific antimicrobial and antitoxic treatment should therefore be administered. Inflammatory round cell infiltration and fibrosis of the insular connective tissue would require treatment with iodides and fibrolysin. Once pathological degenerative, infective, inflammatory and other changes in insular tissue are resolved, we can set about attempting to restore the gland to normal functional tone and efficiency. All functionally active tissues and cells in normal physico-chemical physiological condition respond to a proper physiological treatment by increase in functional efficiency. Some tissues, among them insular cells, are capable of responding to such physiological treatment by hypertrophy and hyperplasia also. It must be remembered that there are not only hormones and factors stimulating the specific cytoplasmic functional activity of different active cells, but there are also hormones and factors stimulating reparative and constructive cellulo-synthetic processes in cells. Thus, in pregnancy, the hormones from the maternal corpus luteum of pregnancy have a powerful cellulo-synthetic stimulant effect on the tissues of the foetus, thus causing natural foetal development, while certain foetal hormones from the placenta have a powerful cellulo-synthetic stimulant effect on the uterine musculature, the mammary gland, and the general maternal system stimulating some of the maternal tissues to greater hypertrophy and hyperplasia. In paragraphs 12 and 17 it has been argued from the fact that insular hypofunction and insular diabetes are rare in individuals leading a strenuous muscular life and common in sedentary individuals and for other reasons that adrenalin is probably a hormone having a powerful cellulo-synthetic stimulant action on insular cells. Insular cells, it has been shown, belong to the nutritive system of tissues. The active tissues of both dynamic and nutritive systems in the course of a day in normal physiological life may go through several cycles of phases of intense cytoplasmic functional activity followed by a phase of exhaustion and intensive reparative cellulo-synthetic processes. The physiological alternation of these phases keeps these tissues in proper functional efficiency and tone. Submission of these tissues to intensive and adequately

prolonged alternate phases of intense specific functional stimulation and of intensive stimulation of reparative cellulo-synthetic construction not only increases the functional efficiency and tone of the component cells, but in certain tissues, among them being insular tissue, causes hypertrophy and hyperplasia. If, therefore we wish to stimulate any tissue of the dynamic or nutritive systems to hyper-tonus and hyper-efficiency, the physiological procedure we should adopt would be as follows. If the tissue is exhausted we must give it rest from specific functional activity and actively stimulate reparative cellulo-synthetic processes in it. When it has fully recovered from functional exhaustion we must stimulate it to specific cytoplasmic functional activity till it becomes exhausted. The treatments of Allen, Newburgh and others, which aim at giving rest to insular cells from specific cytoplasmic functional activity by restriction of carbohydrates in diet must therefore be considered not quite physiologically complete in our object to restore insular tissue to hypertrophy and hyper-efficiency. More than mere rest is required. We cannot expect the muscular system of a puny individual to be made fit for world's championship wrestling contest by being kept for days and months and years continuously in bed with all his limbs in splints with a mere basal metabolism diet, nor could we expect his digestive powers to be equal to those of a strenuous muscular worker on full nutritive diet. To restore and keep such tissue as insular cells in full functional efficiency more than mere rest from specific functional activity is required. Alternate intensive stimulation of reparative cellular synthesis and of cytoplasmic functional activity is the physiological procedure required. Alternation of graduated-increasing amounts of muscular exercise with administration of graduated increasing quantities of carbohydrate in diet seems therefore indicated in the treatment of insular diabetes. Such treatment would undoubtedly give much better physiological result than mere restriction of carbohydrate in diet.

(33) *Conclusion: The Essential nature of the vitiation of metabolism in diabetes mellitus.—*

The object in writing this paper was to attempt to describe the essential nature of the vitiation of metabolism in diabetes mellitus. As a result of this study we may conclude as follows: The liver is the organ for the endogenous formation of glucose from fats. Depletion of the hepatic glycogen store below a certain physiological level stimulates the liver to intensive endogenous glucose formation from fat in proportion to the degree of depletion. When continuous glycosuria takes place the liver must liberate into the blood stream continuously as much glucose as is simultaneously lost in the urine and oxidized in the system. This it does by depleting its glycogen store till the endogenous glucose formation stimulated by the depletion is equal to the glucose simultaneously lost in

the urine and oxidized in the system. Endogenous formation of fat into glucose is accompanied by liberation of acidic and ketonic bodies, the intensity of the liberation of these bodies being proportional to the intensity of endogenous glucose formation, and is attended with wastage of fat in the system. These acidic and ketonic bodies cause various other toxic effects on the system. To neutralize the acidic substances amino-acids are deviated from cellulo-synthetic to antacid purposes. In both varieties of diabetes mellitus, insular as well as renal, we have this same vitiation of normal metabolism.

INSULIN AND ITS USE IN MODERN MEDICINE.

(Contributed.)

Among the many complex chemical changes going on in the human body and upon which life depends, carbohydrates play an important part. Normally the starch taken with the food is converted by digestive processes into monosaccharides of which glucose is the most important. The glucose is absorbed into the blood and carried to the liver and muscles where it is converted into glycogen. Between periods of absorption this glycogen is broken down to glucose which is oxidised for the production of energy. The study of these changes and the explanation of the various steps is very little understood because glycogen and glucose are the only two intermediary products of this carbohydrate metabolism that accumulate in the body—the glucose in blood and other fluids and glycogen in liver and muscle—in sufficient quantities to be measurable and tested by chemical means. This carbohydrate metabolism does not proceed independently but is closely related to the metabolism of fats and proteins as well. When anything goes wrong with carbohydrate metabolism the metabolism of fats too goes out of order and incompletely oxidised derivatives—the Ketone bodies—accumulate in the body and act as poisons. It will, therefore, be evident that if, as in diabetes, somewhere in the series of metabolic changes preceding the oxidation of glucose, a break occurs, the whole chain is disturbed. The oxidation of sugar does not occur as rapidly as other changes in the body with the result that hyperglycaemia and glycosuria result. The metabolism of fats is naturally interfered with and as a result ketones are formed and give rise to coma. The entire changes in body are also necessarily interfered with and emaciation and lowering of natural resistance result. The picture thus produced is practically that of Diabetes.

*Efforts which led to the discovery of Insulin.—*It is clear that in diabetes there is something at fault in the chemical process by which glucose is prepared for oxidation. Long years of experimenting and analogy

with other chemical processes in the body led to the idea that a substance called hormone controlled the metabolism of carbohydrates. Von Mehring and Minkowski as early as 1889, by removing the pancreas of dogs and producing in them severe diabetes, proved beyond doubt that the anti-diabetic hormone was secreted into the blood by the pancreas. Later experiments by ligature of the main pancreatic ducts (and absence of symptoms of diabetes in these cases) proved that an internal secretion probably from the Islets of Langerhans was the hormone in question. It was also shown that microscopic changes could be detected in the islet cells in patients who had died of diabetes. By preparing extracts from pancreas or islets of bony fishes (in which these islets of Langerhans exist as separate glands) and giving them to de-pancreated dogs the symptoms should naturally have disappeared. But these attempts at manufacturing the extracts of the islets were not successful. These failures were attributed partly to the hypothesis that the power of the pancreas to control carbohydrate metabolism depended on some other unknown mechanism and also partly to the destruction of the hormone during the process of extraction by powerful digestive ferments which such extracts of the pancreas must also contain. After years of struggle, F. G. Banting, with the assistance of C. H. Best, tied the secreting ducts and thus secured the degeneration of the pancreatic gland (the islets were shown to be unaffected). This was then removed and its extract when given to de-pancreated dogs relieved hyperglycaemia and glycosuria—two of the main symptoms. Attempts were then made to get extracts from intact glands. Extracts of fetal pancreas and alcoholic extracts of the unde-generated gland of cattle were given with similar results together with an improvement of the general condition. These laboratory experiments which resulted in the discovery of the 'elusive anti-diabetic hormone' were then tried on men. At first the alcoholic extracts caused considerable irritation at the site of injection. The clinical application of the hormone was made possible only by Dr. J. B. Collip who succeeded in preparing pure insulin by a method of fractional precipitation in alcohol which removed from the original alcoholic extract all the irritating substances. His method of preparation depended on the solubility of insulin in Ethyl Alcohol in concentrations up to 90%. Since nearly all proteins are insoluble at concentrations of alcohol that are considerably below this, Collip removed them from the original extract of the pancreas (made with 50-60% alcohol) by fractional precipitation after the extract had been concentrated by evaporation in Vacuo. A watery solution of precipitate which settles out at a concentration of 90% is Insulin. The fats are removed either by ether or by centrifuging. Further purification is then effected by reprecipitating either by alkali (Shaffer-Doisy method) or by means of picric acid (Dudley method).

The Chemical nature of Insulin.—Little is yet known regarding the chemical nature of Insulin. It contains no phosphorus or carbohydrate but a large percentage of sulphur. It is soluble in neutral alcohol up to 80%–90%, in water and in phenol, cresols, glacial acetic acid and formamide; but it is precipitated by very weak acids and in the presence of high concentrations of neutral salts. It is also precipitated by Picric and Benzoic acids and it forms a salt with Hydrochloric acid. In faintly acid solutions it withstands heat but in faintly alkaline solutions heat soon destroys it. In faintly acid solutions it does not dialyse and is readily absorbed by charcoal and kaolin. It gives the biuret reaction. Most of these reactions and the fact that it is very easily destroyed by pepsin and trypsin suggest that insulin is of the nature of albumose. Anyhow the last word as to its chemical composition and nature has not yet been said.

The effect of Insulin on the blood sugar of normal animals.—Within a few minutes of intravenous or subcutaneous injection of a moderate dose of insulin, the percentage of sugar in the blood begins to fall and it continues to do so for a variable period in a manner quite characteristic of insulin itself. After about half an hour the blood sugar begins to rise indicating that insulin is being neutralised by the liberation of sufficient glucose from the glycogen stores of the muscles and liver. It, therefore, follows that animals with small reserves of glycogen will suffer from more hypoglycaemia. This is a fact of great importance. Diabetics as a rule have low glycogen reserves. The blood sugar is therefore apt to go very low and the hypoglycaemic symptoms might be prolonged to a dangerous extent.

The effect of Insulin in experimental hyperglycaemia.—It has now been definitely proved that insulin lowers the blood sugar percentage in all cases of experimental hyperglycaemia irrespective of whether the glucose is added exogenously or endogenously. When given with the former type at different periods preceding the injection of sugar the resulting hyperglycaemic curve was greatly altered. But the best results were obtained when the sugar was given about an hour after the insulin. Stewart, Rogoff and Macleod produced experimental hyperglycaemia by giving morphine and epinephrin. The hyperglycaemia which resulted was greatly checked, if not completely stopped, in animals treated with insulin.

Insulin and Pituitrin.—An interesting antagonism was found to exist between these two drugs by Burn. Given in sufficient doses pituitrin like insulin inhibits the hyperglycaemia due to epinephrin; but when given together to normal animals, no change or (if at all) a little rise in the blood sugar was observed.

Action of insulin.—Eadie and Macleod showed that it acts chiefly on the tissues and not on the blood itself. Thus it was shown that the addition of insulin to blood incubated outside the body does not alter the rate at which sugar disappears from it but when the isolated heart is perfused with suitable saline solution containing the sugar, the addition of insulin to the solution causes it to lose its sugar more rapidly.

The mechanism by which blood sugar is reduced.—The above mentioned experiment shows that glucose is lost in the tissues. Why this occurs is perhaps the chief point around which the interest rests. One of three things perhaps occurs—its polymerisation into glycogen, its oxidation, its reduction to substances similar to fatty acids. The fact that glycogen was stored in the liver of diabetic animals as a result of giving insulin and carbohydrates led many to suppose that the whole process is essentially one of conversion to glycogen. This has now been shown not to be the case in normal animals. In fact, the reverse has been shown to occur (by Mc Corneek, O'Brien; Dale and Dudley.) That insulin does not keep the oxidation of glucose has been proved beyond doubt by Dixon and his co-workers who showed that there was a marked increase in oxygen consumption and in respiratory volume and rate, but very little change in the respiratory quotient. Even these changes occur about half to one hour after giving insulin by which time the blood sugar would have finished its initial fall. Increased oxidation cannot possibly be the cause of decrease of blood sugar. Recent observations regarding the respiratory quotient supports the probability of the remaining possibility—that glucose is converted to substances related to the fatty acids. For the present, however, the problem has to remain unsolved. Further researches may throw some light on this aspect of the question.

The pharmacological unit.—The extent to which insulin lowers the blood sugar is taken to be the basis for its assay, one unit being originally defined as the amount required to lower the blood sugar to 0.045 per cent. within 4 hours in a rabbit weighing 2 Kg and after 24 hours fasting.

Insulin in diabetes mellitus.—The effect of insulin on patients suffering from diabetes mellitus are the same as those on diabetic (depancreated) animals. Clinical experiments and experiences have shown that nearly all patients suffering from diabetes will benefit from the use of insulin. It tends to bring the diabetic individual as near the normal as possible. It supplies the missing link of the metabolic chain in diabetics. The result consequently is a lowering if not a disappearance of the urinary sugar and ketones and a reduction towards normal of the excessive blood sugar. Deleterious effects will therefore not be produced if blood sugar is not brought down to a very low degree. It may be safely taken as a general

rule that so long as glycosuria is present blood sugar is not very low. The dosage can thus be regulated by constant examination of the urine. It is only when urine is sugar free that blood sugar estimations are required as otherwise we will be treading dangerous grounds with eyes blindfolded.

Blood Sugar in diabetes.—The importance of estimating blood sugar in diabetes is twofold. First of all damaged pancreatic cells recovered more quickly when there was not an excessive amount of sugar in the blood. The best chance for recovery was when the blood sugar was as near to the normal as possible. It was in those cases where blood sugar was very high and uncontrollable that death was inevitable. Secondly, there is the problem of low blood sugar-hypoglycaemia—resulting from an overdose of insulin. It is this action of insulin that has always to be guarded against in the routine treatment of diabetes. This aspect of the question will be discussed more fully later on.

The value of insulin.—It is now practically a settled fact that insulin does not cure diabetes. All patients derive considerable benefit from its use. While insulin is administered the symptoms disappear and the patient regains his health and strength, but on stopping the insulin the symptoms usually reappear. But the reduction in the blood sugar and the adaptation to a well-regulated and tolerated diet will enable the diseased and damaged pancreatic cells to recover and carry on the normal metabolic processes. Insulin is also valuable in dispelling Ketosis occurring in diabetes. But it has been found in clinical experience that the longer the duration of the disease the less the benefit of the drug. In spite of all its benefits it is a much discussed question whether general practitioners can safely use it. Prof. Mac Lean says "Insulin like fire is a good servant but a very bad master ... it may be quite safely used by the general practitioner and I have no hesitation whatever in saying that it is the duty of the general practitioner to use it. If given in excessive amounts dangerous symptoms arise. The most important point is to go ahead very slowly and gradually and it is courting disaster to use short cuts in general practice." Another great thing to consider is the selection of the proper type of case. Every case is not fit for treatment with insulin and when given in these cases dangerous symptoms develop.

The Type of case in which insulin is indicated.—The determination whether the case under consideration is fit for treatment with insulin is a very important problem. Where dietetic treatment has failed to stop the ravages of the disease and if the patient is in an emaciated and helpless condition passing large quantities of urine loaded with sugar, acetone and diacetic acid and when there is not the slightest doubt that the end is not far off, insulin is the drug to be

tried. Others there are who show no emaciation but whose urine shows sugar only but no ketone bodies. It is often very difficult to decide whether insulin can be or should be used in these cases. The correct procedure is to put the patient on a dietetic regime and if this does not materially reduce the glycosuria or the blood sugar, insulin ought to be employed. These chronic glycosurists are not very much in danger of coma and usually live to a ripe old age but it must not be forgotten that at a later stage most of these develop one or more of the following symptoms; eye changes especially blindness, neuritis, local and general irritation, muscular weakness and nervous instability, trophic changes such as gangrene, carbuncles, etc. Much can be done to prevent the occurrence of these tragedies at an early date of the disease by dietetic and if this fails by insulin treatment. Once the definite symptoms set in, treatment is very difficult. In diabetic ketosis which has led to hunger and coma, insulin in large doses with plenty of carbohydrates must be given. Either of them given alone is quite useless. As yet insulin is not available on such a large scale as to be used by even those diabetics who can control their disease by dietetic measures only. More extensive production is necessary before it can be used as a luxury.

The type of case in which it is contra-indicated:—Insulin must not be given in cases of renal glycosuria in which the blood sugar is already very low. This condition can easily be excluded by a sugar tolerance test and the absence of acetone in the urine. The patient is given 50 gñ. of glucose dissolved in a little water and the bladder is then emptied in 2½–3 hours afterwards. This will contain glucose. If glucose is absent in this specimen the patient is possibly suffering from renal glycosuria and is not fit for insulin treatment. There are other cases again where the sugar is passed in large quantities and the condition looks grave but where response to diet is very good. Insulin is not indicated in such cases. Perhaps glycosuria alone should not be taken as an indication for insulin. Professor MacLean cites the case of an individual exhibiting glycosuria and undergoing insulin treatment but who really was suffering from carcinoma of the stomach. This patient never got better.

Methods of giving insulin:—Insulin is prepared in aqueous solution to which a small amount of antiseptic has been added. What was mentioned a few pages ago is the physiological unit but it is now taken as equivalent to 3 units. This should be given subcutaneously. If taken by the mouth or administered per rectum or by injection the desired therapeutic effect is not obtained.

The dangers of insulin administration:—Insulin solutions are so carefully prepared that there is no risk from sepsis or protein reactions there-

form. The only danger seems to be the appearance of hypoglycaemic symptoms when the blood sugar falls to 0.07 to 0.05 per cent. A feeling of lassitude, inertia, dizziness, flushings, sweating, pallor and nervousness are among the first symptoms observed. Abdominal pain, a constricted feeling round the chest, tremor of hands and numbness of hands, face or tongue are later symptoms which though not dangerous must be enough to warn the patient that his blood sugar is going down. The dangerous symptoms which are particularly noticeable in neglected cases are unconsciousness and convulsions. These symptoms due to a condition of hypoglycaemia usually come on from 2–4 hours after injecting the drug and luckily respond very easily to treatment.

The treatment of insulin hypoglycaemia.—The essential point is to get glucose into the blood as quickly as possible. It would certainly be better if all diabetics undergoing insulin treatment were asked to carry lumps of sugar or solutions of glucose in their pockets and take them whenever they feel the premonitory symptoms of hypoglycaemia. Prompt measures usually remedy such attacks. If the patient is unconscious the best means of counteracting the drug is by the administration of I. C. C. of 1 in 1000 of adrenalin or in its absence of I. C. C. of pituitrin. If, as it usually happens in about 5 minutes, the patient regains consciousness, he should be given glucose by mouth. If the patient still remains unconscious, up to 200 c. cm. of a 20–30 per cent. solution of glucose may be given intravenously or subcutaneously into the loose tissues of the abdominal wall. Or again a stomach tube may be passed and glucose administered. Where much collapse is present, stimulants such as strychnine or camphor hypodermically or hot coffee per rectum are said to be of much benefit. Most of these symptoms could, however, be prevented if insulin were given at the time of a meal containing carbohydrates and perhaps by giving insulin at least six hours before night as it usually takes about 6 hours for these hypoglycaemic symptoms to appear.

Details in the routine treatment of diabetes with insulin.—It is extremely difficult to lay down any hard and fast rule regarding the dosage of insulin and the diet to be taken during the treatment. Each case must be treated on its own merits. One of the first things is to ascertain whether the particular patient in question is a fit case for insulin treatment. The second point is to fix a diet for the patient which will keep his urine sugar free and if it is possible to always estimate it to keep his blood sugar within normal limits. The third but not the least to consider is the financial position of the patient. Insulin is not a cure for but only a help to cure diabetes, if we may so call it, and as such it requires prolonged administration—a procedure which at the present moment and at the present rate of output of the drug

is beyond the means of most diabetics in India. For insulin to have its best effect the diet must be suitably arranged and this is perhaps the most difficult problem to solve. Two kinds of cases usually arise. In one variety a moderately rich diet with maximum insulin but with glycosuria present is best tolerated. Prof. Maclean prefers this method of treatment to the other variety in which on a starvation diet sugar is occasionally absent in the urine.

Normally every good dietetic allowance should contain 10—15 calories for every pound of body weight, the exact quantity, of course, varying on the amount of work the individual does and such other factors. Of this according to Prof. Maclean about $\frac{1}{2}$ a gramme per pound of body weight must be the protein allowance, the carbohydrate allowance being small and not greater than $\frac{1}{3}$ of the weight of the fat given. [For making diets, tables showing approximate content in grammes of protein, fat and carbohydrate in foods are given at the end of the article.] Having put the patient on this fixed diet, he must be recommended to lie in bed for the first few days and his urine should be examined daily both qualitatively and quantitatively for sugar and ketone bodies. Subcutaneous injections of insulin in 5 unit doses twice a day to begin with are then given. With the rich diet given above the insulin is gradually increased up to a maximum of 50 units per day until the urine becomes sugar free. A good practice is to give insulin twice a day by subcutaneous injection of about 10 units about half an hour before the morning and evening meals. This dose can gradually be increased up to about 30 units twice a day. If the glycosuria still persists the carbohydrate element in the diet should be reduced, after which the insulin too should be cut down gradually and kept on at a dose of perhaps 15-25 units twice a day. Too much insulin should not be given for too long a period for fear of the appearance of hypoglycaemic symptoms. Difficulties arise when blood sugar estimations are not possible and where urinary sugar disappears early in the treatment. These are the cases which might give trouble and it is always safe to be cautious in administering insulin in them. Forewarned is forearmed.

