A British Medical Association Lecture

ON

DIABETES IN RELATION TO THE DUCTLESS GLANDS.

BY

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A LIVING organism, like a spinning top, is only in equilibrium because it keeps going. But, unlike a top, it is provided with delicate mechanisms for readjusting its balance should this be disturbed. Disease is the resultant of the action of some external force and the reaction of the body. The reaction may be adequate to restore equilibrium to its former position—that is, a return to health; or the external force may be sufficient to overthrow the balance altogether—that is, death ensues. But short of these extremes a new though inferior position of equilibrium may be attained—that is, more or less compensation is acquired. In this new inferior position the machinery works with more friction, and the reserves are thereby reduced. Thus the body works best at a temperature of 98.4° , with five million red corpuscles per cubic millimetre, with a gastric juice of 0.2 per cent. HCl, and alveolar air containing 5 per cent. CO_2 , and blood containing 0.1 per cent. of sugar, though capable of working with figures considerably in excess or defect of these. In this regulation of balance the endocrine glands play an important part. Broadly they fall into two groups, the accelerators and the retarders of metabolism. In general terms, the accelerating endocrine glands are controlled by the sympathetic and the retarding co-operate with the parasympathetic. These constitute the two divisions of the lowest level of the nervous system—the visceral, vegetative, or autonomic system, which, acting apart from the will and normally without exciting sensation, subserves the functions of organic life. The sympathetic is katabolic in its activity, converting the reserves of the body into kinetic energy, and defensive in its manifestations, while the parasympathetic is anabolic, building up reserves.

The sympathetic is militant, the parasympathetic peaceful. Emotions which in the primitive state activate us for fight or flight do so through the sympathetic. One of the most important steps in this mobilization concerns the blood sugar. Active muscles consume three and a half times as much sugar as resting muscles, so that it is not surprising to find that sympathetic stimulation leads to increase of the sugar in the blood. This it apparently does partly through the thyroid, pituitary, and adrenals. The close association between endocrine glands and the sympathetic is most clearly seen in the case of the adrenals. In the worms there are cells in the central nervous system which are the common ancestors of both the sympathetic ganglion and the adrenalin-secreting cell. In the lowest vertebrate, the lamprey, the sympathetic cells are scanty, but there is a wide distribution of adrenalin-secreting cells (J. F. Gaskell). As evolution proceeds these secreting cells gradually give way to an increasing development of the sympathetic system, so that ultimately their only surviving remnant is the medulla of the adrenals. Not only is this medulla stimulated to secretion by the sympathetic, but in its turn it increases the sympathetic response. Stimulation of the sympathetic or injection of adrenalin, therefore, alike increase the blood sugar and lower carbohydrate tolerance. There is a similar correlation between the sympathetic and the thyroid. The effect of the pituitary in increasing the blood sugar and lowering carbohydrate tolerance is proved, as is the effect of sympathetic stimulation in increasing its secretion, but the converse effect of its secretion on the sympathetic has not been shown.

The effect of the parasympathetic in building up reserves is seen particularly in its action on the assimilation of food. It plays the chief part, both directly and indirectly, in providing the digestive juices and in maintaining the due tone necessary to peristaltic movements. Now one of the digestive glands, the pancreas, not only provides a powerful

external secretion, but it supplements this by an internal secretion which leads to the building up of ingested sugar into more complex colloid combinations, which enable it to escape excretion by the kidney, thus increasing carbohydrate tolerance. In passing, we may note the advantageous position in the circulation where the pancreas pours this secretion into the blood stream, side by side with the dextrose absorbed from the alimentary tract. We may surmise that some of the absorbed sugar is at once stored in the liver as glycogen, while by means of this internal secretion of the pancreas the rest is built up through a series of increasingly stable colloid combinations into the tissue cells. Support can be adduced for this surmise in two facts—one that the sugar in normal blood behaves like a colloid and the other that the pancreatic internal secretion is much less effective if introduced at any other part of the circulation than the portal vein.