Insulin and diabetic coma.—No routine treatment of coma with insulin has yet been successful. But where due to ketosis and characterised by air hunger, smell of acetone in the breath, diacetic acid in the urine and a low CO_2 in alveolar air (about 2.5%) Dr. Leyton has found much benefit by the following procedure. A large dose of insulin (about 30 units) is given. The blood sugar is estimated. If it is above 0.3%, dextrose need not be given. Otherwise 30 gms. of dextrose is given intravenously. The dose of insulin and

dextrose is repeated if necessary after about 3 or 4 hours. The dextrose alone should be repeated if symptoms of hypoglycaemia manifest themselves. As soon as the patient becomes conscious he is given plenty of lemonade and sugar to drink. If the patient suffers from vomiting, insulin is diminished and sugar is given per rectum (2 gms per unit of insulin). If however diarrhoea is also present the insulin is completely stopped.

Conclusion.—Insulin is certainly the most useful medical discovery of the day. Its benefits to the numerous diabetics all the world over cannot be denied. But yet it has its disadvantages. Its limited supply and output, its cost, its dangers and perhaps the prolonged duration of the treatment are all factors to be counted when recommending it to patients. Its transient effects are liable to shake the confidence of both the physician and his patients. Already a considerable number of medical men while recognising its value for temporarily saving a diabetic's life have already begun to grow sceptic about its 'Curing' powers. Perhaps the tediousness of estimations of blood control and the absence of the necessary patience and the co ordination on the patient's part have all contributed to the formation of this opinion. But these are yet in a minority and time alone with increased output and scope for research will show who is correct.

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A CASE OF ASCITICS AS A COMPLICATION IN DIABETES

By

DR. P. B. KARKAREY, M.B., NAGPUR, C. P.

One Mohammadan female aged 15 years had complaint of diabetes from her early childhood which was first noticed at the age of about 10 years. She was kept under different types of treatment. When she got worse and worse, western treatment was resorted to. One of my colleague practitioners was treating her, but during the short course of his treatment his attention was drawn to her distended pelvis which was tender on pressure and caused agonising pain to the girl. Seeing this gradually increasing tumour one could suspect systemic infection, as my professional brother presumed and he decided to take some operative measures.

I was called in simply as a consulting help in this interesting case particularly. When I examined the patient I found her simply reduced to skeleton. I took her temperature which was rather more or less subnormal. I could elicit absence of Pyrexia from the general informal talk of my colleague and I could exclude systemic infection on that point merely. I then at once decided to make an investigation about the nature of the fluid contents of the pelvis. We both agreed to push in the hypodermic needle with the syringe and on doing so we could suck some clear watery fluid which contained sugar. This made us to proceed boldly with a trocar and canula to drain the whole fluid out. This was done with the same precautions and measures of tapping an elsrless case in general. The fluid measured about 2 pints. The patient was relieved and felt much better than before. We put her on starch free diet. My colleague continued the symptomatic treatment with Tripsoyngen, pancreatic emulsion etc. Treatment by insulin was also contemplated. This diabetes ascitic, as I should call it, was due to renal insufficiency because of late the patient did not complain of Polysuria. So the fluid contents escaped and gravitated to the dependent position in the pelvis in this bedridden condition. This may also be due to the transudation of the fluid from the hyperglycaemic tissue into the pelvic peritonium.

DIABETES MELLITUS IN CHILDREN

By

A. MUKERJEE, M. D., FOERTON ROAD, DELHI.

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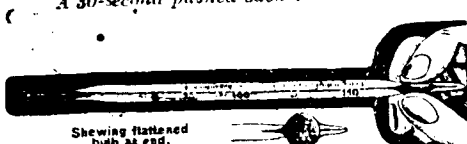
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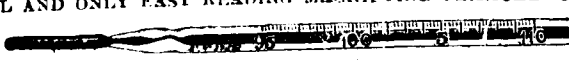
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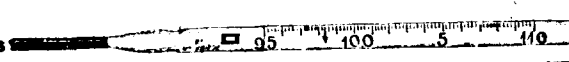
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one half to two per cent. of Diabetes occur during the first ten years of life, increasing in frequency with each year of life.

Etiology.—Possibly the simple fact that the urine is less frequently examined in very young children, owing to the difficulty in securing sufficient quantity for examination, may account for the less frequent diagnosis of this disease. Sex does not figure so prominently in children as in adults, there being about as many females as male children affected.

Race.—While the Hebrew race predominates in diabetic subjects, in the adult, it apparently has no influence in children.

Heredity.—Heredity seems to play a very important part in diabetes mellitus. The family history often shows one or both parents affected and, what is more striking, the parents were either blood relatives, or intermarriages had occurred in close ancestors, suggesting an "endogenous degeneration". Tuberculosis and syphilis, weakening the secretive power of the pancreas, have also been considered as ætiologic factors.

The exciting cause has probably caused as many theories as any other disease and as yet we cannot say positively what is the producing cause, injuries to the central nervous system, such as falls, blows, severe instrumental deliveries or deformity of the brain. Many writers thus believe diabetes has neurogenous origin independent of the pancreas or liver, while others equally contend that the nervous derangement is the cause of the pancreatic and hepatic disturbance, and, therefore, not the direct cause.

Pathology.—There is no constant characteristic lesion in diabetes mellitus. A certain per cent. of cases show a lesion of the nervous system, while others give no evidence of a neurogenous origin. More than half of all autopsies show some lesion of the pancreas, such as atrophy or obstructed ducts.

Symptoms.—The cardinal symptoms of diabetes mellitus, excessive thirst, increase of secretion of urine, excessive hunger, loss of flesh and increased feebleness, are the same in children as in adults.

The first noticeable symptom is enuresis, the first warning that nature gives of the trouble, the danger signal, and an examination of the urine should be made in every case of enuresis. This condition is followed or accompanied by excessive thirst, and not infrequently by excessive hunger. Flecks of sugar may be noticed on the underclothes and irritation of the genitals is a distressing feature. As emaciation becomes more rapid, acetone bodies appear in the urine, the breath has the odour of acetone, and we have acid intoxication, the same as in the adults.

The Urine.—Sugar dextrose is present from the beginning which is increased whenever the carbohydrates is increased in the food. The quantity of urine is often twice or more above the normal and specific gravity may reach 140. As the end approaches albumen may appear in the urine and "coma casts" are often seen. The approach of coma, the beginning of the end, is frequently foretold by nausea, vomiting, gastric pain and great nervous irritability.

Diagnosis.—The diagnosis, in the early stages, is often overlooked owing to the infrequency of examining the urine in children, specially in infants. The diagnosis of diabetes mellitus in children consists in finding on a normal diet, the constant presence of dextrose.

Diabetes mellitus is sometimes wrongly diagnosed on account of a failure on the part of the physician to differentiate lactose, or the split product galactose, which in infants enters the circulation through the intestinal canal and is excreted in the urine and dextrose.

A simple test for distinguishing dextrose from other sugars is to boil the urine with an equal amount of a ten per cent. solution of Sodium Hydrate for three minutes. Add Fehling's Solution and boil again. If the Fehling's Solution is reduced, the sugar is lactose and not dextrose.

Prognosis.—The prognosis in diabetes mellitus in children is even less favourable than in adults.

Treatment.—To overcome acidosis, which is nearly always present, milk of magnesia (Phillip's) is suggested till the blood is rendered alkaline.

In the administration of remedies we are to study each case carefully to determine, if possible, the prominent lesion which is aggravating the disease. If the presence of sugar is due to functional disorder of liver, **Chionanthus** and **Podophyllin** will be used with good results. Where irritability of the nervous system predominates, nerve sedatives will be called for, and wrongs of the digestive system must be overcome. **Arsenic** has long been considered a most important remedy in diabetes and may be combined with any indicated remedy. Astringent diuretics have been used effectively by increasing the pancreatic secretion.

The dietetic treatment must form a very important part in diabetes, though we are to always bear in mind the fact that our patient's strength is to be maintained as far as possible, for we have accomplished little or nothing if our patient is progressively losing weight and strength, even though the sugar has been eliminated. The carbohydrates should be withdrawn till the sugar disappears, then gradually restore them to the point of tolerance. The skin should be kept clean by daily baths. The bowels should move daily.

ESTIMATION OF BLOOD SUGAR.

1. Sugar.—(a) Benedict's Modification of the Method of Lewis and Benedict.¹

Principle.—The red color obtained by heating a glucose solution with picric acid and sodium carbonate is employed as the basis of the colorimetric determination. The blood protein is removed by precipitation with picric acid.

Procedure.—Two c. c. of blood are aspirated through a hypodermic needle and a piece of rubber tubing into an Ostwald pipette, a little powdered potassium oxalate in the tip of the pipette preventing clotting. The blood is drawn up a little above the mark and the end of the pipette is closed with the finger. After the rubber tubing and needle are disconnected, the blood is allowed to flow back to the mark and is discharged at once into a 25 c.c. volumetric flask, or into a large test-tube graduated at 12, 5 c.c. and at 25 c.c. The pipette is twice rinsed with distilled water, these washings being added to the blood. The contents of the flask are shaken to insure thorough mixing and a consequent laking or hemolysis of the blood, which is practically complete after a minute or two. A solution of sodium picrate and picric acid is added to the 25 c. c. mark (using a few drops of alcohol to dispel foam if necessary) and the mixture thoroughly shaken. After a minute or two (or longer) the mixture is poured upon a dry filter, and the clear filtrate collected in a dry beaker. Exactly 8 c.c. of the filtrate are measured into a large test-tube bearing gradations at the 12, 5 c.c. and 25 c.c. mark, and 1 c.c. of 20 per cent. (anhydrous) sodium carbonate solution is added. The tube is plugged with cotton and immersed in boiling water for 10 minutes. It is then removed, and the contents are cooled under running water and diluted to 12.5 c.c. or to 25 c.c. depending on the depth of color. At any time within a half an hour the coloured solution is compared in a colorimeter with a suitable standard solution, the standard being set at a height of 15 mm.

The standard solution may be simultaneously prepared from pure glucose by treating 0.64 mg. of glucose in 4 c.c. of water with 4 c.c. of the picrate-picric acid solution and 1 c.c. of the carbonate, and heating for 10 minutes in boiling water and then diluting to 12.5 c.c. A permanent standard solution may be prepared from picramic acid or from potassium dichromate as mentioned below. The potassium dichromate standard does not match the unknown with absolute exactness, but can be employed with satisfactory results when pure picramic acid is not obtainable.

Calculation.—If directions are followed exactly the calculation is as follows:

$$\frac{\text{Reading of standard}}{\text{Reading of unknown blood}} \div 10 = \text{per cent. of sugar in the original}$$

Where the final dilution of the unknown is made to 25 c.c. instead of 12.5 c.c. the final figure is, of course, multiplied by two.

II. Micro-method of Bang.

Principle.—Two or 3 drops of blood are transferred to a small weighed piece of blotting paper and the paper again weighed to determine the amount of blood. The paper is then treated with a boiling acidified KCl solution which coagulates the protein and allows the sugar to diffuse out. The sugar solution thus obtained is boiled with alkaline cupric chloride solution. The amount of cuprous chloride solution formed by the reducing action of the sugar is determined by titration with standard iodine solution.

Procedure.—Small pieces of good absorbent paper, about 16 X 28 mm. in size, weighing about 100 mg. and held by a small spring clip, are used,

To one of these previously weighed transfer 2—3 drops (about 120 mg.) of blood obtained by piercing the cleansed finger. Weigh again immediately and determine by subtraction the weight of blood taken.

Coagulation of Blood Protein.—Transfer the piece of paper to a test-tube and add 6.5 c.c. of boiling acid-potassium chloride solution and let it stand half an hour. The clear solution containing the sugar is poured into a 50 c.c. Jena flask, the flange of which has been removed. Wash the paper and tube again with 6.5 c.c. of hot salt solution and transfer washings to the flask. Cool.

Reduction of Cupric Chloride.—Attach to the mouth of the flask a piece of tight-fitting rubber tubing about 2 inches long (see fig. 91), provided with a clamp which permits of shutting off the contents of the flask from the outside air. Now add to the flask 1 c.c. of the cupric chloride solution. Heat so that the solution is brought to a boil in one minute and 30 seconds (an error of five seconds may be disregarded). Allow to boil for exactly two minutes; at the end of this time tighten the clamp over the mouth of the flask. At the same time remove from the flame and cool at once under the tap for about a minute.

Titration of Cuprous Chloride Formed.—The titration is made with N/200 iodine solution run in from a very accurate burette (preferably a 2 c.c. burette graduated in 1/50 c.c.). Two or three drops of starch solution (preferably soluble starch) are added as an indicator. During the titration air must be excluded to prevent re-oxidation. This is done by running a slow stream of carbon dioxide from a generating bottle through a small tube which extends nearly to the bottom of the flask. The titration should be carried out against a white background and the end point taken when the blue color persists for 20-30 seconds.

Calculation.—The copper and other solutions used in the test bind about 0.12 c.c. of the iodine solution. This amount must hence be subtracted from the reading. The corrected reading is then divided by 4 to obtain the number of milligrams of glucose in the sample.

Example.—If 0.68 c.c. of N/200 I solution were required, $\frac{0.68 - 0.12}{4} = 0.14$ mg. glucose in the amount of blood used.

If 140 mg. of blood were taken for analysis the per cent. of glucose in the blood would be $\frac{1,000}{140} \times 0.14$ mg. = 0.1 per cent. glucose.

The results obtained by this method are a little higher than those obtained by other reliable methods due to the presence of certain I-binding substances in blood. As these appear to be nearly constant in amount a correction may be applied. To obtain true values for glucose of the blood therefore subtract 0.015 per cent from the value obtained as above, 0.1 per cent—0.015 per cent = 0.085 per cent glucose.

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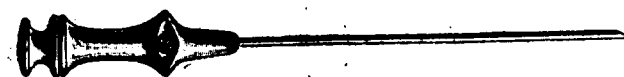
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III. Method of Folin and Wu.*

Principle.—When sugar and an alkaline solution of copper are heated cuprous oxide is formed. In the presence of a molybdate phosphate solution cuprous oxide gives rise to an intense and stable color reaction. The color so obtained is compared with the color given by a standard solution of sugar.

Solutions Needed.—1. °Standard sugar solution.—Three standard sugar solutions should be on hand: (1) a stock solution, 1 per cent. dextrose or invert sugar, preserved with xylene or toluene; (2) a solution containing 1 mg. of sugar per 10 c. c. (5 c. c. of stock solution diluted to 500 c. c.); (3) a solution containing 2 mg. of sugar per 10 c. c. (5 c. c. of the stock solution diluted to 250 c. c.). The diluted solutions should be preserved with a little added toluene or xylene; it is better not to depend on such diluted solutions to keep for more than a month, but the stock solution should keep indefinitely. If pure dextrose is not available, a standard solution of invert sugar may be made from cane sugar and is equally useful. To make this transfer exactly 1 gm. of cane sugar to a 100 c. c. volumetric flask; add 20 c. c. of normal hydrochloric acid and let the mixture stand over night at room temperature (or 70° C). Add 1.68 gm. of sodium bicarbonate and about 0.2 gm. of sodium acetate to neutralize the hydrochloric acid. Shake a few minutes to remove the most of the carbonic acid and fill to the 100 c. c. mark with water. Then add 5 c. c. more of water (1 gm. of cane sugar yields 1.05 gms. of invert sugar) and mix. Transfer to a bottle; add a few drops of xylene or toluene, shake well, and stopper tightly.

2. *Alkaline copper solution.*—Dissolve 40 gms. of anhydrous sodium carbonate in about 400 c. c. of water and transfer to a litre flask. Add 7.5 gms. of tartaric acid and when the latter has dissolved add 4.5 gms. of crystallised copper sulphate; mix and make up to a volume of 1 litre. If the carbonate used is impure a sediment may be formed in the course of a week or so. If this happens, decant the clear solution into another bottle.

3. *Molybdate Phosphate solution*—Transfer to a litre beaker 35 gms of molybdic acid and 5 gms. of sodium tungstate. Add 200 c. c. of 10 per cent sodium hydroxide and 200 c. c. of water. Boil vigorously for twenty to forty minutes so as to remove nearly the whole of the ammonia present in the molybdic acid. Cool, dilute to about 350 c. c. and add 125 c. c. of concentrated (85 per cent) phosphoric acid. Dilute to 500 c. c.

4. *Procedure*—Heat a beaker of water to vigorous boiling. Transfer 2 c. c. of the tungstic acid blood filtrate to a *blood sugar test-tube*, and to two other similar test-tubes (graduated at 25 c. c.) add 2 c. c. of standard sugar solution containing respectively 0.2 and 0.4 mg. of dextrose. To each tube add 2 c. c. of the alkaline copper solution. The surface of the mixtures must now have reached the constricted part of the tube. If the bulb of the tube is too large for the volume (4 c. c.) a little, but not more than 0.5 c. c. of a diluted (1:1) alkaline copper solution may be added. If this does not suffice to bring the contents to the narrow part, the tube should be discarded. Heat the tubes in a boiling water bath for six minutes. Then transfer them

* Folin and Wu: Jour. Biol. Chem., 1929, 41, 367.

to a cold water bath and let cool, without shaking, for two to three minutes. Add to each test tube 2 c. c. of the molybdate phosphate solution. The cuprous oxide dissolves rather slowly if the amount is large but the whole, up to the amount given by 0.8 mg. of dextrose, dissolves easily within two minutes. When the cuprous oxide is dissolved, dilute the resulting blue solutions to the 25 c.c. mark, insert a rubber stopper, and mix. It is essential that adequate attention be given to this mixing, because the greater part of the blue colour is formed in the bulb of the tube. At least five minutes after mixing make the color comparison in the usual manner.

Calculation.—The depth of the standard (in mm.) multiplied by 100 and divided by the reading of the unknown gives the sugar content, in mg., per 100 c. c. of blood.

Remarks.—The two standards given representing 0.2 and 0.4 mg. of glucose are adequate for practically all cases. They cover the range from 70 to nearly 400 mg. of glucose per 100 c. c. of blood.—“BIOCHEMICAL METHODS” by FRANK P. UNDERHILL, PH. D.

IV. Bertrand's Volumetric Method for the Estimation of Glucose.

This method has recently come into vogue for the estimation of glucose in blood, etc. It is not so suitable for urine as Benedict's method, owing to the fact that in many urines cuprous oxide does not easily settle, but remains partially in colloidal suspension.

Principle of the method.—The precipitate of cuprous oxide formed on boiling an excess of Fehling's solution with a glucose solution is filtered off, washed, and dissolved in a solution of ferric sulphate in sulphuric acid. The ferrous salt formed is titrated with a standard permanganate solution.

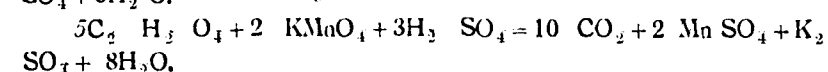
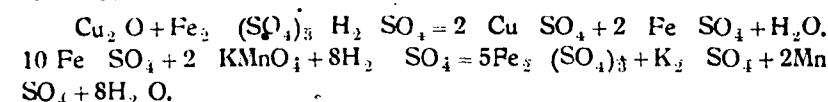
Solutions required.—(1) A solution which contains 40 grammes of pure copper sulphate in one litre. (2) A solution which contains 200 grammes of Rochelle salt and 120 grammes sodium hydroxide in one litre. (3) A solution which contains 50 grammes ferric sulphate (free from ferrous sulphate), and 200 c.c. concentrated sulphuric acid in one litre. (4) A solution which contains 5 grammes potassium permanganate in one litre. This solution is standardised in the following way:—

Weigh out 250 milligrammes ammonium oxalate, dissolve it in 50 c.c. water, add 2 c.c. concentrated sulphuric acid, warm to 60—80°C, and titrate with the permanganate solution until a pink colour persists.

Analysis.—Measure 20 c. c. of the sugar solution into a flask of 150 c.c. capacity, and add 20 c. c. each of the copper sulphate and Rochelle salt solutions. Heat to boiling and keep on the boil for three minutes. Filter through an asbestos filter, leaving most of the precipitate in the flask. Wash the precipitate in the flask with a little distilled water and decant through the same filter. Dissolve the precipitate in the flask in about 20 c. c. of the ferric sulphate solution and pour the solution through the filter. A green solution results which is titrated at once with permanganate until the green colour changes sharply into pink.

The solution must not contain more than 100 milligrammes of glucose, and gives the best results when the amount is between 10 and 90 milligrammes.

Calculation.—The amount of copper, precipitated as cuprous oxide, is calculated first from the following equations taking place during the reaction:—



One molecule of ammonium oxalate corresponds to 2Fe and therefore to 2 Cu. The amount of ammonium oxalate taken multiplied by 0.8951 (63.6 × 2)

— gives, therefore, the amount of copper corresponding to 142.1

the number of c.c. of permanganate used. From the amount of copper found the quantity of sugar is calculated by means of the following table:—

Glucose in mg.	Cu in mg.	Glucose in mg.	Cu in mg.	Glucose in mg.	Cu in mg.	Glucose in mg.	Cu in mg.
10	20.4	33	64.6	56	105.8	79	144.5
11	22.4	34	66.5	57	107.6	80	146.1
12	24.3	35	68.3	58	109.3	81	147.7
13	26.3	36	70.1	59	111.1	82	149.3
14	28.3	37	72.0	60	112.8	83	150.9
15	30.2	38	73.8	61	114.5	84	152.5
16	32.2	39	75.7	62	116.2	85	154.0
17	34.2	40	77.5	63	117.9	86	155.6
18	36.2	41	79.3	64	119.6	87	157.2
19	38.1	42	81.1	65	121.3	88	158.8
20	40.1	43	82.9	66	123.0	89	160.4
21	42.0	44	84.7	67	124.7	90	162.0
22	43.9	45	86.4	68	126.4	91	163.6
23	45.8	46	88.2	69	128.1	92	165.2
24	47.7	47	90.0	70	129.8	93	166.7
25	49.6	48	91.8	71	131.4	94	168.3
26	51.5	49	93.6	72	133.1	95	169.8
27	53.4	50	95.4	73	134.7	96	171.4
28	55.3	51	97.1	74	136.3	97	173.1
29	57.2	52	98.9	75	137.9	98	174.6
30	59.1	53	100.6	76	139.6	99	176.2
31	60.9	54	102.3	77	141.2	100	177.8
32	62.8	55	104.1	78	142.8		

V. Benedict's Method.

"ESSENTIALS OF CHEMICAL PHYSIOLOGY"—HALLIBURTON.

Benedict's quantitative reagent is an alkaline solution of copper sulphate containing potassium thiocyanate. This is kept boiling, and the sugar solution is run into it from burette until the blue colour disappears; the thiocyanate forms a white precipitate with the cuprous oxide formed, so that no red cuprous oxide obscures the blue tint.

Preparation of the Solution.—Sodium citrate, 200 grammes, sodium carbonate (crystalline), 200 grammes (or anhydrous sodium carbonate, 75 grammes), and potassium thiocyanate, 125 grammes, are dissolved in hot water; this when cool is made up with distilled water to 800 c. c., and filtered.

18 grammes of pure copper sulphate are then dissolved in 100 c. c. of water, and poured slowly with constant stirring into the first solution. 5 c. c. of a 5 % solution of potassium ferrocyanide are then added as an additional precaution to prevent any deposition of cuprous oxide; finally the total volume of the mixture is made up to 1000 c. c. with distilled water. 25 c. c. of this solution are reduced by 0.05 gramme glucose.

Analysis—3 or 4 grammes of anhydrous sodium carbonate are placed in a 300 c. c. flask, then 25 c. c. of the above solution. This is kept boiling over a small flame, and the sugar solution run in from a burette until the last trace of blue colour disappears. The amount used for this purpose is then read off.

Benedict's qualitative reagent (p. 19) contains sodium citrate, 175 grammes; anhydrous sodium carbonate, 100 grammes in about 150 c. c. of water; to this are added 17.3 grammes of CuSO_4 in 100 c. c. of water, the whole being then made up to 1 litre.

Calculation—This may be illustrated by an example. If the reading on the burette is 10 c. c. then this amount of urine contained 0.05 gramme of glucose, therefore 100 c. c. contains 0.5 gramme. Should it be found that the first titration gives a result indicating that the percentage is greater than 1 per cent., the urine should be diluted quantitatively so as to bring the concentration between 0.5 and 1.0 per cent. A further example will make the calculation clear. If the sugar solution had been diluted 1-in-5 (that is 10 c. c. with 40 c. c. of water), and the reading of the burette was 10 c. c., then

10 c. c. of the diluted solution = 2 c. c. of the original solution.

2 c. c. of the original solution contain 0.05 gramme of glucose.

$$0.05 \times 100$$

1 c. c. of the original solution contains $\frac{\quad}{2}$ gramme of glucose,

2

= 2.5 per cent glucose.

Estimation of other Reducing Sugars.—The same two methods may be used for the estimation of other reducing sugars; the only difference is in the final calculation:—

10 c. c. of Fehling's solution (or 25 c. c.

of Benedict's solution)

= 0.05 gramme of Glucose.

= 0.053 gramme of fructose.

= 0.0676 gramme of lactose.

= 0.074 gramme of maltose.

"ESSENTIALS OF CHEMICAL PHYSIOLOGY"—HALLIBURTON.

VI. Ling and Rendle's Volumetric Method.

The sugar solution (diabetic urine) is allowed to run from a burette into a known volume of boiling Fehling's solution. The end point, i.e., the complete reduction of the cupric salt, is recognised by means of a solution of ferrous thiocyanate. When a drop of this indicator is placed on a slab, and a drop of a solution containing a cupric salt brought in contact with it, oxidation of the ferrous salt occurs, with the immediate production of the well-known red colour of ferric thiocyanate.

Preparation of the Indicator.—One gramme of ferrous ammonium sulphate and 1.5 grammes of ammonium thiocyanate are dissolved in 10 c. c. of water at a moderate temperature and immediately cooled; 2.5 c. c. of concentrated hydrochloric acid are then added. The solution so obtained has invariably a brownish-red colour, due to the presence of ferric salt, which latter must therefore be reduced. This is effected by shaking with a small quantity of zinc dust.

Preparation of Fehling's Solution.—Solution No. 1—69.278 grammes of crystallised copper sulphate are dissolved in water, and the solution made up to 1 litre.

Solution No. 2—346 grammes of crystallised potassium-sodium tartrate (Rochelle salt) are dissolved in hot water, mixed with 142 grammes of caustic soda also dissolved in water, and after cooling made up to 1 litre.

Equal volumes of these two solutions are accurately measured and mixed in a dry flask before use. 10 c. c. of the mixed solution are equivalent to 0.05—gramme of glucose.

Analysis.—Freshly mixed Fehling's solution (10 c. c.) is accurately measured into a 200 c. c. boiling flask, diluted with about an equal quantity of water, and raised to boiling point. The urine is diluted with water and placed in a burette; the dilution should be adjusted so that 20 to 30 c. c. of the diluted urine are required to reduce the 10 c. c. of Fehling's solution. In the first experiment the urine should be diluted with nine times its volume of water. This diluted urine is then run into the boiling liquid in small amounts, commencing with 5 c. c. After each addition of sugar solution the mixture is boiled, the liquid being kept moving. About a dozen drops of the indicator are placed on a porcelain slab, and when it is judged that the precipitation of cuprous oxide is complete (that is, when the blue colour of the solution is disappearing), a drop of the liquid is withdrawn by a clean glass rod or by a capillary tube, and brought in contact with the middle of one of the drops of the indicator on the slab. The end point is reached when the mixture ceases to give a red colour with a drop of the indicator. It is essential to perform the titration as rapidly as possible, as an atmosphere of steam is then kept in the neck of

the flask and the influence of atmosphere oxygen avoided. At the final point the liquid is boiled for about ten seconds. As in all volumetric methods the first titration may only give approximate results and a second will then be necessary to establish accurately the end point. Each titration should take from two to three minutes.

Example.—Suppose that the urine has been diluted tenfold with water, and that 20 c. c. of the diluted urine are found necessary to reduce the 10 c. c. of Fehling's solution; this will be equivalent to 2 c. c. of the original urine, and that amount will therefore contain 0.05, gramme of sugar; 1 c.c.

0.05 0.05X
will contain— and 100 c. c. will contain—100—is equal to
2 2
2.5 grammes of sugar.

Pavy's modification of Fehling's solution is sometimes used. Here ammonia holds the cuprous oxide in solution, so that no precipitate forms in boiling Pavy's solution with a reducing sugar. The reduction is complete when the blue colour disappears. 10 c.c. of Pavy's solution, is equal to 1 c. c. of Fehling's solution which is equal to 0.001 gramme of glucose.

VII. Polarimeters.

Polarimeters are instruments for determining the strength of solutions of sugar, albumin, etc., by the direction and amount of rotation they produce on the plane of polarised light. They are often called saccharimeters, as they are specially useful in the estimation of sugar. The following is the one most frequently employed at the present time.

Laurent's Polarimeter.—Instead of using daylight or the light of a lamp, monochromatic light (a sodium flame produced by volatilising common salt in a colourless gas flame) is employed; the amount of rotation varies for different colours; and observations are usually recorded as having been taken with light corresponding to the D or sodium line of the spectrum. The essentials of the instrument are a polariser, a tube for the solution, and an analyser. The polarised light before passing into the solution traverses a quartz plate, which, however covers only half the field, and retards the rays passing through it by half a wave-length. In the 0° position the two halves of the field appear equally illuminated; in any other position, or if rotation has been produced by the solution when the nicols have been set at zero, the two halves appear unequally illuminated. The amount of rotation produced by the solution is ascertained by rotating the analyser until the two halves of the field are once more equally illuminated. This is measured in degrees by a scale and vernier attached to the instrument.

The specific rotatory power of any substance is the amount of rotation in degrees of a circle of the plane of polarised light produced by 1 gramme of the substance dissolved in 1 c. c. of liquid examined in a column 1 decimetre long.

If a = rotation observed.

w = weight in grammes of the substance per cubic centimetre.

l = length of tube in decimetres.

1 In the most delicate instruments the field is divided into three parts or into two concentric circles.

$(a)_d^t$ = specific rotation for light with wave-length corresponding to the D line (sodium flame) at temperature t .

Then $(a)_d^t = \frac{+a}{H \frac{w}{l}}$.

In this formula + indicates that the substance is dextro-rotatory,—that it is levo-rotatory.

If, on the other hand, $(a)_d^t$ is known, and we wish to find the value of w , then

$$W = \frac{a}{(a)_d^t \times l}, \text{ and the percentage amount is}$$

$$p = \frac{a \times 100}{(a)_d^t \times l}$$

If, for example we find the rotation of a urine in a 2-decimetre tube + 2.12° and the $(a)_d^t$ of glucose is 53°, then

$$p = \frac{2.12 \times 100}{53 \times 2} = 2$$

that is, the urine contains 2 per cent. of glucose.

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Acetone.—Qualitative tests.

1. *Frommer's test.*—To 10 c. c. of urine add 1 gramme of solid potassium hydrate. Before solution has occurred add 10 to 12 drops of a solution of salicyl aldehyde in absolute alcohol and warm the mixture to about 70° C. At the zone of contact of the alkali and urine an intense purplish red ring develops in the presence of acetone. This test is specific for acetone, and gives no reaction with diacetic acid; it is extremely delicate and it may be performed without distilling the urine, as must be done with the older tests. It is recommended for routine use.

2. *Legal's test for Acetone*—To 5 c.c. of urine add a small crystal of sodium nitroprusside. After solution has occurred (the urine should be cool) sodium or potassium hydrate is added in excess.

A ruby red colour appears, which fades rapidly to yellow. This colour is given both by creatinin and acetone. Therefore, before the ruby red colour has faded glacial acetic acid is added in excess. In the presence of acetone the red colour will change to a purple or violet tint, while, with creatinin, the red colour will fade at once to a clear yellow or green.

This test is positive with diacetic acid. It will be positive with the untreated urine only when considerable amounts of acetone are present. To render the method more delicate, the distillate from the urine should be used.

Diacetic acid.—Qualitative tests.

1. *Arnold's test.*—Two solutions are necessary.

Solution I.

Paramidoacetophenon	...	...	1 gr. n.
Distilled water	...	...	100 c.c.
Concentrated HCl.	...	...	2 c.c.

The paramidoacetophenon is dissolved in the water with constant shaking and the HCl is added drop by drop till the yellow solution becomes water clear.

Solution II. Sodium nitrite...1 per cent.

Aqueous solution.

To 10 c. c. of Solution I, add 5 c. c. of Solution II and 15 c. c. of urine. Drop in one or two drops of concentrated ammonium hydrate. A non-characteristic brownish red colour appears. Add an excess of strong HCl. If diacetic acid be present a beautiful purple colour develops. In the absence of diacetic acid the red colour changes to yellow on the addition of acid.

This test is not positive with acetone, with B-oxybutyric acid, nor with drugs.—It is thus much preferable to the ferric chloride test.

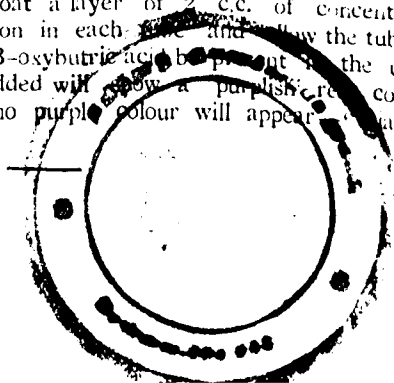
2. *Gerhardt's test for diacetic acid*—To 10 c. c. of urine a ten per cent solution of ferric chloride is added drop by drop till no further clouding is noted.

The mixture is then filtered. A few drops more of ferric chloride are then added to the filtrate. If diacetic acid be present, the filtrate should now show a claret red precipitate. This colour may be due not to diacetic acid, but to certain drugs, many of which are used by some people in the treatment of diabetes and which are certainly in common use. The chief of these are salicylic acid, diuretin, aspirin, salol, phenacetin, acetates and formates. If the solution be boiled the red colour due to the volatile diacetic acid will fade, whilst that due to all of the above drugs (except the acetates and formates) will remain.

Oxybutyric Acid Qualitative Test.—Hurt's test. This test is not very delicate, but it is the most suitable for clinical work.

To 20 c. c. of urine add an equal quantity of water and a few drops of acetic acid.

The mixture is boiled down to about 10 c. c. in volume (to remove acetone and diacetic acid). The volume is then brought up to 20 c. c. with water and 10 c. c. of the mixture is put in each of two test-tubes. To one of these add 1 c. c. of hydrogen peroxide, warm gently and allow to cool. Then add to each tube 0.5 c. c. of glacial acetic acid and a small crystal of sodium nitroprusside; next float a layer of 2 c.c. of concentrated ammonium hydrate on the solution in each tube and allow the tubes to stand for four or five hours. If B-oxybutyric acid be present in the urine, the tube to which peroxide was added will show a purplish red contact ring, whilst in the control tube no purple colour will appear. Diabetes by Ernest E. Waters.



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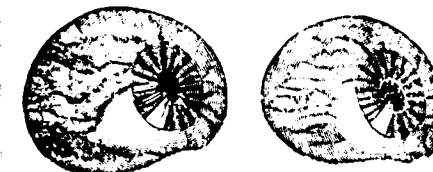
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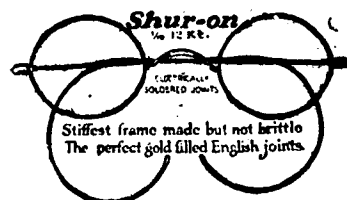
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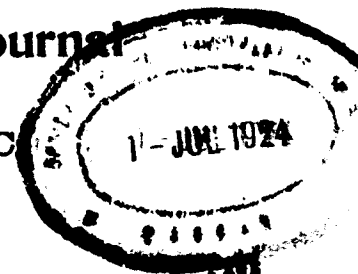
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FEBRUARY, 1924.

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A Monthly Medical Journal

EDITED BY
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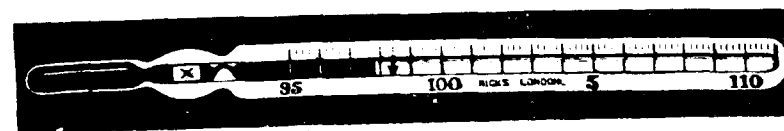
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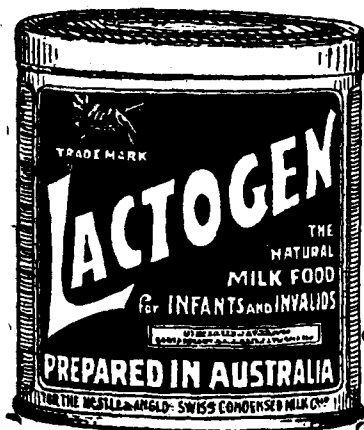
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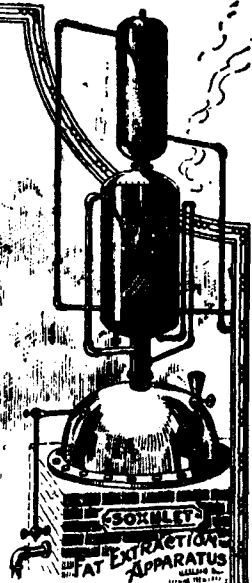


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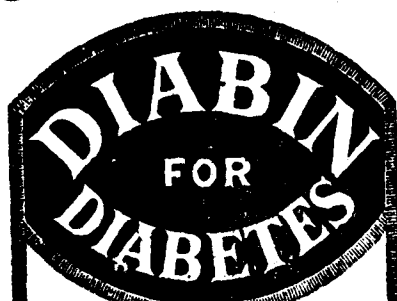
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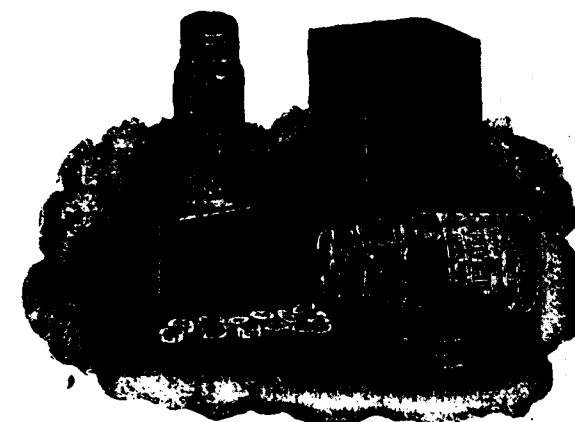
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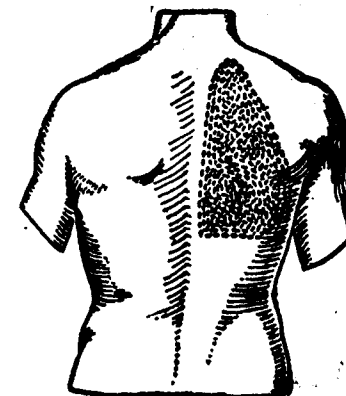
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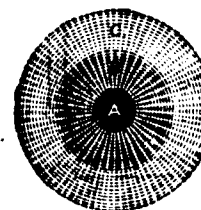


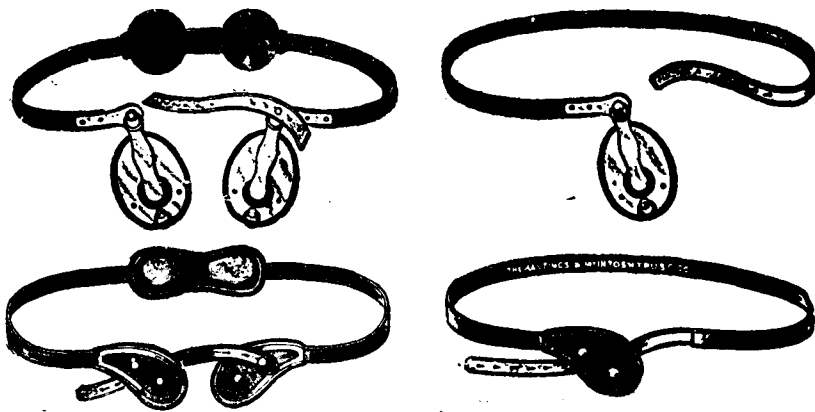
Diagram represents inflamed area. In zone "C" blood is flowing freely through underlying vessels. This forms a current away from the Antiphlogistine, whose liquid contents, therefore, follow the line of least resistance and enter the circulation through the physical process of endosmosis. In zone "A" there is stasis, no current tending to overcome Antiphlogistine's hygroscopic property. The line of least resistance for the liquid exudate is therefore, in the direction of the Antiphlogistine. In obedience to the same law exosmosis is going on in this zone, and the excess of moisture is thus accounted for.



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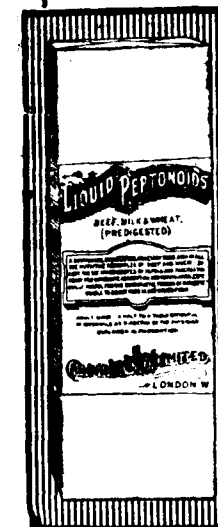
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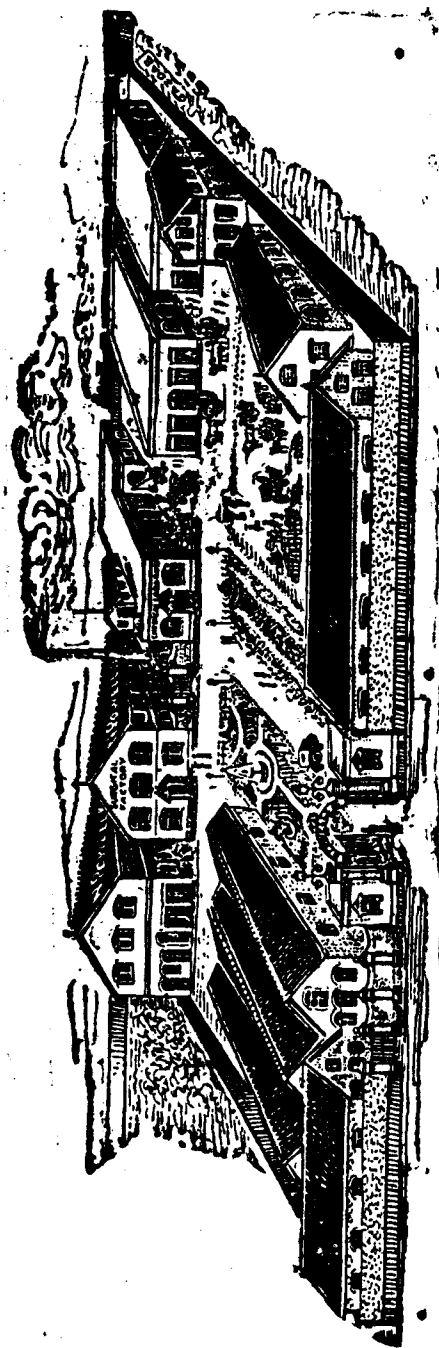
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The Antiseptic

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FEBRUARY, 1924.

ANNUAL
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ORIGINAL ARTICLES.

* A DISCUSSION OF THE ETIOLOGY OF RICKETS

By

ASHUTOSH ROY, L.M.S., HAZARIBAGH.

Rickets is included in the category of "deficiency diseases" which convey into our mental horizon, the picture of an ill-nourished child, the effect of mal-nutrition.