Now, whenever sympathetic and parasympathetic fibres are distributed to the same structures they are antagonistic in function. The pancreas is not as much under nervous control as some other glands, but, so far as the evidence goes, it points to the vagus having a secretory effect upon it, although it is true that in large part its secretion is excited by chemical means. It would be in accordance with this general plan for the sympathetic to be inhibitory to the pancreas, both for its external and internal secretions, and it would therefore appear that sympathetic stimulation would increase blood sugar and lower carbohydrate tolerance both by stimulating the accelerating or katabolic glands and by checking the anabolic gland. My first generalization is then:

The sympathetic mobilizes the sugar into the blood by means of the endocrine glands, for purposes of defence, while the parasympathetic stores it in the tissues as a reserve.

Sugar serves three useful purposes in the body: it can be stored in the liver as glycogen, or in various tissues as fat, or it can be utilized at once as a source of muscular energy. If these three methods are inadequate, some will appear in the urine. Recent work has shown that we get a much more accurate idea of what is happening by examination of the sugar in the blood than in the urine. I have given the normal level of the blood sugar as 0.1 per cent. It rises within about ten minutes of drinking a solution containing sugar, and more gradually after eating starch, to about 0.14 per cent. If it rises above this point an overflow is likely to occur into the urine. This Graham has called the "leak-point," and it varies in different individuals. In some it is abnormally low. Such persons constantly pass about 1 to 2 per cent. of sugar, whatever the diet. Their glycosuria is non-progressive and causes no symptoms, being only discovered on routine examination. There may be other evidence of a renal lesion, more often there is not. Such a condition has been compared to the glycosuria temporarily induced by injecting phloridzin, when the kidney actively secretes sugar. A glycosuric of the ordinary type who only passes 1 to 2 per cent. of sugar on a mixed diet might be expected to pass none on a strict diet. But these cases continue to do so, and can therefore often be recognized without the aid of an estimation of the blood sugar. Strict diet appears to do them harm.

I recently saw an interesting variation from this type. A lady, aged 52, was known to have had glycosuria for twelve years. Sometimes she had dieted, sometimes not—it made little difference to her health; if anything, she was better on a mixed diet. Her blood sugar was 0.061 per cent. On strict diet she generally passed some sugar, and she also developed acetonuria. As a result of strict dieting her blood sugar rose to 0.19 per cent., and on giving her 25 grams of dextrose it rose to a maximum of 0.36 per cent. in half an hour. Yet she only excreted 4 grams of the 25 grams given her. Now, this shows that she could assimilate sugar, but that the effect of dieting was actually to push her leak-point up. This would appear to be an attempt on the part of the body to prevent the escape of a valuable foodstuff. Seeing that I was actually producing a hyperglycaemia by dieting I abstained from further treatment which was likely to do harm; I allowed starches freely, which would enter the blood gradually, and merely prohibited sugar as such.

An ordinary diabetic shows an excess of sugar in the blood—a usual figure is 0.25 per cent., but it may rise to 0.3 or 0.4. Even when he has been freed from glycosuria

the blood sugar may remain as high as 0.25. Yet, as we have seen, a normal individual will leak sugar somewhere about 0.15, so that the kidney must raise its leak-point in diabetes. Here again it seems there must have been an attempt to establish a new position of equilibrium and to prevent escape of foodstuff. It may be asked, In what respect is such an individual in an inferior position to the normal? Why is 0.1 per cent. the optimum blood sugar content? Apparently the 0.1 per cent. is carried in combination, whereas sugar in excess of this amount is in simple solution. To carry an excess of crystalloid in simple solution has certain definite drawbacks—it alters the osmotic pressure of the body fluids, which may, for instance, damage the translucency of the crystalline lens; may infiltrate the nerves, causing interstitial neuritis; and provide a pabulum for septic organisms, producing boils and carbuncles. All these seem to be the direct results of hyperglycaemia. I have even known gangrene occur in a diabetic during a time when he was free from glycosuria but still had hyperglycaemia. In the same way any wide departure from the optimum five million red corpuscles per cubic millimetre produces disturbances. If much below this figure, insufficient haemoglobin is carried for the needs of the organism; if much in excess, the viscosity of the blood is raised so much that the circulation is seriously slowed. Indeed, if the new figures were better than the old they would tend to replace them in evolution.