The victims of the various deficiency diseases are generally ill-nourished. The peculiarity of rickets amongst this group of diseases is that it attacks ill-nourished children of the poor, living in insanitary surroundings as well as apparently healthy children of the well-to-do living in sanitary surroundings.

Our idea of naming certain diseases as *deficiency diseases* first used by Girjut has considerably changed. We now know that deficiency diseases may be caused by upsetting of the metabolic cycle by any defective link in the chain. Amongst the various factors may be mentioned, endocrine insufficiency, vitamine defect in diet, upsetting of mineral metabolism, defective secretion of digestive enzymes, upsetting of the physiological relative quantity between the three proximate food principles, want of protein of good biological value, defective or excessive calories of food ingested. These are some of the links in the chain, defect or absence or excess of any one of these factors may upset the metabolic cycle. As a result there is imperfect digestion and assimilation, malnutrition and marasmus; toxins are generated, acidosis results, body resistance is lowered, liability to infection and intoxication increases; exo-toxins or endo-toxins affect the system, bacteria and bacilli find ready entrance into the organism where they lodge and flourish, various tissues and organs are

* Specially contributed to the 'Antiseptic.'

affected and under certain conditions deficiency diseases result. Unless we take this broad view, we cannot satisfactorily explain or harmonize the various known etiological factors of deficiency diseases.

The central feature in rickets is want of calcification or mineralization of developing bone. The close association of calcium and phosphorous in bone formation is universally admitted. *Rickets is due to upsetting of the metabolism of calcium and phosphorous.*

All varieties of malnutrition occurring during infancy and early childhood terminate in rickets. Malnutrition in embryonic life may cause rickets under certain conditions.

The sequence of events resulting in rickets are as follows: malnutrition, acidosis, upsetting of calcium and phosphorous metabolism, rickets.

If we remember these salient features, these successive course of events, we can satisfactorily explain all the various theories which have arisen from time to time with reference to the etiology of this disease.

We must recapitulate that the immediate cause of rickets is upsetting of calcium phosphate metabolism. Our knowledge of endocrinology, vitamins and mineral metabolism, three of the latest advances in modern medicine, being still in its infancy, we are still to know the special etiological factor or factors which upset phosphate of calcium metabolism and cause Rickets.

The various theories of Etiology of Rickets.

These may be conveniently grouped as follows:—

Group (I) The hereditary or parental theory of Rickets.

Group (II) The environment theories:—

- (a) The domesticated theory of Von Hansmann.
- (b) The hygienic theory of Noel Paton.
- (c) The want of muscular exercise theory of Findlay.
- (d) The respiratory theory of Kassovitch.

Group (III) The infective theory of Cameron.

Group (IV) The intoxication theories:—

- (a) The oxypathic theory of Stelzner.
- (b) The theory of excessive fatty acids in the bowels.
- (c) The lactic acid theory.
- (d) The theory of acid condition of the blood of Wachsmuth.
- (e) The intestinal intoxication theory.
- (f) The acidosis theory of Pritchard.

Group (V) The metabolic theories: (defective food factors.)

- (a) Defective lime in food.
- (b) Defective absorption of lime.
- (c) Defective vitamine theory of Mellanby.
- (d) Defective calory in diet.
- (e) Relative over-feeding.
- (f) One-sided dietary.
 - (1) Excessive and prolonged use of proprietary milk food.
 - (2) Use of cow's milk with excess of starch.
 - (3) Use of cow's milk without starch.
 - (4) Wrong balance between protein and starch.
 - (5) Use of diet deficient in fat protein and vitamine.
- (g) Inorganic-Ion theory.

Group (VI) Endocrine insufficiency theory.

It may be noted that clinical or experimental proofs are not wanting for or against each of the theories.

It may also be stated that in different countries and under different conditions, varying etiological factors predominate. Thus catarrhal conditions, and infections of the respiratory tract play a leading role in cold countries, just as malaria and gastro-intestinal infections are important etiological factors in tropical India. From a practical point of view it is desirable to ascertain what are the commonest and what are the rare etiological factors in a particular country. For the treatment of individual cases, every known etiological factor should be remembered and mentally reviewed and the cause or causes which led to the disease should be carefully enquired and ascertained.

Group I.—The hereditary or parental theory.

It explains malnutrition in embryonic life. The factors are:—

- (a) Inherited or acquired endocrine insufficiency in the parents often transmit a functional inferiority of such organs in the offspring. Thyro-parathyroid, for example, is chiefly concerned with mineral metabolism, and, if defective, will upset it and may lead to Rickets.
- (b) The maternal blood of the embryo and the mother's milk at child birth should satisfy the calcium and vitamine need of the child. If these are defective in the food of women during gestation and lactation there will be malnutrition which may lead to rickets in infants and children of such mothers. It may be noted that vitamine A is associated with calcium and fat metabolism.

Clinical proofs of the Hereditary theory of Rickets :—

Hess and Unger in New York found that 90% of Negro children develop rickets, while American children living under identical conditions and taking the same kind of food do not develop it.

Group II.—The environment theories.

Von Hansmann found rickets amongst children of poor people living in slums of large cities, in ill-ventilated and ill-lighted houses with over crowding and deficient dietary. Accordingly he started his "*domesticated-theory*."

Noel Paton has performed animal experiments in support of his theory against the vitamine deficiency theory of Mellanby. He kept one set of puppies in the laboratory and fed these with diet rich in vitamine A; another set he kept in the open country and fed them with diet deficient in vitamine A. The former showed rickets while the latter did not. Accordingly he started his "*hygienic theory*" of rickets.

Recently after the European war, Pirquet of Vienna tried the effect of sun-light (ultra-violet rays) on war-babies, a large number of whom showed rickets. To one set he gave diet rich in vitamine A, to the other set he gave diet poor in vitamine A, but exposed them daily to ultra-violet rays of the sun for sometime. Both sets improved and got cured.

Further experiments on that line proved that not only ultra-violet rays of the sun, but also exposure to rays from mercury vapour lamp cured rickets. The effect on the disease is the same as administration of vitamine A in the infant's diet.

The usual explanation that is given is that *infants and children synthesise vitamine A by the process of photo-synthesis*, though it is generally believed that vitamins as a rule can only be synthesised by plants and not by animals.

Kassovitch thinks that deleterious respiratory influences or foul impure air (odour of the poor) plays a direct part in the etiology of rickets. Accordingly he started his "*respiratory theory*." No doubt foul air depresses vital functions and leads to malnutrition and rickets.

Findlay conducted some animal experiments to prove that want of muscular exercise plays a leading part in the etiology of rickets. No doubt exercise creates a demand for energy, an output of kinetic energy resulting in increased oxidation and better digestion. Accordingly he started his "*want of muscular exercise theory*" of rickets.

Findlay with Paton and Miss Fergusson found in their study of 500 rachitic children that overcrowding at home, want of muscular exercise, want of open air and sunlight *without deficient diet*, are the main etiological factors in rickets.

Impure air, want of sunshine and fresh air, want of exercise, want of surface stimulation (due to absence of cool fresh air, free ventilation and bath) are no doubt important etiological factors as they depress the vital functions, lead to malassimilation of food and malnutrition in infants and children, and if associated with *relative physiological overfeeding* may lead to rickets.

If there is no output of kinetic energy as in debilitated states, want of muscular exercise, and too much tight clothing, the physiological use of food is seriously restricted. There will be lessened incentive for oxidation processes, incomplete combustion of otherwise normal physiological quantity of food which would result in *relative overfeeding*, defective digestion, malnutrition leading to rickets.

Group III—The infective theory of Cameron.

It is a well-known clinical fact that rickets often follow infections in infants and children. Infection is an important factor during pregnancy and lactation in the mother affecting the health of the child.

Every variety of infection *e.g.*, infectious fevers, repeated attacks of the respiratory or gastro-intestinal tract, general infections like tuberculosis, syphilis, malaria and colli infection of the bowels, occurring in infants and children may be followed by rickets. No specific organism is thus found.

Cameron suggests that factors like overcrowding, want of fresh air and sunshine, lack of physical exercise, etc., lower the infants' and children's resistance to infection. He suffers from repeated catarrh of the respiratory tract. His muscles waste and loose tone. He gets excessive perspiration, coldness and blueness of extremities (defective oxidation) and gradually rickets appear.

Infection leads to depression of vital functions, injuriously affects the endocrine system, upsets digestion and assimilation, leads to malnutrition and rickets. When attacking infants and children, infection is an important etiological factor leading to rickets.

Like unhygienic conditions, infection cannot be the specific etiological factor in rickets.

Group IV—Intoxication theories.

*The oxypathic theory of Stelzner:—*There is no doubt that any condition which interferes with oxidation *e.g.*, want of fresh air, mechanical obstruction of the respiratory passages by adenoids, embarrassment of the respiratory movements by tight clothing (*e.g.*, infants swaddled up in tight clothing so that movement of the limbs is restricted) would lead to "anoxemia" or defective oxidation. This will

restrict the completeness of the metabolic processes. Further any interference with due oxidation of food elements to their normal end products (carbonic acid, urea, water) will conduce to the formation of semioxidized acid bodies of large molecules and if continued long and not neutralised will lead to a dangerous rise of H. Ion concentration. Thus there will be relative physiological overfeeding, and imperfect digestion, acidosis leading to rickets.

The theory of *excessive fatty acids in the bowels* finds clinical proof by the fact that soap stools are common in rickets. Two factors are concerned in this pathological process. First there is the overfeeding of fat more than the organism needs or can digest. Secondly the upsetting of fatty metabolism resulting in formation of excessive fatty acids in the bowels. It is a form of local accumulation of special acids and unless neutralised raises the H. Ion. concentration and leads to systemic acidosis or acidosis proper leading to rickets.

We know that calcium is intimately associated with metabolism of fat and both calcium and fat metabolism are intimately connected with vitamine A in the diet.

The lactic acid theory of rickets:—According to this theory there is accumulation of excess of lactic acid produced in condition of defective oxidation of food like milk. But this acid like other acids *e.g.*, uric, oxalic, butyric, propionic, as also sulphuric and phosphoric acids are produced as a result of faulty metabolism and unless neutralised leads to acidosis and rickets.

We know that loss of calcium from the kidneys is increased by injection of large quantities of lactic acid. We further know that sodium phosphate combines with lactic acid.

The theory of excessive acids in the blood (Wachsmuth):—Blood is always alkaline. Complete acid condition of blood is incompatible with life. It is a condition of relative increase of acid salts in the blood quickly neutralised by the reserve alkali. But the acid-base equilibrium of the body is upset, leading to acidosis and rickets.

The intestinal intoxication theory supposes that as a result of faulty metabolism, acid toxins (the end products of defective digestion) are produced in the intestines and unless neutralised, lead to a condition of acidosis and rickets.

The acidosis theory of Pritchard:—Pritchard has taken great pains to prove that the various etiological factors of rickets start with malassimilation and malnutrition. All conditions of malnutrition occurring in infants and children up to a certain age lead to acidosis. This upsetting of the acid-base

equilibrium will be corrected by neutralisation with alkalies and alkaline earths, specially calcium before any of it is available for bone formation. That is, in presence of acidosis, there is calcium starvation of the bones leading to rickets.

He admits that calcium will be one of the last basic elements which constitute the alkaline reserve of the body to be used for such purposes. We know that acids formed in the blood always prefer neutralisation by salts of alkalies (Na and K.) than by alkaline earths (calcium and magnesium.)

The weak link in the chain of his argument is that in absence of our knowledge, we don't know what acids, local or general, will prefer calcium salts for neutralisation. Some authorities believe that the fatty acids prefer calcium, considering that the latter is associated with fat metabolism; others believe that lactic acid prefers calcium and phosphorus, but we know that it prefers sodium phosphate over calcium phosphate. Others believe that it is no local accumulation of any particular acids but acidosis of the body draws the alkaline reserve, depletes calcium with the rest and leads to calcium starvation. The fact that bone requires both calcium and phosphorus still complicates matter. Until we know in full the metabolism of calcium phosphate, until we know what acids will prefer this salt over others to be neutralised, we can't determine with certainty the etiology of rickets. All we can say is that there is local accumulation of acid or acidosis of the system which produces lime and other alkaline salt starvation and leads to rickets in infants and children.

Group V—Metabolic theories of rickets.

We have seen how the different etiological factors lead to defective digestion, malnutrition and acidosis. In this group we will consider what defects in quantity or quality of diet lead to malassimilation and acidosis. Considering that rickets occur amongst ill-nourished and apparently well-nourished children living in sanitary and insanitary surroundings, there is not the least doubt that food factor must be a very important etiological factor and hence its inclusion in the group of deficiency diseases.

Theory of deficient lime in diet:—Without lime bone cannot be formed. Since there is an adequate supply of lime in breast milk and excess in cow's milk *normally*, want of lime *per se* cannot be the cause of rickets. None the less deficiency of lime in a small number of cases cause the disease *e.g.*, when lactating mother's milk is deficient in lime or when certain proprietary milk foods are exclusively used or when bad milk of unhealthy cows so common in India for example are used in infant feeding.

Theory of defective absorption of lime :—This is a more reasonable theory. It is a well-known clinical fact that more lime is excreted out of the system with the faeces in rickets. This shows that there is defective absorption of lime. Possibly the lime lying in the intestines is seized to neutralise the acid bodies in condition of local formation or accumulation of acids in the intestines and so nothing is absorbed and there is lime starvation.

Administration of extra calcium salts is followed in rickets by an equivalent output in the urine. This shows that the extra lime is absorbed but it is used up in correcting the acid base equilibrium in the blood, upset the acidosis and all the lime taken and thus neutralised is excreted out of the system with the urine.

Further, we know that calcium metabolism is intimately associated with the integrity of the Thyro-parathyroid gland and vitamine A in diet. Thus defective vitamine in diet or hormone secretion may be factors in preventing proper assimilation of lime.

Theory of defective vitamine A in diet :—Mellanby has taken great pains and performed several experiments to prove the theory. There is no doubt that vitamine A and B are important factors in the metabolic chain as well as in calcium and phosphorous metabolism (as we find in rickets and beri-beri). Hess and Unger as well as Noel Paton have successfully performed experiments which go against the theory.

Theory of defective calory intake :—This is no doubt a potent factor in producing malnutrition in infants and children of poor people. Insufficient quantity leads to malnutrition due to semi-starvation, acidosis and rickets. Thus in mothers getting insufficient diet during lactation, their milk is poor and will adversely affect the health of the infant. Poor cow's milk giving insufficient nutrition will lead to the same result.

Relative overfeeding theory :—We have already discussed one aspect of relative overfeeding when the caloric need of the infant is lowered under certain conditions.

General overfeeding takes place in the case of infants and children of well-to-do people.

Overfeeding leads to malassimilation, malnutrition and acidosis.

Theory of one-sided dietory :—Excessive and prolonged use of patent milk foods make the diet deficient in quality (deficient salt and vitamine) if not in quantity.

Use of cow's milk with excess of starch upsets the physiological balance of the three food principles and leads to malassimilation.

Conversely Hess and Unger have shown that milk up to a certain age in infants keeps them in good health. After that period milk alone is insufficient to meet the greater need of the child and unless starch is added growth is arrested and rickets ensue, which is not relieved by the addition of cod-liver oil in the diet.

Use of diet deficient in lime, fat and protein has been shown by Mellanby to cause rickets. In case of proteins, they must be of good biological value. Thus gelatine, gliadin and zein are not of good biological value. Similarly vegetable fat and lard are wanting in vitamine A. Again wrong balance between protein and starch upsets digestion and cause rickets.

Inorganic Ion theory of Rickets.

Shermane and Pappenheim have proved by actual experiment that if the diet is so modified that a little potassium phosphate replaces calcium lactate in it, rickets do not occur. We know that potassium is necessary for growth of cells and phosphorus for growth of bones.

According to those two authorities, protein and vitamine are of no importance in the etiology of rickets. They suggest that disturbance of quantitative relation of the "Inorganic Ions" or the complete absence of one of them is the determining factor when the balance between organic foodstuffs in diet is not physiologically perfect.

We thus see that all the above food factors are different links in one aspect of the metabolic chain e.g., quantity and quality of foodstuff and their proper physiological relation quantitatively. Any defective link in the chain upsets the whole metabolic cycle, leads to malassimilation, malnutrition, acidosis and rickets.

Group VI.—Defective Endocrine theory.

We have seen how defective endocrine organs in the parents may transmit a defective endocrine system in the offspring (*Hereditary endocrine defect*).

Acquired endocrine defect may follow from

- (a) Want of vitamins in diet.
- (b) Quality and quantity of food, if defective.
- (c) Unhygienic mode of living.
- (d) Defective oxidation.
- (e) Infections.
- (f) Intoxications.

These facts are co-related.

Thus we have discussed all the etiological factors of rickets. None are specific. Our want of detailed knowledge of mineral metabolism and their

relation with hormones and vitamins prevent us to understand the specific etiological factor or factors which are intimately and directly concerned with the different aspects of calcium and phosphorus metabolism, upsetting of which causes rickets.

Special etiological factors of rickets in India.

We find in India as in the West rickets occurring amongst well-fed and ill-fed children and infants.

As to *heredity*—our social custom of early marriage is a special factor. Our child-mothers cannot bear the strain of gestation and lactation, specially amongst the middle class and well-to-do families. Their health suffers, their milk is poor and their offsprings suffer accordingly.

As to *environments* our infants and children rarely suffer from want of sun-shine. The other factors may be present.

As to *defective oxidation*.—The fashion of putting too much clothing on our infants and children in apish imitation of European mode of dressing, is quite unsuited for our country and may be a potent factor in well-to-do families.

As regards *infection*—malaria, colli infection gastro-intestinal disturbances are potent factors. The progressive increase of tuberculosis in India, the increase of venereal disease amongst floating population, the attack of infectious fevers in early childhood, the increase of dust and smoke of the atmosphere of manufacturing towns making children susceptible to frequent cold and respiratory attacks, are potent factors.

As regards *acidosis and intestinal intoxication* they are perhaps more important in our country than respiratory attacks. Infantile liver is so common.

As regards *defective dietary*—one-sided dietary is the rule in India. Deficient protein and vitamins (in stall-fed cow's milk) adulterated milk, butter milk, excessive starch in the milk, excessive use of proprietary milk foods (so prevalent now-a-days) defective balance of the three-fold principles, defective assimilation of lime, are potent factors.

The absence of good milk of healthy cows is particularly felt all over India.

As regards *Endocrine factors*:—It is too much to expect well-developed endocrine system in the large number of child-mothers of India. Acquired endocrine deficiency due to infectious fevers in early childhood are rather frequent.

Conclusion.—For the purpose of successful treatment of rickets we must therefore take particular care to ascertain the history of the case

and critically judge what factors have caused the disease. All varieties of malnutrition in infants and children should be promptly corrected to avert the disease.

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2. Abdul Wahed's article on Vitamins in the *Indian Medical Record* February 1923 and subsequent issues.
3. The writer's article on Acidosis in the *Indian Medical Record*, March 1923.
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MY CONCLUSIONS OF THE ETIOLOGY AND TREATMENT OF BERI-BERI*

By

S. SUBBA RAO, L. M. P.

In spite of rapid advancement as regards the etiology and treatment of many tropical diseases such as Kala-azar, Dysentery and Leprosy, much light was not thrown on beri-beri. It is one of those diseases whose victims are able-bodied young adults who are snatched away from use suddenly with alarming rapid cardiac and paralytic symptoms. When I was a student at Vizagapatam Medical School, I had the opportunity of studying many cases of different varieties at bed-side. One case where general anasarca rapidly developed within two days and became fatal after two hours of his being admitted into the hospital in spite of the best available treatment. Subsequently I settled at Madras and for the last 12 years of my practice here I had not the occasion of treating or studying one single indigenous case. I collected up to now fifteen cases, all being imported from Circars and Burma. Recently I treated two worse cases of dropsical variety with cardiac distress and dilatation.

I am not going to discuss here the various theories and causes of this disease, but merely point out my impressions and conclusions thereby with the treatment I pursued with some beneficial results. Any text-book on tropical medicine deals with a detailed list.

Beriberi is a specific form of multiple neuritis due to an unknown virus which lies dormant somewhere within the human body in the epidemic and endemic areas. When conditions become favourable it becomes

* Specially contributed to the *Antiseptic*.

active creating toxins which chiefly have an affinity to the heart, motor and vaso-motor nerves.

Low-lying damp deltas with humidity around favour the growth of the virus. It is essentially a disease of malnutrition due to the deficiency of vitamins and mineral salts. Scurvy is a predisposing factor since many sailors suffer from it so much so it is called Ship beri-beri. How it is communicable from man to man it is difficult to conclude. Probably it is through one of the blood-sucking insects which infest persons of damp fields, ships, seacoasts with verdure and insanitary dwellings and camps.

Bad-stored food, flesh, stale-shelled rice prepared from mills and in fact every food where there is a natural deficiency of vitamins and mineral salts causes degeneration of the heart muscle and vasomotor nerves and thereby the power of resistance to the virus is enfeebled and the ever-watching foe lurking somewhere in the human body in the epidemic districts finds the field favourable for its campaign and invasion. It then becomes active sending its troop of toxins to attack the enfeebled nerves and the heart.

A month ago a woman aged 40 years, Brahman by caste, staple food being milled rice, came under my observation and treatment from Vizagapatam District where in some parts beriberi is endemic. The general appearance suggested to me the possibility of beriberi and my views were strengthened as she came from an endemic area. I was not prejudiced by my views and I carefully examined her.

The following were the chief signs and symptoms:—There was general anasarca. Her face was puffy and heavy. Arms, hands, legs, trunk and almost all dependent parts were distended with oedema. The oedema was firm and there was slight pitting on hard pressure. There was marked tenderness on pressure through the oedema over the calf muscles. Knee jerk was absent. There was tingling and numbness over the skins and fingertips. The amount of urine was reduced to a few ounces.

There was slight trace of albumen. The cardiac impulse was diffuse. The pre-cardiac area was enlarged to the right. Systolic murmurs were present in all areas. The heart was very irritable with a quick gallop rhythm with the signs of dilatation with breathlessness and cyanosis. I concluded the case to be beriberi and I directed the treatment on that line.

Treatment:—The profession gradually began to learn that these diseases cannot be treated mainly with the drugs alone. I put the patient on a strict salt-free diet which is very important in these cases. As a

matter of fact we do not want at all artificial sodium chloride if we cultivate the habit of eating food raw and natural. Fresh cow's milk without being boiled was the sole diet with fresh orange juice. I also instructed the patient to take tender fine husk of rice including the pericarp in small quantities of $\frac{1}{4}$ ounce mixed with plantain tree juice half an ounce three times a day. I have seen once in Vizag District an old Indian woman prescribing lotus flower juice and seeds to a patient with marked improvement. I have accordingly added this also to my list of dietary. My aim in prescribing this diet is to supply mineral salts in their natural state as they exist. Gradually I added only wheat cakes baked from flour prepared from raw wheat grain with pericarp on. The improvement was marvellous. Within four days almost the whole dropsy disappeared, the heart improved. She is still under my observation. She is now completely free from every symptom of the disease. Only she has a longing for pungent chutnies with salt. Poor woman! Can I sanction? She was disappointed when I told her that she should continue this diet for two months more. Even then very little salt and chillies she has to consume. Rice is a bad food though unshelled. Other important appendices to the treatment is to remove the patient at once from the endemic area to a dry inland locality. Good dry clothing and sanitary lodgings are necessary.

On the first day of the patient coming under my observation when the cardiac symptoms were alarming I gave a small dose of lequittales—a digitalis preparation which made the patient worse. Subsequently I gave four drops of one p.c. solution of Nitroglycerine twice within one hour with no relief.

I at once let out four to five ounces of blood from the veins at the elbow and on that day the patient was in fact saved by this process. I do not know what would have become of the patient if I had not had recourse to this process. Just like in Eclampsia, blood-letting is a sheet-anchor in these cases rapidly relieving the engorgement of toxinated blood in the heart and vessels. I did not give any medicine internally to the patient except a placebo with little ferri-ataman, strychnia and sodii-citras. In fact no medicine is necessary except small doses of iron and strychnia.

The patient once or twice asked me if I was inclined to give any special injection and pressed me about the same. I thought that she will thank me to be a doctor not up-to-date and therefore I have given her after dropsy disappeared 10 c.c. of distilled sterile water intravenously with a double object. It created confidence in the patient that I am an up-to-date doctor and it gave me two more chances to practise intraven.

ous injection. The patient after these injections, as she says, 'found rapid relief, perhaps mentally.' What does the patient think if she comes to know of the specific of these injections. She thinks me to be a big fraud. Any how belief is relief.

* THE INFANTILE LIVER

By

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The infantile liver, or infantile enlargement of the liver, or infantile billiary cirrhosis, as it is differently called, is not a disease of new origin, though it has recently attracted greater notice, and received better attention under its new designations. When I read the articles by Dr. Mallick and Dr. Sarma in 1922 April and August issues of the "Antiseptic", some previous cases which exactly corresponded to their descriptions occurred to my mind, and I have since been ever on the alert to look for and record such cases. Having now actually seen some of them and compared them with those of past recollections I feel inclined to believe that this disease was in existence as long ago as any other disease, the only difference being that it was till this loosely named 'cirrhosis of the liver' and that the word 'infantile' was newly added to it. The disease claims its importance from its grave prognosis. Any account of it, therefore, is made only to sound a note of warning to the general practitioners and the intelligent public who are likely to come across more such cases than are now supposed, and whose knowledge of the disease, especially of its causative factors, and early detection might, at all events, serve to help its prevention, and sometimes to avert its fatal consequence. It is not necessary here to go into the etiology of the disease which has been elaborately dealt with by the aforesaid writers, nor is there anything new for me to suggest in the matter of dietetic or therapeutic treatment. Broadly speaking, it may be fairly accurately laid down that the use of excessively starchy food is the main cause of the disease. Overfeeding either by way of quantity, or by the number of feeds, or by the variety of foods which are highly starchy in character is a general habit of most of the Indians, and especially of some particular classes of people and that is the prime cause of the disease as evidenced by the previous dietetic history of almost all such recorded cases. The other factors appear to be only contributory. Since no such cases are found reported from these parts of Malabar where they are not so uncommon, I think that a short account of

*Specially contributed to the 'Antiseptic.'

one of the three cases which I had the opportunity to see recently, will be of some interest.

The patient was a male child, aged 1 year and six months, Hindu (Brahmin), the fourth child to the mother, all the three above, living and in health. Mother lean and weak. Having no sufficient milk in the mother's breast, she supplemented it by cow's milk for nursing.

History:—The parents noticed an enlargement in the abdomen at the epigastric region a few months ago. A month and a half prior to the date of my visit, jaundice set in, which, they said, gradually increased. Took to native treatment of various kinds and lastly manthravadam (necromancy) was resorted to. Nevertheless the jaundice persisted and increased in intensity, and slight anasarca appeared on the abdomen. Urine, of highly yellowish colour. Bowels, highly costive, but moving under native Kashayams (decoctions). Four days previous to consultation, fever also set in, which generally manifested itself well towards the evening, and became more pronounced at night.

I was called in to see the child on 24—9—1923 at 11-30 A.M. and noted the following on examination. Appearance, lethargic and dull; Skin dry; A sluggish ulcer (abscess burst and contents drained some days ago) was found on the right side of the body in the mid-axillary line and a little below the ribs. A scar of a similar abscess (healed) found on the opposite side. Temperature 99. F. Liver, greatly enlarged—as far as the umbilicus—hard, and resistant, and slightly tender too. The left lobe of it was prominent. Spleen also enlarged to some extent, but could not be correctly gauged down on account of the enlargement of the liver. Fluid in the abdomen, felt. Pulse, feeble and quick. Nothing noteworthy in the lungs. Conjunctiva, highly icteric. Urine, highly billious which was confirmed by examination, and the clothes soiled by it retained the stain even after washing.

Diagnosis:—Infantile cirrhosis of the liver was diagnosed, and the parents were appraised of its grave prognosis and the already helpless stage at which recovery was out of the question.

Treatment:—Something by way of treatment was advised for the solace of the parents. A mixture containing Rhubarb, soda bicarb, ammon: chloride, chionia, Taraxicum, etc. as medicine; and milk and egg albumen, Scott's emulsion, etc., as diet, prescribed. In the same evening, the case was shown to Dr. A. Krishna Menon, M.B., B.S., House Physician, General Hospital, Madras, who had been here then on a short stay. He also unhesitatingly diagnosed it to be infantile billiary cirrhosis, and expressed the same prognosis. Taraxicum was got down from Ernakulam the

second day, chionia promised to be got from Cochin the day after, and two courses of mixture taken from the Dispensary; but nothing further was heard of afterwards. One day I accidentally met the father of the child on the street; and he then said to me that the child had died on the fourth day of our visit. In this case the main food of the child in its infancy was reported to be boiled ragi three times a day.

Another case which came to my notice a week since, was also one of infantile liver, but not so advanced as the above. Here the father of the child (also Brahmin) after consultation and advice never turned up for treatment, either because he turned to native Vaidyans for cheaper treatment, or because he gave it up as a wild-goose-chase to waste his money on patent drugs and diets with no guarantee of cure.

The third case also was in a Brahmin house where the main food of the child was sago which was pushed on from the 28th day after birth. This also ended fatally under native treatment and black magic. There is a popular belief among the people here that there is no proper treatment in western science for Kamala (jaundice) and that a mere wave of the magician's wand will quickly blow it off.

*PYORRHOEA ALVEOLARIS

By

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Pyorrhoea Alveolaris or alveolo-dental pyorrhoea or even, as lately called, alveolo-dental traumarthrititis because of its traumatic origin is a local infection of teeth characterised in chronic state by a purulent discharge, together with the destruction of the alveolar tissues and consequent loosening of the teeth.

Pyorrhoea alveolaris is a disease of the roots and surrounding tissues of the teeth, but however the term is only a description of the advanced stage of the disease, the earlier stages being either over-looked or not followed. It is at the neck of the teeth (between the root and the body) that the gum margin, in health, blends with the periosteum covering the roots of the teeth and by firmly encircling it and by a "phagocytic" action, protects the periosteum from infection.

In "Pyorrhoea Alveolaris" these actions of the gum margin are by degrees taken off, a pocket is formed between the roots of the teeth and

*Specially contributed to "The Antiseptic."

the gum margin allowing free access of tartar, food debris and also bacteria which infect and destroy the tissues round the roots of the teeth and later the alveolar processes of the jaw bone. Thus a large area of ulceration and in many cases a very large area of tooth and bone tissue open to infection result. The septic matter from this "stagnation area" is not only swallowed with saliva or food, but also passes direct into the circulation through the tooth and bony tissues.

Causation :—The disease has been considered to be due to a microbe—streptococcus, a staphylococcus, a lactose-fermenting bacillus, a diplococcus or quite recently to a spirochæte. Some believe that the disease is local due to irritation by tartar or calculus, bacteria or food debris, and some trace it to mouth breathing, the presence of bacteria being explained as only casual or passive. That the disease is merely a manifestation of a constitutional breakdown has of late gained considerable support by the "vitamine" theory of causation of diseases.

Signs and symptoms :— "A thin red line about a millimetre wide running parallel to, and about its own width distant from the gum margin, is a sure sign of commencing Pyorrhoea." Sometimes, however, the gum is thick and spongy—especially at the processes between the neck of the teeth—and easily bleeds in brushing teeth. Later the gums recede, tartar collects under the gum margin, gums become much inflamed and turn darkish, due to the engorgement of veins. On pressing the gum, pus oozes out in the last stage. Finally the alveolus is involved, becomes first inflamed and then absorbed, thereby loosening the teeth.

Like syphilis, the local symptoms of this dire disease are too simple to require any urgent medical relief, much less dental. Tenderness of the gums on one side of the jaw shifts the mastication process to the other side, the bad taste in the mouth and the bad smell noticed by others are generally unheeded. But when the pus upsets the general system, a loss of energy, anaemia, a feeling of irritability and inability to get up cheerfully in the morning are noticed.

Consequences :— On swallowing the pus and toxins with food or saliva, the stomach is upset, resulting in loss of appetite, vomiting, flatulence heart-burn and pain in the stomach. A septic gastritis will be set up, organisms invade the wall of the stomach or bowels, and a gastric or duodenal ulcer may result. From the intestine, gall bladder or appendix may be infected and inflamed, or a diarrhoea set up.

The pus and toxins may directly infect and produce tonsillitis, pharyngitis or adenoids and through the media of the Eustachian tube middle ear disease or neuritis of the auditory nerve (tinnitus). The eyes

and nose stand to be invaded directly though the naso pharynx or indirectly through blood stream and often affections as iritis, optic neuritis, etc., are traceable to pyorrhoea. From pharyngeal affection, laryngitis follows or even Bronchitis. Pneumonia, Pleurisy, Asthma, or Consumption may be lighted up.

With an intense poison circulating in the blood, the frail organs of the body cannot but yield to it sooner or later. The heart muscle, the arterial wall and the kidneys are too often damaged, resulting in anaemia, as well as depressed or suppressed excretory function of kidneys, and thereby increased poisoning of the system. Many authorities hold that diabetes may be caused thus from oral sepsis.

Rheumatoid arthritis is universally attributed to pyorrhoea either due to direct poisoning or its effect on intestinal stasis or insufficient nourishment to the nerve cells by anaemia.

Deaths from pyorrhoea directly are uncommon; no doubt, many die of the consequences above referred to, of 'septic absorption.'

Treatment :—In early stages of inflammation, the disease is amenable to simple remedies. All that is necessary is to maintain hygiene of the mouth, to drain and clean the "Stagnation areas" from tartar, food debris, etc., and to correct any defect in constitution or to supplement any deficiency in food. In case of mouth-breathing, adenoids or tonsillitis have to be eradicated.

The treatment of Pyorrhoea Alveolaris during purulent stage so far followed, is most discouraging and hopeless to the safety of teeth. This has been due to the neglect in disregarding the early signs of the disease and also in restricting one's attention to local conditions alone. Thanks to the researches during the great European war, the early recognition of the disease and safe and sure requisition of Nature's own resisting power to combat the disease even in its purulent stage have been the latest medical practice or fashion of the day.

The doubtful etiological factor will not come in the way to recognise that the purulent state of the jaw and mouth bring on a state of acute poisoning in the body. One of the elements essential in the fight against infection or toxins circulating in blood has been found to be calcium. Calcium occurs in blood in two forms, "ionic" and combined. The "ionic" calcium in blood is much reduced in chronic and acute infections and in case of Pyorrhoea Alveolaris is being lost and deposited round the teeth as tartar. Hence the importance of diet containing absorbable calcium. In considering the nutritious factor of the diet, the benefit of vitamins cannot be lost sight of. Pyorrhoea is a disease of "civilized condition of

life," as birds and monkeys kept in captivity are prone to catch it. Diets consisting of fresh vegetables, fruits, raw milk, eggs and greens constitute most to prevent or cure the disease.

If under-feeding in diseased condition is bad, over-feeding is suicidal. For one must not forget that the digestive, assimilative, circulatory and excretory organs are working uphill to get rid of the toxin. Fasting, complete or partial, will thus be a useful factor in not only giving rest to all the above named organs but also to burn up the toxins by the living cells.

In *glandular therapy* there is a ray of hope that the so-called hopeless diseases will yield to it one day or other. The internal secretions of "endocrine glands" act as an adjunct in excretion of the toxin and production of immune bodies, thereby correcting the constitutional defects.

After however warning against the grave dangers of purulent stage of pyorrhoea alveolaris in allowing it to continue its ravages, and after removing the hopeless teeth and local irritating factors as tartar, etc., the patient must be given a good opportunity to fight out the disease by fasting treatment, glandular therapy and sufficient "vitamine" containing diets not to mention other hygienic ways of living, eating and cleansing.

A good saline purge to start with and the application of Tr. of Iodine and salt water to the gum are adjuncts to relieve the swelling and tension of aching gum and to reduce the pus from tooth sockets. The hygiene of mouth is easily kept by cleansing teeth with a brush and non-adhering tooth powder—or a soft and well-formed tooth kuchi. Mouth can and will be advantageously gargled with Thymol or eucalyptus water or even with fresh cold or hot water at least after taking food, eatables or meals and also last thing at night. Smoking or chewing tobacco, drinking coffee, tea and such slimy or milky drinks and taking too much and too often sweets are to be discouraged. Butter milk, especially got from cow or buffalo fed on chaff of cereals, greens and vegetables is highly recommended as a preventive and curative for tooth troubles and also as an "Elixir of Life."

CASES AND COMMENTS.

A NUMBER OF PRACTICAL SUGGESTIONS OR THERAPEUTIC NOTES

By

RAO BAHADUR THAKUR RAMDHARI SINHA, MEDICAL PRACTITIONER,
&c. &c., MOTIHARI P. O. (BEHAR AND ORISSA.)

1. *Colinsonia* and *hammelis* are specifics for the treatment of piles. External use of oil of cade (empyreumatic oil of Juniper Tar) has been very effective.

DIFFERENTIAL DIAGNOSIS OF BRAIN AFFECTIONS ACCOMPANIED BY COMA

BY

K. Kochunni Menon, L. M. P., Sub-Assistant Surgeon, Trippunittura Dispensary, Coochin State.

2. In the treatment of pimples on the face (acne) of young girls and boys I give a reliable preparation of Berberis aquifolium—4 times a day very efficacious. Any acidity of the stomach should be corrected with a good alkaline mixture.
3. In Gonorrhœa a mixture of one grain hydrastine muriate; five grains Zinc sulphocarbolate; one ounce rosewater—20 drops of this added to an ounce of warm sterilized water, injected 4 times a day after urination, together with a good preparation of calcium sulphide and Methylene Blue has given me entire satisfaction.
4. Echinacea augustifolia is a stimulant, diaphoretic, diuretic, sialagogue, cathartic and antipyretic. It increases the cardiac functions, diminishes the frequency of pulse, contracts the large arteries and veins causes rapid re-oxygenation of the blood, increases the leucocytes and red corpuscles and is an antiseptic bactericidc and germicide.
5. In the habitual Epistaxis (nosebleed) I have found bromide of potash in five grains doses every 6 hours or thrice daily in half a glassful of water very efficacious. It contracts the arterioles of the nasal mucous membranes and stops the rush of blood to the head.
6. When the tongue is broad, thick, pale, covered with a white pasty coat, it is evidence that there is an excess of acids in the system and therefore there is the necessity of their neutralization by genereal alkaline treatment.
7. Where the tongue is narrow and thin, the mucous membranes dark, instead of pale, dry and sordas appearing on the teeth, acids should be given. Of course the bowels should be made regular.
8. The sulphates of magnesium and sodium do not purge by the stimulation of peristalsis but by increasing the secretion of intestinal fluid and retarding its absorption. These properties make these salts a very valuable intestinal antiseptics, because amæbic and bacillary organisms are flushed out by a sort of back pressure from the blood itself.
9. For constipation with deficient secretion and peristalsis, I give 20 or 30 minims with 2 ounces of tepid water of the tinctures of Physostigma, Nux vomica and Belladonna every morning and evening and with good results.
10. Warts on hand or fingers are removable without pain or discoloration by daily application of essential oil of cinnamon. In the course of a few weeks they disappear.

Signs and symptoms.	Uræmic Coma.	Opium Coma.	Alcoholic Coma.	Hysterical Coma.	Epileptic Coma.	Apoplectic Coma.	Concussion of Brain.	Compression of Brain
Odor.	Urinus and offensive breath.	Of opium.	Alcoholic.					
Pulse.	Rapid and high tensioned.	Slow, forcible, good at first rapid in fatal cases.	1st rapid and strong then feeble.	Normal.	Slightly increased	Slow and full.	Frequent, feeble and fluttering.	Early slow, full, regular blood pressure increased. Late irregular and rapid B. P. falls.
Respiration.	Irregular; First fast, then slow; dyspnœa.	Greatly reduced 2-12 per minute.	"Puffy" decreased but comfortable.	Spasmodic breathing.	Decreased at first; then stertorous.	Stertorous, deep, noisy.	Frequent and sighing.	Early, slow and regular Late. Irregular and rapid Cheyne-Stokes.
Temperature.	At first elevated, then subnormal.	Often sub normal; dry skin.	Sub-normal.	Normal.	Normal or slightly increased.	Normal or subnormal during Coma.	Subnormal, Cold and Clamy.	Slight fever. Flushed Warm and dry.
Pupils.	More often contracted than dilated.	Pin point do not react to light.	Moderately dilated.	Normal.	Dilated and insensitive.	Dilated and unequal. Do not react to light conjugate deviation of eyes.	Contracted.	Dilated as a rule, but may be dilated or contracted or unequal.
Face.	Dusky countenance and œdema.	Cyanotic. Profuse sweating and warm.	First engorged; then pale, cold and clammy.	Pale.	First pale, then cyanotic; Fixed and turned to one side. Froth at mouth and bitten tongue.	Ashen grey or cyanotic.		Livid. Cheeks puff-out at each respiration.
Urine.	Contains albumin.	Very often suppressed.		"			There may be involuntary passage of urine.	
Previous history.	Convulsions precede the onset of coma and sometimes dropsy.			Prodromes: Convulsions.	Cry when falling, convulsions in "grand-mal"	Signs of arterio-sclerosis.		A blow or a fall—a wound or swelling on the head seen.
Coma.		Gradual not deep, can be roused to answer questions, profound in bad cases.	Deep and irrational it not profound.	Of very short duration. Restored by cold application.	Short duration.	Often deep; onset usually abrupt. c-Coma.	Not deep. Can be roused by shouting near the ear.	Deep.

Mainly after Irving Berger, M. D.

PERISPLENITIS IN KALA-AZAR

By

ABANI MOHAN DAS, L. M. F. (BENGAL), MEDICAL PRACTITIONER, DACCA.

During the last year I myself observed several cases of Kala-Azar in the western part of the Dacca city. Of these a case took a particular bend which we rarely come by in the K. A. cases. As it turned to be a special sort of a case, I take it upon myself to give out the facts to our medical world.

It was during the first week of November, 1922, that a Hindu lad, aged about 15 years, was brought to me for his treatment of fever with enlarged spleen and liver.

History and present condition:—In the month of July, 1922, he had a sudden onset of fever with diarrhoea which subsided in 2 weeks, but again the fever relapsed and he suffered much from epistaxis. It happened that, after a period of Quinine treatment, the patient ceased to respond to it. Since then he was practically getting attacks once or twice every month *i. e.*, pyrexia followed by apyrexia.

But when it was referred to me, I found that he was suffering from continuous fever for the last 15 days, intermittent, with double rise in 24 hrs. and not on alternating days.

The spleen extended 2 finger's breadth downward below the navel and 3 fingers' breadth to the right of the midline. The liver was plainly palpable. Besides I observed marked emaciation, pale conjunctiva, poor musculature, thin dull lustreless hair falling off, oedema of the lower extremities and diastolic murmurs in the pulmonary region.

Clinically, it was diagnosed to be Kala Azar and testing the serum of his blood with Liqr. formaldehyde found it to be positive. The differential counts of leucocytes were as follows:—

Number of Erythrocytes per c.m.m. 2000000.

Number of Leucocytes per c.m.m. 2600.

Polymorpho nuclears 41%

Large mono nuclears 3%

Small mono nuclears 56%

Eosinophiles, mast cells. nil.