If we seek for the explanation of the difference between free and combined sugar we must turn again to the pancreas. Its internal secretion acts like an amboceptor joining the sugar to larger molecules such as protein. Normally there are enough of them to carry 0.1 per cent. of sugar in this way. If there is more sugar than this to be carried in the blood, overflow occurs; though this may be diminished by raising the leak-point. But if the pancreatic secretion is inadequate, less sugar is carried in the combined form and glycosuria occurs on an ordinary diet. There is thus no hard and fast line between simple alimentary glycosuria and "total" diabetes when no sugar is assimilated. It is merely a question of the relation between the pancreatic amboceptor and the needs of the body.

In health there is quite sufficient amboceptor to deal with an ordinary diet, though if 150 to 200 grams of dextrose are given at one time some will appear in the urine. But this is an amount which no one will voluntarily take, and the administration of such an amount may provoke its rejection by vomiting. The capacity of the pancreatic internal secretion thus has a definite limit, and if other agencies tend to keep up a persistently high level of blood sugar overflow will occur. Such agencies exist, as we have seen, in the adrenals, thyroid, and pituitary.

We may next inquire whether a similar antagonism between this group and the pancreas can be shown to exist in respect of their other functions. To a certain extent it can. Thus Pemberton and Sweet showed that removal of the adrenals was followed by a continuous flow of pancreatic juice, which could be temporarily inhibited by injection of suprarenal extract. Again, it is well known that adrenalin dilates the pupil of an excised eye, but not of the intact eye of a normal individual. Therefore something inhibits the adrenalin effect. Now, Loewi found that in a depancreatized dog adrenalin did dilate the pupil. He therefore suggested the instillation of adrenalin into the eye as a test for pancreatic insufficiency. Though not infallible, it is a useful aid to diagnosis. A drop of the 1 in 1,000 solution is put into the right conjunctival sac, five minutes later it is repeated, and dilatation of the pupil, often eccentric in form, is looked for between twenty minutes and one hour afterwards. If this reaction is positive, it is certainly suggestive of pancreatic insufficiency. But it may also be present in hyperthyroidism, suggesting that the thyroid excess inhibits pancreatic activity, allowing adrenalin to show its dilator action unchecked.

It must be admitted that the ordinary case of diabetes shows no evidence of endocrine disease, and conversely if endocrine disease is present it presents other manifestations besides altered sugar tolerance. Allen, who strongly upholds the pancreatic theory of diabetes, admits that the disease may reach a fatal issue and yet the pancreas be as good as normal. And more recently he has stated that the hydropic degeneration of the cell islets of the pancreas

which has been observed may be the result and not the cause of diabetes. He inclines, that is to say, to the view that diabetes shows itself *through* the pancreas, rather than that it is a disease of the pancreas. And he is prepared to accept a nervous origin for this pancreatic insufficiency.

If there is organic disease of the pancreas we should expect to find other signs besides glycosuria. The comparative value of these signs has recently been fully discussed by Sir Archibald Garrod in his Schorstein lecture. It will be sufficient if I say here that the signs on which I have learned to rely most are:

(1) An excess of unsplit fat in the stools—since the pancreas provides the lipase for fat-splitting.

(2) A high diastase content in the urine—since if there is any obstruction to the outflow of pancreatic juice excess of pancreatic diastase will be reabsorbed into the urine and show its presence by the digestion of an unduly large amount of starch paste. (For technique see Corbett, *Quarterly Journal of Medicine*, 1913, vol. 6, page 351.)

To these I might add (3) the adrenalin eye test.

If these three tests were positive and glycosuria were present the diagnosis of pancreatic disease might safely be made. Jaundice would further suggest that the head of the gland was involved in the process, while severe pain would suggest pressure on the nerves. In acute disease of the pancreas the eye test would probably be positive and the diastase in the urine be very high—say, 300 to 500 units, as against the normal 10 to 30. The pain would probably be agonizing and cause inhibition of intestinal peristalsis, with resulting obstruction. But glycosuria only occasionally occurs in such cases. With a chronic obstruction to the head of the gland, such as a new growth, the eye test would probably be positive and the diastase moderately raised, say, 50 to 150 units, but pain would not be at all a marked feature. Jaundice would be present, but not glycosuria. It is in a chronic inflammatory lesion of the whole pancreas that glycosuria might be expected, but then there would also be a positive eye test, a moderately raised diastase output in the urine, fatty stools, and probably some jaundice. Now all these other symptoms are usually lacking in diabetes, where the glycosuria remains the only symptom we can attribute to the pancreas. We must therefore look further afield for an explanation of diabetes.

That thyroid extract will excite glycosuria is well known. Graves's disease is accompanied by lowered sugar tolerance and sometimes by actual glycosuria, while in myxoedema the sugar tolerance is much increased. Only last week a girl of 25 was sent to see me with a history of thyroid enlargement for the last four or five years, which had increased recently. There was no exophthalmos and no tremors, but the pulse was 100. At the beginning of the illness she had unfortunately been treated with thyroid extract. In March, 1920, she was noticed to be wasting rapidly, and on routine examination glycosuria was found. Even after restricted diet this amounted on June 2nd to 2,800 grains a day. There was also pronounced acetonaemia. The hyperglycaemia was well marked, the blood sugar amounting to 0.308 per cent.—that is, three times the normal. This would appear to be a glycosuria of thyroid origin, but it only differed from ordinary diabetes in showing other definite endocrine symptoms. My impression is that giving thyroid extract may have helped to tip the balance towards diabetes by increasing katabolism and hyperglycaemia.

In a similar way oversecretion by the pars intermedia of the pituitary lowers sugar tolerance and may excite glycosuria. The following are examples:

A man of 33 came under my care in hospital for glycosuria which was accompanied by a right ophthalmoplegia and left-sided facial paralysis. There was severe headache and occasional vomiting. The last two symptoms suggested increased intracranial pressure, though he had no optic neuritis then. A gummatous meningitis involving the base of the brain, and including the pituitary, seemed the most probable diagnosis. In confirmation of this the Wassermann reaction was found to be strongly positive. While under treatment his headache, vomiting, and cranial palsies cleared up, and his carbohydrate tolerance rose to more than 100 grams. Unfortunately he relapsed some months later, and developed right hemiplegia, apparently due to endarteritis of the middle cerebral.

Quite lately I saw an officer who in 1915 had been hit on the forehead by a fragment of high explosive shell which entered the orbit. There seems to have been a free escape of cerebro-spinal fluid from the nose. An attempt to remove the fragment by operation had been unsuccessful. A skiagram pointed to some damage to the posterior wall of the frontal sinus and the left frontal lobe. Two years later he began to show some failure of mental concentration and was occasionally odd in his manner. Two years after this he began to have fits, starting in the muscles of the right side of the face. He seemed dazed in the fits, but did not entirely lose consciousness. He was found to have 8 to 10 per cent. of sugar in the urine, and his blood sugar was at the upper limit of normal, 0.15 per cent. The left visual field was found to be defective on the temporal side.

Here again the pituitary was probably responsible, though an element of doubt arises through a diabetic family history. Yet the x-ray findings, the fits, the mental and ocular symptoms, are very suggestive, and in true diabetes a higher blood sugar might be expected. I hear that under treatment he is keeping free from sugar. At any rate the pituitary, like the thyroid cases, generally declare themselves by their associated symptoms.

The glycosuria, as distinct from the lactosuria, of pregnancy is probably due to the known stimulating effect of pregnancy on the thyroid and pituitary. I was recently consulted in a case of glycosuria in pregnancy which had caused considerable anxiety because there was acetoneuria as well. But the patient had been vomiting a good deal, and I suggested the acetoneuria was merely due to this. Normal blood sugar pointed to the same conclusion. At any rate she was delivered of a healthy child, and the glycosuria cleared up.