Treatment:—I commenced the intravenous injections of 1% sodium antim. tart solutions and prescribed

sol. adrenalin chloride m vii

aqua ad 3i

to be taken
twice per diem. After 14 injections (45 c.c.), all of a sudden he had a severe attack of diarrhoea wherein he passed watery motions several times in the

first day (of his attack) and suffered a good deal from Perisplenitis, i.e., severe unbearable stabbing, pain in the splenic region with splenic murmurs quite audible (by stetho), so much so that it became excessively tender to touch. He was exhausted, with hurried respiration and I injected morphine sulph tab gr. $\frac{1}{4}$ and atropine sulph gr. $\frac{1}{180}$ (B. W. & Co.) for two consecutive days. And along with it Emetine Hydrochlor ampule (gr. i) every alternate day And he was relieved after 3 days.

I then withheld the antimony injections for 20 days. And as his spleen was much fibrous, I put in 2 injections of 5 minims each of T.C.C.O. solution in his gluteal regions once a week.

But during the antimony treatment I could not increase the dose regularly as he had always a tendency to dysentery and lung trouble.

It took me 50 injections to complete 200 c.c.'s of Dr. Knowles' dictum. The last injection was administered on 27th June, 1923. For these last 3 months no relapses have occurred. The patient is now comfortable and has regained his former health.

THE TYPICAL SYMPTOMS OF PYORRHOEA

By

AMAR NATH YAJNIKA, M. C. P. & S.

In these days, pyorrhoea is so common a disease that one likely gets cases of this type in course of the general practice. It is still rare among the old and orthodox persons who spare a considerable portion of their early morning hours in brushing their teeth with fresh green-neem sticks. They enjoy the full blessings of teeth even in good old age, whereas it is observed with surprise among the present day civilised young generation that 75% of them have manifestations of the above disease. Either they resort to treatment for the primary infection or for the secondary symptoms for which they are quite ignorant as for the causative factor. The result is disappointing and they have to sacrifice natural property for the artificial dentures for more cosmetic purpose.

I do not propose to discuss the main disease as to its etiology but I shall now consider some of the typical symptoms for the interest of the readers of this Journal.

The disease begins as a marginal gingivitis. It also begins early in life since the conditions, favouring periodontal stagnation are present as soon as the tooth is erupted, and hence is often found at least as marginal

gingivitis among the temporary teeth and before long in more advanced stage among the recently erupted permanent teeth.

It is, in fact, just as in dental caries a disease of childhood and early adult life. It is quite common, when enquiring into the history of adult pyorrhoea cases, to find that in an early life there were intermittent attacks of swollen and tender gums accompanied by tenderness and loosening of the teeth. Those attacks I regard as characteristic of the earlier stages of pyorrhoea. Again, just as with caries, on account of bilateral symmetry, it is symmetrical in its attack. It may attack first and most vitally the earlier teeth, e.g., incisors and first molars of the permanent set. It is common among the front teeth. The pyorrhoea represents the results of germ stagnation-not germ-sticky food stagnation of a life-time-and shows itself among the larger and less cleansable teeth.

Pain, including tenderness is generally not a permanent symptom till such time as abscess formation sets in; sometimes pain is of neuralgic character.

Bleeding from the gum is a common symptom. The amount of the blood lost is usually small. On superficial examination of the mouth no bleeding point is to be seen. Pocketing is a common occurrence in the course of pyorrhoea. It is brought about by the destruction of peri-odontal membrane and bone in advance of gum destruction and is an expression of progressive tissue infection and insufficient drainage.

Fætid odour of breath is a common symptom of dental sepsis. Nose, tonsils, lungs, tongue or stomach *via* the blood stream account for some cases but in cases of tonsils and tongue, dental sepsis may have been the primary cause. Generally the smell is one of putrid decomposition. The patient is unaware of the factor realising it neither by the sense of smell or taste; he may experience foul taste especially, on waking in the morning at unexpected moments. The odour is due to putrid decomposition.

An obvious discharge of pus from the gums is a striking symptom. As a reaction to infection marginal hypertrophy of the gums follows and next atrophy proceeds. Excessive salivation is sometimes a most distressing symptom. Recession of the gums, inflammatory swelling and true hypertrophy of the gums, loosening, elongation and separation of the teeth are common symptoms. Abscess formation is a common event in the course of pyorrhoea. The peri-odontal membrane gets enlarged many times its original thickness and the surrounding bone gets destroyed to make room for the increasing inflamed granulation tissue. It is responsible for much of the loosening of teeth.

In some cases gum is of a deep blue colour of chronic congestion. These are cases of long standing.

Sometimes the tooth assumes blackish colour. This blackness may be of bacterial or haemic origin.

RATIONALE OF AUTO-HÆMIC THERAPY

By

KSHETTRA MOHAN GUPTA, M.B., M.R.A.S., CHIEF MEDICAL OFFICER,
MOBISHADAL RAJ HOSPITAL.

In my short article on "Auto-Hæmic treatment" which appeared in the "Antiseptic" May of 1922, I tried to give the detail of my technique, which I generally follow in treating my cases. In response to that article, I received some correspondence and literature from Dr. Chas. B. Graf, M.D., the then Vice President of the Allied Medical Association of America and the Chairman of its Auto-Hæmic Department. There I found the detailed exposition of the rationale of the said therapy. I shall try in this short article to represent the rationale in a concise form.

Some physicians refer to it as a serum treatment. This misnomer is not only incorrect but misleading. With serum treatment, an animal is inoculated with the virus of some disease. When the subject has become ill with the disease, a portion of its blood is drawn off, the serum extracted therefrom and injected subcutaneously into the body of the patient, diagnosed to be suffering from the same disease. The diagnosis may have been correct; the serum may have been prepared from the blood of an animal having a similar disease; but the conditions cannot possibly be identical; therefore, you cannot get proper results.

Auto-therapy should not be confused with Auto-Hæmic Therapy. Auto-therapy is the method of treating the patient with unmodified toxic substance, elaborated within the latter's body, by the action of the infectious agent, on his body tissue; against which the tissues react in a curative manner. The unmodified toxic substances are obtained by filtering the pathogenic exudate or other body fluids containing the toxic substances produced by the action of the infecting agent. The bacterial free filtrate is injected subcutaneously in all acute localized infectious diseases.

Auto means self or same. Hæmic is understood to signify the blood. Therapy is that part of medicine which represents the discovery and application of remedies for disease. Here the preparation is made up from

the whole blood of the individual patient. As above defined, it is a remedy for disease, prepared from the blood of the patient's own self. It is an example of "*Similia Similibus curantur*."

In other words, the patient's own blood is converted into a remedial agent without the admixture of any drugs or bacteria or anything foreign to the constituency of the patient's own blood itself. Every blood corpuscle contains ferments. These ferments are inactive as long as the blood corpuscle is intact. The moment the blood is put into the distilled water, the intra-cellular pressure of the corpuscle being much greater than the extra-cellular pressure of the distilled water, it liberates the ferments which at once begin to digest the protein of the corpuscles, resulting finally in the decomposition of the protein, until it becomes "Amino Acids", and these acids act as stimulants, reduce the imperfect cell products in the circulation that are the real instigators of diseases. All must recognise that faulty metabolism is responsible for all our common disorders.

The blood contains in it the healing force of Nature. It carries nourishment to every cell of the body to select from for its own use, and in return carries waste to be excreted by the way of the lungs, kidneys and skin. It contains the means of defence against the invasion of micro-organisms and toxins and when injury is done, it carries the means of repair whenever they are needed. Therefore, to purify the blood must be the basis of all treatment.

The Rationale of this method of treatment depends upon the following suppositions:—

First supposition.—Hydrogen ion concentration and chemistry of colloids, probably have to do in par with Auto-Hæmic Therapy. The old conception of chemistry was suspension and solution and now to that we have a third or colloidal suspension. Particulate matter, down to one micron in size, in a fluid, may be regarded, as being in suspension. Particles of a size, ranging from 1 micron to 1 m.m. or micro-micron or a millionth of a millimetre, are colloids. They are affected by electric charges. Like charges will precipitate colloids from suspension. Also one colloid, in proper concentration, can be used to precipitate another colloid. They are easily affected by salts and electrolytes.

The blood is a colloid. It is not the usual handler of protein. When it does so act, however, as for example in disease, there results a paren-teral (outside the digestive tract) digestion, in which some serum is digested, some body cells and some foreign protein.

The character of symptom resulting, depends upon relativity. If foreign protein be small in amount, as a few bacterial cells, tryptic activity or paren-

teral digestion results in small amount of serum split products and body cell split products and split products from foreign protein. These split products act anti-tryptically and tend to slow down tryptic activity. Because the toxic split products from all three, serum, body cell and foreign protein, are small in bulk, symptoms are such as we expect in disease.

Second supposition.—If foreign protein be large in amount or great enough, tryptic activity or parenteral digestion is of much larger order or measure, resulting in enormous production of toxic split products from the same three sources, serum, body cell and foreign protein, which results in anaphylaxis. Hence the importance of providing continually circulatory Antigens, in Anaphylactic conditions, to provide the necessary amounts of toxic split products, which will then act anti-tryptically, slow down tryptic digestion (parenteral) and do away with the symptoms. The above reference to Hydrogen ion concentration as well as to the probable mechanism responsible at one, and the same time for anaphylaxis and immunity, gives a hint, as to the extremely complex character of and complicated inter-relationship, to the various colloids, electrolytes and solutes, in the blood stream.

Triple distilled water (which the above Association use, though it is not used in my technique) seems to exercise a peculiar property and propensity in precipitating the above, in the blood stream, when removed therefrom in relatively small amount, diluted to the proper degree and proportion and potentized to a high radio-activity by rapid succession (A rotatory and perpendicular electric automatic shaker is used by the Allied Medical Associations of America)—of the mixture of blood drawn from patient's vein and the triple distilled water. They say, a rapid succession of a fluid produces certain amount of radio-activity in the fluid used.

When studied from the preceding view point, it would appear that there are well founded reasons for the success, in practice, of a therapy now attracting considerable attention.

RAT-BITE DISEASE

By

DR. SATI BUSHON MITRA, B.Sc., M.B., CALCUTTA.

Poisonous snakes attack on rats as a food. If, by chance, the rat escapes from being devoured by the said snake, the rat becomes poisoned. If the size of rat is larger than that of the mouth of the snake, then the above condition takes place; otherwise the smaller rats are easily devoured by the snakes. We know that when a snake bites, poison is discharged from the

poison bag closely connected with a special tooth. When taking food, such a poison is never discharged. But the toxicity that is found in the body of the rat is produced by the saliva of the snake. The condition simulates hydrophobia. At this rats become mad and so they bite any body when found on their way. Else, hundreds of rats that walk about at night fearlessly, biting every person on their passage of walking, might depopulate the country within a very short time. Those escaped mad rats are commonly known as "Dadur". Recently I had a chance to treat a rat-bite patient, whose signs and symptoms are mentioned below.

On the 7th June, 1923, an elderly Muhammadan woman came to me for better treatment. She told me that she was bitten by "Dadur" about two months ago. At this, signs and symptoms of toxicity became evident on her body. She told me further that she used various drugs in her native village but to no effect. So, she came to me for treatment.

Signs and symptoms:—Morning temp. was 100 °F. Every evening she got fever with rigor. Her fever remitted with profuse perspiration that night. She got pain all over the body and excruciating pain even in the bones. She had no appetite for food at all. Since rat bite, her menstruation commenced and still continues for the last one and a half months. Discharge was profuse every day. Before this event, she had never any disorder of menstruation. In the interval, she suffered from dropsy but now she is devoid of it. There was no thirst. Constipation was present. On examination, I found a scar mark at the site of bite by rat. There was no evidence of ulcer within on pressing the part carefully. Rat had bitten her on the upper part of left arm. I came to learn that such disorders took place gradually since her bite by rat. Hence there was no doubt that she was ill of rat bite disease. So, there being no specific treatment for the disease, I had recourse to symptomatic treatment.

I prescribed the following mixture:—

Re Sodi salicylas gr. iii
Spt am aromat—m x
Spt chloroform—m x
Tinc Hyoscyanas—m x
Tinc Rostox—m v
Tinc Nux vom—m iii
Aqua.—ad 3i

Sig. One to be taken every 4 hours.

This mixture was continued till 9th June.

On the 10th June I visited the patient. This time the patient had no fever. She had no fever since the last two days. There was one motion

only. Patient did not perspire. But she had become very weak owing to sufficient blood discharge from the uterus.

Sago with milk was given as diet. As the patient was not satisfied with this diet I advised her to take one seer of milk daily. The following mixture was prescribed :—

Re Calcium Chloride—gr x
Tinc Pulsatilla—m v
Tinc Hydrastis m v
Ext Ergot liq—3p
Aqua——ad 3i

One to be taken 4 times a day. This mixture was continued for 6 days. This time, patient's blood discharge from uterus was lessened considerably. As the patient was very eager to have an alteration of diet, I allowed her to take rice on the 14th June.

The following Tonic mixture was prescribed.

Re Quinine sulph. gr. ii
Acid Sulph dil m x
Tinc Ferri Perchlbr—m x
Tinc Hydrastis—m v
Sodi Sulphatis—3 i
Aqua Anethi, ad 3 i

Sig. One to be taken twice daily after food.

This mixture was continued for a week. At this time, the patient did not complain of blood discharge from uterus and the patient got strength. I applied symptomatic treatment in this case but I can't tell for certain if there was any rat poison within the body of the patient. But this was certain that the patient's bodily health was excellent before the present incidence. I am not telling you that the medicines I prescribed heretofore are the only medicines for the rat-bite disease in human beings. Truth to say, that all rats do not bite on human beings. But those rats who come in contact with poisonous snakes are responsible for the disease. The room wherein the patient used to live at night contained two cobra snakes within its floor, from which they were dug out.

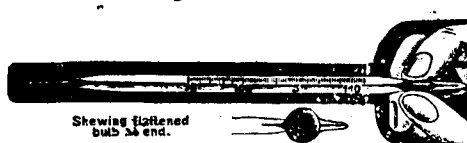
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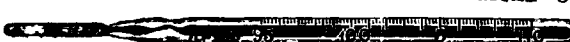
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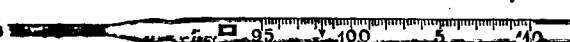
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 CASES.**

By

U. VENKATA RAO, L. M. P., MEDICAL PRACTITIONER, "SUDARSANA", MADRAS.

(Continued from page 337.)

CASE No. 7.—Mr. Balasubramanya Iyer, aged 28 years, had an attack of fever with shivering and swelling of the left leg beneath the knee and of the inguinal glands, about the middle of June. Two days later the fever subsided, but the swelling continued. There was difficulty in walking and raising the foot. He applied Pigment Belladonna and Tinct. Iodine and the effect was poor. On the sixth day he consulted me. The left leg was double the size of the right leg. The swelling extended from about 3 inches below the knee and involved the ankle. On the whole it looked like elephantiasis. The skin was thickened, no pain, but pitted deeply on pressure and the veins were marked beyond the swelling. He had no previous attacks of a similar nature. I applied electric heat pad firmly over the swelling for about 20 minutes every day. The heat was uniform and the pad on removal was found completely wet with perspiration. The patient began to feel better from the second day onwards and in a week the swelling was reduced by half. I then began to apply the electric pad every alternate day. In about four weeks the swelling almost disappeared. Twice a week the pad was applied for another two weeks, by which time no trace of the swelling was left.

CASE No 8.—On the 12th July I was asked to examine Mr. Kuppu Chettiar, aged 35 years. He had an abscess in the left axilla for the past one week and was attended to by a barber. The patient had 103° F. temperature and was restless. The whole of the left shoulder was swollen and tender and the abscess was an inch deep; chain-like eruptions were seen on the shoulder extending from the deltoid and scapular regions to the nape of the neck in two ranges. The eruptions looked like herpes. There was much pain and swelling and the patient was unable to lie down on that side. His urine had sugar. I dressed the wound antiseptically and applied hot Antiphlogestine over the shoulder. The whole of next day the temperature was the same 103° F. and the eruptions and swelling continued. On the third day I gave him an injection of anti-streptococcus serum P. D. & Co. 10 c.c., applied blue 16 C.P. carbon light for 15 minutes and antiphlogestine. The temperature came to 101° F. in the evening and the next morning it was normal; the discharge from the abscess diminished considerably and the eruptions began to fade. The same treatment continued for five days. On the fifth day the eruptions began to dry up and on the 8th day the

superficial desquamated skin began to peel off, the swelling and pain subsided and the patient was able to lie on that side. Stopped antiphlogistine, but continued the light treatment for another week by which time the patient was completely alright.

CASE NO. 9.—Mr. Shanmugam Chetty, aged 28 years, had Eczema on both the legs for about three years. The Eczema extended from three inches below the knee to an inch above the ankle joint of the right foot and had involved even the ankle joint over the left foot. I began to apply electric blue light carbon 16 c.p. for 15 min. daily after cleaning the parts with boiled cocoanut oil mixed with neem oil, dusted with powder containing equal parts of zinc oxide, starch, Boric acid and Hydrargyri ammoniate and lightly bandaged. There was much serious discharge which gradually diminished in ten days. After two weeks I began to dress the parts with simple Boric ointment. I persistently continued the electric light and heat treatment for nearly six weeks. During this time the patient was asked not to wash the legs with water. The eczema completely disappeared and had not recurred till now, that is six months.

CASE NO. 10.—Mr. Anthony aged 12 years from Cuddalore, had Eczema on both the feet for the last six months and plenty of eruption with thin watery discharge and the feet swollen. Continued the above plan of treatment for four weeks and the eczema completely disappeared. It is now nearly four months and there has been no recurrence.

(To be continued.)

SUCCESSFUL TREATMENT OF PLAGUE.

By

S. R. KIRLOSKAR, M.B., B.S., FAMILY MEDICAL HALL,
DHARWAR.

The type of plague that is playing havoc at Dharwar is as everywhere of a very severe nature indeed. For the attack is always very sudden and the patient succumbs within a couple of days. Every official and non official drug from materia medica was tried without effect. After prolonged thinking and formulating theories in my own way I began to use the following method and I am glad to say that since the adoption of this new line of treatment I have rarely lost a plague case. For every plague case I used to raise a rupee-sized blister on or over the bubo. I draw the contents of the blister about 1 to 2 c.c. in a clean watch glass or a hypodermic syringe and inject the same sub-cutaneously. There was

no secretion worth mentioning at the site of injection. The temperature used to come down by a degree or two in every case after 12 hours. Only some cardiac stimulants were given internally. By this line of treatment I have been able to save almost all my plague cases that came to me before brain was affected. I earnestly request my fellow practitioners all over India to give a fair trial to this line of Auto-inoculation treatment and take the credit of saving their brothers and sisters from falling victim to this dire disease.

CHYLUR A

By

MANAYATH ANANDAN, S. A. S. ON LEAVE, PAYYOLI.

Kalyani, a female, aged 15 years, and married two years ago was passing-milk coloured thick urine for about eight months. Complained of pain in the loins ever since the disease appeared. She was under native treatment for 3 months with no effect whatever. It is said that she went to the Dispensary at Quilandy for about two weeks and as she was not cured her husband thought of divorcing her to marry another woman thinking that the disease was an incurable one. She was brought to me by her father crying, as her condition was considered to be very pitiable owing to the disease on one side and the threatened divorce on the other. I gave him a promise to cure the disease and gave her first worm powder (Santonin) in the night followed by castor oil in the morning. She passed six large round worms and the urine was yellowish white. The same evening she was given a mixture containing Ferri Sulphur, Pot acetat and soda salicylas 2 grains each in an ounce of water and on examination the urine was found to have changed in colour slightly. The next day she was given 10 grains of gallic acid once and the same mixture three times in the day. On the third day the urine was almost clear. The same mixture was continued for 3 days more and she was completely cured. She did not suffer from it ever since. I saw her with her husband 4 months after, very happy, with no fear of divorce.

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FEBRUARY, 1924.

THE LEE COMMISSION AND THE MEDICAL SERVICES.

The Lee Commission had come and gone and Madras had a very lively session of it. That huge, richly caparisoned "White Elephant"—the creature of Lord Peel and his die-hard colleagues in the Cabinet—ushered into being rather prematurely, and that in an evil hour of political unrest and ferment, blessed and baptized by Royalty, and thrust for maintenance and support on an unwilling Indian populace at a time of financial stress and stringency, has, as already anticipated, done more harm than good to the land of its adoption. Besides imposing, or rather to be more accurate, endeavouring to impose further financial responsibilities on the Indian Government in the shape of increased emoluments to the services, the Commission has served as the safest medium through which the European element in the Services were emboldened not merely to ventilate their own grievances but also to give vent openly to the feelings of jealousy, hatred and contempt which they had so long engendered against their Indian fellow-officials and to pour forth, at the same time, their vials of wrath against the Indian politicians generally, thus widening, all the more, the gulf between the rulers and the ruled. The Commission has further helped to

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disillusion the minds of the Indian public of the accommodative nature of the Services to the Reforms and their pliability to the control of the Indian Ministers so loudly proclaimed from house-tops at their first inception. The climax was reached in Madras where the European members of the Indian Medical Service refused to consult the Indian members of their profession before drafting the answers to the questionnaire and practically ignored their very existence. This treatment had naturally aroused strong resentment at the hands of the Indian I.M.S. Officers and provoked a scene at the time of the examination of the witnesses, which was hardly edifying and dignified. Even Lord Lee, the Chairman of the Commission, was unable to shake himself of and rise above all racial prejudices and passions, and while he characterized the separate memorandum of the Indian I. M. S. Officers as highly provocative and strongly-worded, he had not had the fairness to raise even his little finger by way of protest against the disrespectful and discourteous treatment accorded by the European Officers to their Indian brethren. This is really deplorable. We shall leave this unpleasant episode here and turn to the work of the Commission in Madras, in so far as it related to the Medical Services.