Although glycosuria may be excited by adrenalin, the case for a definite adrenal diabetes is not clear. Emotional temporary glycosuria is naturally regarded as due to adrenal stimulation through the sympathetic, and probably some of the cases in soldiers are of this type. It also seems probable that some of the cases of glycosuria in later life, accompanied by raised blood pressure, are of this nature, for the two most obvious results of adrenal excess would be raised blood pressure and hyperglycaemia; but we lack precise evidence. I think we may now advance a second generalization:

Ordinary diabetes shows no other signs of endocrine disease, while endocrine glycosuria betrays other evidences of that origin.

This difficulty led Eppinger and Falta to advance the theory that diabetes was not due to any one endocrine gland but to loss of balance between them. It is easy to understand how such a loss of balance could be brought about if there were definite disease of one of them, just as when one group of muscles is paralysed the opponents show a contracture due to unopposed contraction. Hughlings Jackson's dictum concerning diseases of the brain may be applied to the endocrine system. Destruction of tissue, he affirmed, cannot produce positive symptoms. They must be due to the activities of those elements which remain intact. Thus the obesity of pituitary defect and the general infiltration of the tissues in myxoedema may be referred to unchecked action of anabolic glands. Conversely, when a dog has been rendered diabetic by removal of sufficient pancreatic tissue, the glycosuria is greatly reduced by removal of the adrenals, and when the posterior lobe of the pituitary with the pars intermedia has been removed, subsequent removal of the pancreas does not cause glycosuria. The balance, which had been disturbed by the first operation, tended to recover as the result of the second, although in other respects the animal suffered an additional loss.

But how is the balance upset when there is no disease of any one endocrine gland? I maintain that it must be through the sympathetic, which simultaneously stimulates the glands that lower carbohydrate metabolism while inhibiting the one that increases it. This can be expressed by modifying Eppinger and Falta's diagram thus:

Sympathetic irritation would tend to flood the blood with sugar while diminishing the secretion which combines

that sugar into a form in which it would escape excretion by the kidney.

To some extent the escape might be checked by raising the leak-point, but this is not usually adequate, and is apt to bring complications in its train.

This sympathetic irritation might be due to an emotional cause. As Crile says: "When stocks go down in New York, diabetes goes up." The factor of strain, anxiety, and emotion is clear in many cases of diabetes. Even when it is not clear we cannot always exclude it because of the tendency to push a disagreeable emotion "out of the mind," which merely means pushing it into the deeper levels of the unconscious where it continues to produce its effect. There is a close analogy between diabetes and Graves's disease in this respect. Normally the sympathetic nervous system should act as a whole—as an intensive preparation for action or defence. But in disease dissociation is a recognized phenomenon—for example, the dissociation of sensory symptoms in tabes. In diabetes there is a dissociated sympathetic response which is specialized in the direction of endocrine katabolism, and once this is started it will tend to continue because nervous impulses tend to run along accustomed channels. Taking the three levels of the nervous system as the psychic, the sensori-motor, and the vegetative, an emotional disturbance tends to assert itself at the highest level as an obsession, at the middle level as a functional paralysis, contracture or anaesthesia, and at the lowest level as diabetes, Graves's disease, asthma, or certain digestive neuroses. I am not going to claim all cases of diabetes as emotional in origin. We are far too ignorant of the morbid anatomy of the sympathetic nervous system to assert this. It has been shown, for instance, that in pellagra there are structural changes in the sympathetic which is responsible for the skin changes among others, and it is quite likely that in some cases of diabetes there are organic changes there also. But I do think the present state of our knowledge does justify, as my third generalization, the following classification of cases of glycosuria:

- (1) Organic origin, with structural changes in the endocrine glands leading to (a) overaction of adrenal, thyroid, pituitary, or (b) underaction of the pancreas.
- (2) Sympathetic origin, with no evidence of structural changes in any endocrine gland, but producing a functional (a) overaction of adrenal, thyroid, pituitary, and (b) underaction of pancreas.