THE EUROPEAN I.M.S. OFFICERS' DEMANDS.

(i.) *Indianisation of Services.*—The European I. M. S. Officers are getting anxious about the rapid Indianisation of the Services and are of opinion that, if Indianisation of the Medical Service is taken to mean provincialisation, recruiting should stop instantly and that, in any case, such a policy should not be carried out until those officers who are already recruited in the Service and who wish to join the Civil Department have been absorbed. In their memorandum, they say : "*The main cause of the present discontent amongst members of our Service is the feeling of insecurity and uncertainty of the future, which has been created by hostile*

Indian opinion openly expressed in the press and on the platform towards a Service which has done so much in the past for the benefit of the country. The feeling that good work as such is not wanted and that rapid Indianisation rather than efficiency is the sole aim, has created an atmosphere of deep concern amongst the officers of the Service and is destroying the spirit and morale of the Service as a whole." This is to over-state their own case if not to exaggerate to an alarming extent the situation created by the rapid Indianisation of the Services - a policy to which the Ministry is bound to pledge itself. While we acknowledge, with gratitude, the good work done by the European I.M.S. men in the past and while we still welcome them in our midst under certain necessarily restricted conditions of pay and service, we cannot but express our strong resentment and enter our emphatic protest against the statement made by them that efficiency is sacrificed at the altar of patronage. On the other hand, we are convinced that there are more efficient medical men among the Indians than are to be found now among the European element; and the sole aim of the Indianisation of Services is to encourage Indian talent and to replace the costly European Services by a cheap and none-the-less efficient Indian machinery. Another amazing statement, the European I.M.S. Officers make in their memorandum runs thus:—"The Service appreciates the difficulties of a Minister who is the servant of his party but would urge that appointments in a Scientific Department must primarily depend upon professional and academic merit and not upon political patronage. Further evidence of the unhealthy influence of politics is the present tendency of subordinates to seek advantage by private and direct communication with the Minister on official subjects without the knowledge of their own immediate superiors. The knowledge that such a state of affairs can exist is extremely distasteful to the European official for it destroys all confidence in the loyalty of subordinates and by so doing renders impossible that co-operation which is essential to efficiency." We do admit that such a state of affairs is highly undesirable

and is likely to lead to constant disharmony and friction between the head of the department and his staff, but those that made that observation seem to have forgotten the fact that the same game was played by them perhaps in the inverse order, when the bureaucratic regime was in the height of its glory and the head of the department had his will enforced against his poor subordinates by that very tactics of direct communication with the powers-that-be nullifying all efforts by the subordinates to obtain a fair and impartial hearing. That was surely autocracy pure and simple and if during the period of transition, such unpleasant over-ridings of official etiquette and decorum should happen with a view to get their long-standing grievances redressed and secure bare justice against autocratic methods which still continue, they must patiently be borne. We have the recent incident of a senior Indian I.M.S. Officer in Madras, with better credentials and qualifications being superseded by a comparatively junior European, even, it is said, over-ruling the choice of the Minister in the matter. We mean the appointment to the Principalship of the Madras Medical College and if it is not a breach of official etiquette, we ask what else is it and how far is it pardonable, though perpetrated by the highest power in the Province? Even according to Col. Bradfield's own admission before the Commission that in the matter of appointments preference should be given to men of real merit, Major Pandalai possessed by far superior qualifications to himself for the post in question. What is sauce for the gander must be sauce for the goose as well is a saying which aptly fits in in such cases.

(II) *Private Practice*.—It is true that private practice, so far as the European I. M. S. Officers are concerned, is not now quite so alluring or promising as it once was. The spirit of reservedness, the growing consciousness of their

own racial superiority, the distance of their abodes to Indian habitations, the difficulty of their accessibility without ceremony and previous engagement, the high fees demanded by them, the low means of the generality of the patients, and, above all, the availability of equally good and competent men among Indians themselves are the contributory factors that have brought about this state of affairs. It is hard to expect patients, especially Indian patients of high or low degree of status, to seek the aid of medical men who like parallel lines do never meet. Everything would go well if only they care to adjust themselves to the changing times and circumstances. To compensate for all this, however, the European I. M. S. Officers have almost the monopoly of the practice among patients suffering from special diseases, for the successful treatment of which their position affords them all the facilities.

(iii) *Status, Pay, Prospects and Pensions of the I. M. S.*—It is the considered opinion of the European I. M. S. Officers that the time has not yet arrived for the throwing open of all appointments to Indians and that certain posts should be reserved for them including the office of "the Director of Public Health"; that all appointments should be made by H. E. the Governor acting on the advice of his Chief Medical Adviser, the Surgeon-General to Government; that all appointments in the Provincial Service should be left entirely in the hands of the Surgeon-General and that no decision regarding the personnel or policy of the Medical Services should be arrived at without previous consultation with the Surgeon-General. This is, in sum and substance, to ignore the existence of the Minister in whom the power is now vested, and to retain the *status quo* of the bureaucratic days. The European I. M. S. Officers even go a step further. They demand that the Surgeon-General should be the Secretary to Government as well. This is what they say:

"The Director-General I. M. S. and the Provincial Surgeon-General should rank as Secretaries to their respective governments and should have all the privileges of that position. It is difficult, if not impossible, for a lay officer, however brilliant, to grasp adequately the problems of a highly technical subject such as medicine and much correspondence which is essential at present would be avoidable if the head of a Medical Department was a Secretary to Government and had direct access to the Minister. Further, it is only such a status which will ensure that close connection with H. E. the Governor, which we consider essential for the future safety of the Service." This is merely a preposterous claim unworthy of consideration and savours something of the nature of a judge and prosecutor being combined in one. We have already too many appointments of this nature in the subordinate ranks of the services such, for instance, where the executive and the judicial functions are conferred in one and the same person and we still have a relic of the bureaucratic regime in the superior service such as the Chief Engineer being also the Secretary to Government, P. W. D. We wish to give these harmful combinations of appointments a speedy go-by and see them relegated soon to the limbo of oblivion. What will the European I. M. S. officers say if it is suggested that the Minister should be a medical man, instead of the Surgeon-General being the Secretary to Government? Why did they fail to consider this aspect of the question, in their memorandum? Is it because such a proposal would not suit their tastes that they have conveniently brushed it aside? We wait for an answer. The European I. M. S. Officers demand increased pay and allowances on the following grounds:—(1) the increase of Indian prices prior to, during and after the war; (2) the increased cost of living; (3) the failure to give sufficient financial relief during and after the war; and (4) the enhanced cost of passages to England. They further demand a marriage allowance Rs. 150 p. m., in view of the fact that the burden falls more heavily on the married than on the unmarried people. They

consider the claim all the more reasonable on account of the total cessation of private practice. They insist on the Government providing quarters for the I. M. S. Officers in Madras owing to the difficulty in finding suitable accommodation in Madras and in their having to pay exorbitant rents. They further think that 10 % of their pay deducted for rent is too high and suggests 7 % consolidated without any additions for electric and sanitary fittings. Regarding pensions they say: "*The officers of the Service are not satisfied regarding the security of their pensions. Failing a guarantee, commutation of the whole pension should be permissible; commutation of pension should be a right, not a concession.*" To meet the present inadequacy of their pensions, they suggest that the pension earned after 20 years of service be increased to £ 500 in the 21st year of service rising by £50 yearly to £ 1,000 after 30 years of service. As regards leave, they urge that "*the restriction on the computation of half-pay furlough into leave on average pay should be removed and that officers subject to the special leave rules should be allowed to enjoy the whole of the leave entered in their leave account on average pay.*" Knowing as we do the pre-disposition of the Commission, we shall not be surprised if all their demands are conceded and more too.

(iv) *Medical attendance* :—This is a novel claim to ask Government to provide for British medical attendance on European officers and families stationed in the mofussal. "*To some extent, the problem can be met,*" they say "*by grouping the districts of the Presidency and by insisting that there should be a British Medical Officer always stationed to one station in a group. The head-quarters of such grouped districts should be within easy reach of each other by rail and if reasonable facilities were given in the provision of travelling allowances, the services of British Doctors could be made available for officers posted to them.*"

(v) *Specialist treatment* :—With regard to this question, the European I. M. S. officers observe as follows:—"*We consider, however, that British Medical Officers must be retained*

in such a proportion of these teaching appointments as will provide one expert and one under-study in each of the subjects (medicine, surgery, ophthalmology, diseases of women &c.,) mentioned. To meet this demand, the special appointments in Madras should be considered as four groups and two appointments in each group should be held by British Medical appointments." This is not economically a sound suggestion to adopt, as there will be no proper return for the money spent on training birds of passage as these European officers are. We, however, are prepared to give them such reasonable facilities for specialist training, as would be consistent with the interests of the sons of the soil.

THE INDIAN I.M.S. OFFICERS' VIEWS.

We give below extracts from the separate memorandum submitted by the Indian I.M.S. Officers to the Commission which serve also as answers to the European demands. We entirely agree with the views therein expressed by them.

"i. *Indianisation of Services*:—All the appointments in the Medical Department can now be easily filled by Indians, who, in addition to being equally qualified professionally, are much more suited to the needs of the people as they are familiar with the language, customs and habits of the people."

"There is absolutely no justification for the feeling of insecurity and uncertainty as to the future for such Europeans as are prepared to work in India as her servants and who by their ability, honesty and devotion to duty are worth the high emoluments they are paid. The country is not prepared to employ more Europeans than are absolutely necessary when competent Indians are available in large numbers."

"The Service exists for India and not India for the Services."

"Employment of Europeans of ordinary ability when abler Indians are available in the country wastes Indian money, and creates more difficulty for those responsible for good government."

"Absolute equality of treatment should be guaranteed to the Indian members of the Service. The promotion of junior European members of the Service over the heads of senior Indian members admitted to be very competent as has recently been done in Madras in the appointment of the Principal of the Medical College should cease, as this practice is not only unjust and iniquitous but creates discontent among the Indian members and destroys harmony and good feeling, which are so highly essential for friendly co-operation."

"The chief reason for the reduction of European recruitment and the alleged early retirement of European Officers is the fact that Europeans have found that they are no longer professionally superior to Indians (in the service or in private practice).

"We strongly resent the attempts of European members of the Service to keep members of the Provincial Service from rising higher."

"British and Colonial recruitment to the Indian Medical Service should be stopped at once. India's ideal is to entirely Indianise the service."

ii. *Private Practice* :—"The loss of private practice complained of by the European members is only explained by their own inability to compete with the Indian practitioners for which certainly the Secretary of State is not to blame. This does not strengthen their claim for enhanced pay and allowances, but is a strong argument for replacing them by Indians or Europeans who can do the work as well if not better and are cheaper."

iii. *Status, Pay, Prospects, Pensions, etc., of the I.M.S.* :—"The head of the Department of the Transferred Subject must be an Indian so that the Minister may be able to discharge his duty to the country as well as he is expected to do without obstruction from the head of the department based on racial discrimination. In no case that we know of an Indian has been appointed to an office previously held by a European has

efficiency been sacrificed for political expediency. We are of opinion that all appointments should be made by the Minister, in consultation of course with the Surgeon-General."

"We strongly oppose the proposal made by the European members and are of opinion that the Surgeon-General and Director-General should have no direct access to the Heads of Government. Systematic opposition to the elevation of Indians to the offices of Surgeon-General and Director-General of the Indian Medical Service, should in fairness stop now."

"No one who is not familiar with the social habits and customs of people can do justice to the appointments of Director or Assistant Director of Public Health and as even the most gifted Indians cannot equal the European in this respect we hold that these appointments should be held by Indians."

"We are very strongly opposed to any increase of pay or allowances of the Indian Medical Service and to the grant of family passage allowances to the European members of the Service. Indeed the pay of the Service should be reduced in 1924 as promised by the Secretary of State, because the cost of living both in India and England has come down at least 20 per cent. since 1920 when the last increase of pay was granted. The country can bear no more burden. We strongly oppose any discrimination between an Indian and a European member of the Indian Medical Service in the grant not only of pay but also allowances of any kind now or hereafter."

"The Civil and the Military side of the I.M.S. should be separated, the Military side to remain under the control of the Government of India as an all-India Service to be recruited by competitive examination to be held in India; the Civil side, on the other hand, should be entirely provincialised and be under

the control of the Local Government. The salary to be Rs. 300—50—1200 with permission to practise and with an efficiency bar at Rs. 800."

"The grant of exchange compensation allowance is demoralising for one who receives it, for the simple reason that he is prepared to receive additional sums of money when the exchange is against him but is not prepared to accept less pay when it is in his favour; as in 1918-19 when the sovereign went down to Rs. 6-8-0."

"We agree that construction of quarters for Government servants is a desirable concession, but we do not consider the charging of rent at 10 per cent of the salary is too much, seeing that the price of the material and cost of labour have gone so high. We oppose the proposal (1) to reduce the rent to 7 per cent. of the officer's salary and (2) that the Government should be responsible for providing a house for every officer either by giving him a Government bungalow or by renting one for him."

"The present rates of pension are quite generous and no change is justified. We are opposed to commutation of more than one-third of the pension and the concession if made into a right is very likely to lead to abuse."

"The demand of European members (in regard to leave) is preposterous and the country cannot afford it. We consider that the present rates are satisfactory."

iv. *Medical Attendance*:—"Europeans coming to India should be prepared to submit to treatment by Indian doctors. No Indian doctor has been appointed in England to look after Indian officers and other Indian residents in England of whom there are several thousands, and there are several Indian doctors settled in England who have extensive practice exclusively among English people."

"An Indian life is as valuable as a European life; and if a European is entitled to a European doctor, the millions of Indian people in this Presidency are equally entitled to Indian doctors."

"The suggestion of giving the European Medical officers the best stations and the best equipped hospitals in groups in the Presidency is extremely mischievous and cuts at the root of the principle of Indianisation. We strongly object to any preferential treatment on racial grounds."

"We do not find any justification for retaining so many European members of the Indian Medical Service in the Presidency Town of Madras, while they could be better employed in looking after their needy countrymen in the districts."

v. *Specialist Treatment*:—"We challenge the suggestion that facilities for training as specialists in medicine, surgery &c, should be provided for British doctors only. The suggestion is mischievous for the additional reason that India would be expected to train foreigners as specialists, at her expense, who may, at any moment, and certainly on retirement, leave the country with all the knowledge so gained at the great expense of the Indian patients and the Indian purse."

"India wants her children to be trained as specialists and not those of a foreign land. The suggestion, therefore, that an expert and his understudy should be British in each of the subjects mentioned (medicine, &c.) clearly aims at keeping India perpetually dependent on foreign skilled labour. At this rate there is no hope of India now or in the near future becoming self-sufficient so far as the medical needs are concerned. Could any suggestion be more wicked?"

The South Indian Medical Union's Memorandum.—We cull the following extracts from the memorandum submitted to the Public Services Commission by the South Indian Medical Union:—

"The Union urges the long-felt need of confining the I.M.S. to purely military duties."

"The present system is such that all superior appointments in the State Hospitals and Medical Schools are almost entirely monopolised by the Indian Medical Service and that there is so little scope left for the Indian talent to distinguish itself in the Civil medical work."

"The I.M.S. takes away the cream of medical practice in the country, as the members of that Service are placed in an exceptionally favoured position with such special privileges not ordinarily enjoyed by an average medical practitioner in the country, of utilising hospital apparatus, Nursing Homes, Bacteriological Institutes, X-Ray plants and such like aids which are ready at hand and provided for them by the State."

"At the present time, owing to Service conditions, the I.M.S. Doctor is considered to be a specialist in all branches of medical science so that one finds him a physician one day, a surgeon the next day, an optician or a sanitarian next and so on. Such a rapid evolution is an obvious impossibility."

"The South Indian Medical Union is in entire agreement with the Medical Unions of Bombay and suggests that Honorary system of staffing the Civil Hospitals with private medical practitioners as in the United Kingdom should be extensively adopted."

"They see no reason why British doctors should not settle down in practice in this country as the English Barristers, Dentists, Engineers and such like professionals."

"The Union puts forward the suggestion that all further recruitment to the Medical Services, both the I. M. S. and the Provincial Medical Service, should be stopped. The existing members of these Services should continue to serve their time, and as they retire or otherwise vacate their posts, their places

should be gradually replaced by private medical practitioners as Honorary Physicians or Surgeons, the paid staff in the Civil Hospitals being entirely composed of House Physicians and House Surgeons appointed every six months, and these latter are freshly qualified young doctors, resident in the hospital premises with free board, lodging and laundry supplied, and a small honorarium given."

"The Union is strongly opposed to the creation of any new superior Provincial Service in addition to the already existing Provincial Service. Although they do not advocate the immediate abolition of the existing Provincial Service, they certainly feel convinced that all further recruitment to it on service basis should stop and the scheme of honorary workers brought in gradually as vacancies arise. The Union feels convinced that a large number of fully qualified and efficient practitioners will be willing to offer their services for such honorary appointments in the Hospital and Medical Schools, and that this will not only stimulate the free growth of the independent medical profession, but also help to maintain a higher standard of scientific methods in their medical work both inside and outside these institutions. The result is, that the public at large, and not merely the sick that need institutional treatment, are thus provided with a highly efficient, up-to-date and skilful set of medical men and women."

It is most unfortunate that the work of the Lee Commission in Madras was not so smooth-sailing as it was in the other provinces. There was practically no agreement between the evidence, oral or written, tendered by the European element in the Services and that tendered by the Indians. In the Civil Administration, in Judiciary, Educational, Medical, Police or Public Works, the same tale of "No more Europeans" and "India for Indians" was found to be repeated. The Commission was greatly exasperated on this

score and Lord Lee and his European colleagues in the Commission were considerably embarrassed on the bold, persistent and unflinching attitude taken by the Indian witnesses. A tiresome and twisting cross-examination of the Indian witnesses by the European members of the Commission did not bring about any marked change in their attitude. Whether, under such circumstances, the Commission will change colour, it is too hard for us to prophesy. The die-hard Government which set this Commission afloat has set and "Labour" has risen in the political horizon. How far the present Labour Ministry is going to influence the work of the Commission and how far it is going to acquit itself if the views of the Commission were not in accord with its traditional sympathy with Indian aspirations, time alone can tell.

CURRENT TOPICS.

MEDICINE.

The Treatment of Pyelitis.—(*Deut. Med. Woch.*) By G. DANIEL.

Apart from secondary infections of the renal pelvis, most cases of pyelitis are due to infection with the colon bacillus. The growth of this bacillus is checked in acid urine, and urotropine gives off formaldehyde only in an acid medium. Activity of growth of the colon bacillus is inversely proportional to the concentration of the urine. On the basis of these observations, a system has been adopted, the chief features of which are reduction of fluids and the prescription of an acid-forming diet, with phosphoric acid, urotropine, salicyl preparations and—further to concentrate the urine—diaphoretic treatment. Under this regime the patient excretes concentrated acid urine, and the quantity being reduced, micturition becomes less frequent. The urine being retained a considerable time in the bladder, the chances of its disinfecting the structures with which it comes in contact are increased. The results are excellent, fever, pain, pyuria, and frequency of micturition disappearing, and the urine becoming sterile. Failures occur only when, on account of renal disease, the patients cannot excrete concentrated urine, or when they harbour a particularly refractory strain of colon bacillus.

Since 1917 the writer has practised a modified form of this treatment at the University Hospital in Budapest. Fourteen cases of pyelitis were treated under ambulant conditions, and twenty-four in hospital. For the first three or four days the fluid intake was restricted, not more than 300 gm. being given every day from the second to the fourth day. Three times a day 1 gm. of urotropine and 1 gm. of sodium salicylate were given with a little phosphoric acid. Twice a day hot air baths were given for twenty to thirty minutes to promote sweating. From the fifth to the seventh day the drugs hitherto given were discontinued, and the system flushed with large quantities of water. Every day the s.g. and reaction of the urine were noted, and on the last day a microscopic and bacteriological examination of the urine was made. According to the findings of these examinations, the treatment was or was not repeated. When a bacteriological examination was not feasible, the results were gauged by the temperature, symptoms and the presence or absence of pyuria.

In practically every case this treatment reduced the temperature, whether it was of an intermittent or uniformly raised character. This effect usually followed only one course of treatment, and it was seldom necessary to repeat it once or twice to reduce the temperature to normal. Symptoms, such as pain in the back and elsewhere, usually disappeared a day or two after the temperature had fallen to normal. Frequency of micturition varied with the pyuria; when this diminished, micturition became less frequent, and when the pollakiuria continued, pus was always to be found in the urine. Of all the symptoms, pyuria is the most slow to disappear. It bears no relation to the temperature, and is not always proportional to the severity of the disease. When the disease is complicated by cystitis, pyuria is always considerable. In about one-quarter of all his cases the writer was unable to investigate the final results, because the patients left hospital after the fever and other symptoms had disappeared, but before the urine had become pus-free. In the remaining cases treatment was continued till pyuria was no longer microscopically demonstrable, and this was the case, with two exceptions, after one to four courses of treatment had been given. The great advantage of this treatment is its simplicity, and, apart from the hot air baths, which are not essential, it can be carried out at home.—*The Medical Review.*

Tests for Drunken Motor Drivers.—J. Fog (*Ugeskrift for Læger*, p. 597).—In 1921 the Danish law dealing with drunkenness among motor drivers was revised, and it was enacted that a driver convicted of drunkenness must permanently lose his licence. This severity made it

incumbent to give all cases the fairest and most searching trial. Accordingly, the police co-operated with the Danish Medico-Legal Council, which co-opted for the occasion an alienist and a professor of pharmacology. They devised a system of examination which was so detailed that any practitioner could report satisfactorily, provided he followed it conscientiously. Police surgeons are instructed to make the following investigations:

1. What is the appearance of the suspect? Is he drowsy, are his upper eyelids and features relaxed, are his eyes and face congested, and is he sweating or slobbering?
2. How does he behave? Is he noisy, boisterous, silly, excited, garrulous, restless or heavy?
3. Is his conception of time and space normal? (If it is, say "Yes"; if it is not, repeat statements indicating the contrary.)
4. Test his memory. Ask him, for example, to remember a couple of addresses, or to describe the accident which led to his arrest. Or ask him to describe some event indicated by a picture in an illustrated paper.
5. Note his speech. Is it thick, nasal, lisping, stammering or stumbling? Make him repeat difficult words or read aloud a small newspaper notice.
6. Notice his gait. Is it swaying and ataxic? Perform Romberg's test.
7. Are the movements of the hands steady? Test his handwriting by making him write his name, age, occupation and address.
8. Examine his pulse, noting whether it is irregular and rapid; note the size of his pupils and their reaction to light, and test his sense of pain.
9. Does he smell of alcohol?
10. Are there signs of other diseases such as epilepsy or apoplexy?
11. Add any other observations bearing on this matter: (a) Has the examination revealed symptoms indicating that his condition is not normal? (b) Is it proved that the symptoms found are due to alcohol? (c) Is the condition one of drunkenness?

Fog was commissioned by the police to conduct on these lines medical examinations of persons suspected of drunkenness while driving. In the course of about 9 months he investigated 50 cases, taking great care to examine the suspect at once, lest the condition in which he was

arrested should have changed. Unless the arrested person was likely to be violent and dangerous, he was left alone with the medical examiner, who did his best at once to disarm suspicion of hostile motives by assuring the culprit of the impartiality of the examination. Without such an assurance the atmosphere in which the examination is conducted is liable to be unsatisfactory. Great patience must be exercised, just as in an examination for lunacy. The questions must not give the impression of being deliberate pitfalls. It is remarkable how smoothly the examination may proceed once the excitement and suspicion of the arrested person have been allayed. About three-quarters of an hour were required on the average for each examination. Unless the examiner has had some practice in these cases, he is apt to misconstrue the excitement of a perfectly sober man who has recently played the principle part in a motor accident, and has been further upset by falling into the hands of the police. Such excitement usually wears off in the judicial atmosphere in which the medical examination is conducted. The long time taken to complete the test serves another useful purpose. A motorist may have acted under the influence of alcohol, but if he is an old tippler, he may pull himself together for a short time, fulfilling the requirements of even stringent tests. But if he is really under the influence of alcohol, he cannot maintain this state of pseudo-sobriety for long; sooner or later he commits himself.

It was found necessary to recognize two degrees of alcoholic poisoning—a slighter degree described as "under the influence of alcohol," and a more severe one described as "drunkenness." In the period under review, 9 persons were discharged as sober, 30 as suffering from "slight alcoholic poisoning," and 11 as being "drunk." In 34 of the 50 cases, the examination was made between 8 p.m. and 8 a.m. The value of the various tests differed greatly. The fourth test proved very searching. All the persons certified as drunk, and nearly all certified as suffering from slight alcoholic poisoning, were betrayed by it. Two of the persons discharged as sober could not do simple sums in arithmetic or count figures backwards. The gait of some sober persons is apt to be clumsy, and Fog found that movements of the hands and handwriting were uncertain guides to sobriety; a bad handwriting is a common characteristic of many sober persons. The pulse-rate was no guide whatever. Indeed, it was slightly more rapid in the persons discharged as sober than in those certified as under the influence of alcohol. The examination of the pupils was also not very helpful, and smelling the driver for alcohol was helpful only when he did not smell at all of it. The absence of such a smell is of great value. In none of the cases examined were signs of disease other than alcoholic poisoning found. In one case, a car collided with a cyclist. It was reported that the car had been pulled up quickly and surely. The driver was a business man, aged 36. His face was slightly flushed, but otherwise looked natural. He was somewhat excited and annoyed at being detained by the police. He smelt of alcohol, but refused to state what he had recently drunk. He passed practically every test, and was discharged as sober.

The scheme of examination is not exhaustive, and such incidents as violent vomiting or severe hiccough may have to be recorded in addition to the answers to the regulation questions. Violent outbreaks of passion

have also sometimes to be recorded as well as great lassitude. But, on the whole, the system is effective, and the practitioner who carries it out conscientiously is not likely to go astray.

Alaying Thirst with Sea-Water.—In view of the terrible experiences undergone by the shipwrecked crew of the *Treasure*, the writer gives the following extract from Warbasse's "System of Surgery." The method is of great practical importance, and not sufficiently known.

"Thirst assuaged by sea-water is a possibility which has developed out of the surgeon's experience with protoclisis. M. Roberts (*Brit. Med. Journ.*, February 16, 1918) reported the experience of R. Graham, who fasted during a sea voyage and injected daily 8 litres (2 gallons) of clean sea-water into the rectum. He drank no fluids during the first 5 days, and experienced no sensation of thirst until the sixth day, when he drank half a glass of water. During this period he felt well, his pulse was normal, his strength was good, and his bowels moved regularly. This demonstration is important because of its possibilities in the event of shipwreck or other circumstances depriving the individual of access to fresh water. In the absence of a fountain syringe, apparatus may be extemporized. A bottle with its bottom removed, to act as a funnel; a piece of rubber, wooden or metallic tubing; a tobacco pipe; or a piece of rubber cloth or tarpaulin, rolled into a tube about a piece of spirally wound wire, may be used for insertion into the rectum for the purpose of introducing the sea-water. A funnel may be extemporized from any of the above mentioned materials. For lubricating purposes, grease, oil, soap, saliva or other glairy or viscid fluid may be used.

"When no material for constructing a tube is at hand, the individual may be placed in the exaggerated knee-chest position with the anus in the superior position; the anus may be opened by means of three or four lead pencils, pegs, or rolls of cloth of a similar size, inserted about 5 or 6 cm. (2 or 2½ inches), and salt water poured through the interstices. If no such apparatus is available, two fingers may be inserted into the rectum, slightly separated, and the water poured between. If no receptacle is to be had, the water may be dipped up in the hand."—*The Medical Review*.

Intravenous Injection of Peptone in Epilepsy.—By D. M. VASAVADA, *Asst. Surgeon*.—Taking a hint from the Medical Annual for 1922 (page 304) that peptone injection may prove of value in Epilepsy, I tried this treatment on three of my patients. Two of these were men past adult age and one a young boy of 14 years. My first patient an adult of 48 years had suffered from epilepsy for about two years and had broken his clavicle in one of the fits. I put him on Peptone injections and fits which used to come about twice a week immediately stopped and no more have occurred for the past 7 months. He had previous to this treatment received full doses of bromides without any appreciable change. The next was a boy of 14 years. He had the fits for more than 3 years. He took 4 injections of peptone. The interval began to lengthen but the fits did not stop altogether and he left the treat-

ment before a thorough trial could be given. Third patient, also a man of 45 years, had the fits for two months only and used to have them at intervals of about 8 or 10 days. The fits had started after an indefinite illness of one year. He was put on Peptone treatment direct without any bromides. The fits have not appeared since then and it is now over two months since last fit and he says he is feeling much better than what he was for the past year.

It may be noted that in all these cases there was no history of suspicion of alcohol, syphilis or excesses of any kind. In fact no exciting cause could be found except the declining vitality in the two adults. Those who benefitted remarkably were the adults, the younger boy not responding to the treatment so well. I therefore believe that there are some forms of epilepsy in which Peptone injections seem to exert an almost specific effect.

The Peptone I used was bacteriological Peptone 4 p.c. solution sterilized in Autoclave. The first dose was 2 c. c., and 2nd 5 c. c., and the 3rd 10 c. c. of this solution on alternate days intravenously and subsequently 10 c. c. till 6 of these were given also on alternate days, and then at longer intervals of once a week or fortnight. My first patient is still taking an injection about once a month for fear of a relapse, but this may not be quite necessary. Those who have greater opportunities of dealing with such patients might give it a trial and let us know of their experiences. If by this treatment we are enabled to drag out a few from those passing into incurable wrecks we can claim to have done something for humanity.—*I. M. Record*.

BOOK REVIEWS.

STAMMERING, CLEFT PALATE SPEECH LISPING.

By K. EMIL BEHRE. Second Edition 101 Pages. Price 3s. 6d. Baillière, Tindall and Cox, 8, Henrietta Street, Covent Garden London 1922.

This little book deals with stammering, its causes and treatment. Next in importance to stammering, the author deals with the Cleft-palate speech. Stammering, the author rightly regards as a nervous disorder. So, every effort must be made to remedy the psychic causes and stabilise the nervous system. The author strongly advises breathing exercises and re-education of the muscles of speech. The author deals with the subject in a very concise way. To one who is interested in curing the causes of stammering this little book will be very useful.

AROMATICS AND THE SOUL—A STUDY OF SMELLS.

By DAN MCKENZIE, M. D. 164 pages Price 7s. 6d. William Heinemann (Medical Books) Ltd. London 1923.

The author, a distinguished laryngologist, treats in this book various theories of olfaction at length and clearly enough to be understood by ordinary people. The chapter on 'Dust of the Rose Petal' gives some of the author's vivid pictures of his own memories roused by smell. The whole book shows how much of mystery and imagination lies within the domain of strictly scientific work, equally to be appreciated by both scientific man and the ordinary layman.

The subject is dealt with under the following headings viz Olfaction and public health; Olfactory memory; Smell and speech; Smell and the personality; Theories of olfaction &c, &c. The book contains a fund of information, which is interesting even to a layman.

ASTHMA.

By FRANK COKE, F.R.C.S. 260 Pages, Price 15 S. John Wright & Sons Ltd., Bristol, Simpkin, Marshall Hamilton, Kent & Co., Ltd., London.

Asthma is a disease which defies even the best of physicians. Its treatment is a matter of speculation. One line of treatment in one case does not suit the other. The treatment which has cured at one time does not suit at another time in the same patient. The causes of asthma are very many. The author has done a great service to the profession by publishing the book under review which deals with the subject under the following chief headings, viz., Anatomical and Physiological aspects; Heredity cause of asthma; Types of asthma; treatment of acute paroxysm, and between attacks; Nasal factor in Asthma and its treatment &c. As for the treatment of acute Paroxysm, Adrenalin stands first, other drugs come next in importance in their order such as Morphia, Atropine, Heroin, Pituitrin, Chloroform &c. As for the treatment between the attacks and cure of the complaint, the author's treatment may be summarized as follows:—

Avoidance of the protein to which the patient is sensitive; Specific protein therapy; Vaccine and non-specific protein therapy; Exercise; Diet; Change of Climate &c. We entirely agree with the author in many of his observations. The book is very well-written and we recommend it to every physician in this country.

THE PATIENT'S VIEW POINT.

By P. J. FLAGG, M. D. Author of "The Art of Anaesthesia" 182 Pages S. 1-30. The Bruce Publishing Co., Milwaukee, Wisconsin, U.S.A.

The first chapter deals with the process by which materialism is acquired in the medical school. Succeeding chapters deal with the intramural life before and after graduation, Incense to mercury, Frankincense to God, general practitioner and specialist, the physician and the Nurse, Professional fees &c. The book is worth reading and interesting.

ENVIRONMENT AND RESISTANCE IN TUBERCULOSIS.

By ALLEN K. KRAUSE. 137 Pages. Williams and Wilkins Co. Baltimore, 1923.

This book presents the nature of environment and resistance and their relation to the pathology, diagnosis, symptoms and treatment of tuberculosis. The discovery of the tubercle bacillus has made environment a new force in tuberculosis. An environment of dirt and darkness and overcrowding became an un-failing reservoir of germs and therefore of consumption. The old saying "clean up your lung blocks and you will clean up tuberculosis", is perfectly true. The book contains very useful and instructive information.

WHAT TO EAT AND HOW TO PREPARE IT.

By ELIZABETH A. Monaghan published by George H. Doran Company, New-York—Price 1-50 Dollars—Rs. 6, postage 10 as.

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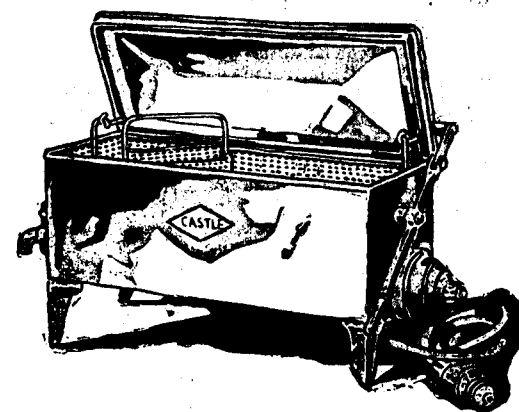
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THE HYGIENE OF MARRIAGE.

By ISABEL EMBLIE HUTTON, M.D. with a foreword by Prof. A. Louise McIlroy M.D., D.S.C., O.B.E., Published by William Heinemann (Medical Books) Ltd, London 1923. Price 5 S. Rs. 5-4-0 Postage 0-8-0.

It is an admitted fact that shyness and ignorance in men and women often stand as impregnable barriers in the way of consummation of happy marriages. Divorces and domestic quarrels have become the order of the day and a correct understanding, therefore, of the mutual sex relationship, of the sanctity of marriage and other allied physiological conditions will doubtless dispel some of the horrors of married life and help a good deal in the solution of the many perplexing sex problems that now confront the modern world. This book which treats about the various aspects of marital life supplies a long-felt want and must certainly prove to be a good guide, friend and philosopher of every married or about to be married couple. It is written in a clear, concise and non-technical style and is evidently intended to appeal even to the average intellects among the laity. We strongly recommend its use by the public who care for healthy, happy and harmonious homes.

THERAPEUTICS OF SNAKE-POISONINGS.

By Parashnath Banerji, printed by the Indian Daily Press, Calcutta Price 1 Re Eng 2s. nett.

In a tropical country like India, the mortality from snake-bite is heavy and increasing annually and though mighty medical intellects have struggled and are still struggling hard to find a satisfactory remedy for this deadly poison, their efforts so far, have not been crowned with complete success. In these circumstances, this booklet, which treats about the therapeutics of Snake-Poisonings will prove a welcome and valuable guide to every individual. The author claims to have invented a "specific" for snake-bite and assures us that it has been tried in several cases with excellent results. Mere inhalation of the Antivenin Drops, for, that is the name of the specific, is enough to bring a person actually dead from snake-bite, to life again, if administered within three hours of death, by means of artificial respiration. If it be so, this specific will be a real boon and a blessing to humanity and no praise will be too high and no reward will be too great for the services the inventor has rendered to the medical world. Let every home in every clime buy it, try it, and pass its unbiassed verdict on it. Till then, we reserve our judgment on its efficacy and usefulness.

DENTAL AND ORAL RADIOGRAPHY.

By JAMES DAVID MCCOY M. S., D. D. S. F. A. C. D. with 116 illustrations, III Edition. 197 Pages. Price Rs 13. St. Louis C. V. Mosby Company U. S. A. 1922.

This is a text book for students and practitioners of dentistry. The radiography is as essential in the practice of dentistry, as in the field of medicine and surgery. The author has taken a good deal of pains to place before the reader all the necessary technic to handle X-rays apparatus, even though he may be a beginner. In this part of the country dental surgery is in its infancy, though the scope is very wide. The chapters on 'Technic of Dental and Oral Radiography—the intra and extra oral methods, are very instructive and interesting. The chapter on the interpretation of dental and oral radiograms and another chapter on the indications for the use of the X-ray in the practice of dentistry, are fully illustrated and is sure to be of great use to the practitioners, especially for beginners. The last chapter deals with the dangers of the X-ray and the method of protection, which is very essential for every X ray worker. The illustrations in the work are very instructive. We recommend the work to every dental practitioner.

ANATOMY OF THE FEMALE PELVIS.

By F. A. MAGUIRE, D.S.O., M.B., C.H.M., F.R.C.S. with 4 illustrations. 1922. 115 Pages. Angus and Robertson Ltd. Sydney.

This book is based on lectures and demonstrations given to third year medical students in anatomy and to fourth and fifth year students in gynaecology, by the author. The writer has collected under one head all the anatomical knowledge required of a student when he approaches the practical side of his work in gynaecology and further it gives the relation of the soft parts to the bony landmarks. The chapter on 'Summary of Gynaecological Examination' is very instructive and practical. This book is very useful not only to students, but also to practitioners. We recommend the book to our readers.

THE 'NAUHEIM' TREATMENT OF DISEASES OF THE HEART AND VESSELS IN ENGLAND.

By L. THORNE THORNE, M.D. B.S. M.R.C.S. Eng. L.R.C.P. Lon. Sixth Edition. 232 Pages. 7s. 6d. Bailliere, Tindall and Cox, 8, Henrietta Street, Covent Garden, London 1923.

This useful book gives a practical description of the methods of administering "Nauheim" treatment in England. It gives in such detail that even medical men who have no previous knowledge of the treatment may, by carefully following the directions, avail themselves of this very valuable Therapeutic agent, for the treatment of affection of the heart and vessels. The subject of the book is dealt under the following most important chapters.

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and administration of the exercises. The author lays a good deal of stress on resistance. Exercises which are of use as an adjunct to the bath and may be very beneficially given in cases where it is not possible to give the baths or as a

preliminary treatment before beginning the course. The Poligraphs given in the book to illustrate all pulse tracing are very interesting and instructive. The chapter on Arterio-sclerosis and High Blood-pressure contains the latest researches on the subject. This book will be of immense use for every physician.

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BOOKS RECEIVED.

Therapeutics of Snake Poisoning. By Panesh Nath Banerjee. Pages 47. Price Re. 1. Indian Daily News Press. Printers. Calcutta.

Surgical 'Dont's and Do's' by C. Hamilton Whiteford M.R.C.S. L.R.C.P. Honorary Surgeon to the Plymouth Infirmary. Consulting Surgeon to the Great Western Railway. (Plymouth centre). Page 46. Harrison & Co. Ltd., 44-47. St. Martin's Lane London W. E. 2. Price 3 Sh.

Clinical Memoranda. For General Practitioners by Alex Theodore Brand M.D., C.M., V.D. & John Robert Keith M.A. M.D., C.M. Second (Edin) (1923). Pages 375. Price 7 Sh. 6d. Bailliere-Tindall & Cox 8 Henrietta St. London.

Carriers in infectious diseases. A manual on the importance Pathology, diagnosis, and treatment of human carriers. By Henry J. Nicholas. Medical corps. N.S. Army. Asst. Professor. of Bacteriology, Parasitology and preventive medicine, Army Medical School. Washington D. C. With a chapter on carriers in Veterinary medicine. By R. A. Kelar. Pages 184 with illustrations. Price 3.50 Dollars. William Wilkins Company, Baltimore.

The Bacteriophage. (Eng Edition) by F. D'Herelle. Pasteur Institute. Authorised translation by George H. Smith P.H.D. Asst. Professor of Bacteriology and Pathology, Yale University School of medicine, (1922). Pages; 287 with illustrations. Price 4.50 Dollars. William Wilkins Co, Baltimore.

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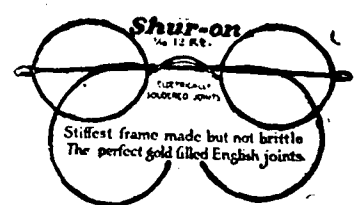
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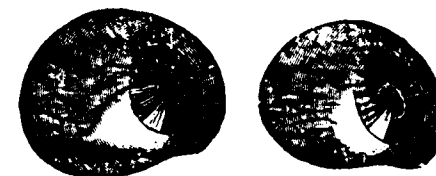
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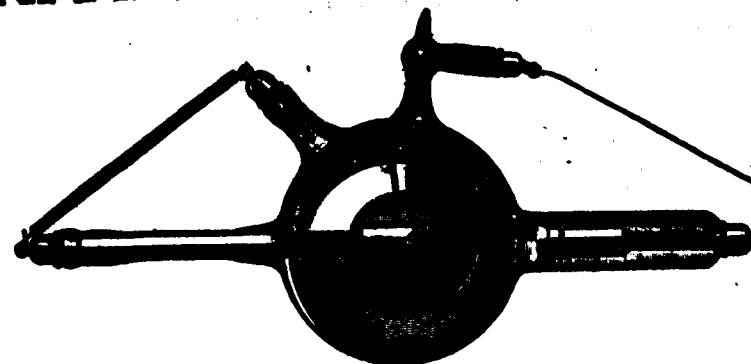
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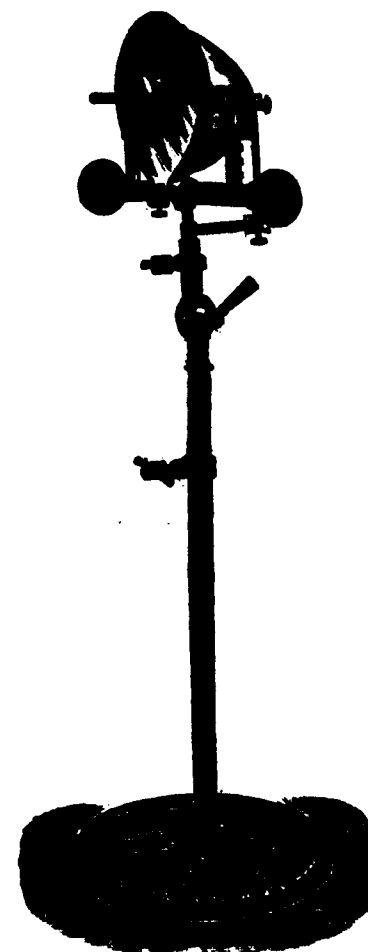
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