But if I stopped here I could rightly be criticized as only explaining part of the disease. As Abderhalden says:

Up to the present, the most pronounced symptom, that of glycosuria, has dominated the entire investigation of problems concerning diabetes, and it is very probable that this is the reason why the disease is, as a whole, so little understood.

I have long felt it a mistake to look upon the disease merely as a disturbance of carbohydrate metabolism. But we must remember that carbohydrate represents at least 70 per cent. of an ordinary diet, and that the carbohydrates are the most readily mobilized of the foodstuffs. If the metabolism is only slightly affected, it will, therefore, be most apt to show this in relation to carbohydrates. There may be a stage in which, although the power of dealing with sugar as such is impaired, the power of converting it into fat is not. Consequently, rather than permit the escape of valuable food material, the body converts it into fat. Hence the glycosuria associated sometimes with obesity. As von Noorden says, some cases of obesity may be looked upon as latent glycosuria. But as the disease proceeds fat metabolism is also impaired, and diacetic acid appears. Further, it has been shown that protein metabolism is involved, and even the metabolism of inorganic salts.

Diabetes is, then, a sign of exaggerated metabolism, evoked through the sympathetic and the associated endocrine glands, which first asserts itself in relation to the most abundant food material, but as it advances expresses itself in relation to all.

Alimentary Rest.

The recent advance in the treatment of this disease by alimentary rest supports this view. The quickest way of compelling metabolism to adopt economical lines is to cut off supplies. In proof of this, we have only to notice the rapid fall of nitrogenous output as soon as no protein food is taken. The old method of treatment was greatly to

increase the amount of protein in the food, thus throwing fuel into the flames, for excess of protein is a great quickener of all metabolic processes. This was accompanied by excess of fat, which was incompletely oxidized with the formation of toxic products. We all had to learn during the war that we could balance our metabolism at a much lower level than we previously thought possible; and what may be but a passing phase for the normal individual must remain a permanent state for the diabetic. He must be permanently underfed to avoid overtaxing his damaged powers of assimilation. Clearly the outlook depends on how much these powers are damaged. If he can balance his metabolism when the calorie value of his food is adequate to maintain life and a fair display of energy, the outlook is good; if he cannot acquire a balance until the intake is reduced too much for this, the outlook is bad; for there must be a limit to this process of reduction.

It follows that some loss in weight is almost inevitable under the treatment, and to a certain extent this is beneficial; the amount of food required bears a ratio to the total weight of the body. The diabetic does best when his weight is somewhat below that which he possessed before the onset of the disease.

The first and most important effect, then, of alimentary rest in the treatment of diabetes is to slow down metabolism. Another subsidiary effect is that pancreatic activity is diverted from the production of external secretion to producing internal secretion, for, as Allen showed, anything which increases the former diminishes the latter. On Graham's plan, which is the one I usually employ, a mitigated fast of forty-eight hours is prescribed—only some tea or coffee, clear soup, and lemonade are allowed; though 2 to 3 ounces of whisky may also be given in suitable cases. This is followed by two days on which nothing but eggs and green vegetables are given. This diet has a calorie value of 1,170 and a carbohydrate intake of about 10 grams. Cautious additions are made every other day to this diet, so that twelve days after the initial fast about 2,000 calories are taken in the day. A large number of patients will become free from sugar on the fast, and will keep free during the successive increases in the diet. In that case I then cautiously add carbohydrate to the diet—say, 5 oz. of milk first, and then every other day $\frac{1}{2}$ oz. of ordinary bread until glycosuria just returns. The carbohydrate tolerance can thus be quickly calculated.

Say, for example, there was no sugar passed on $3\frac{1}{2}$ oz. of bread, but some appeared when 4 oz. were given; 5 oz. of milk contains 6 grams of carbohydrate, and each ounce of bread contains 16 grams of carbohydrate. Therefore 62 grams of carbohydrate were tolerated in addition to the 10 grams in the so-called carbohydrate-free diet.

Having just exceeded the limit of tolerance it will be found quicker to re-establish the balance by giving one day of egg and vegetable diet, then returning to the diet on which sugar appeared, but with only about three-quarters of the previous amount of carbohydrate. After that it is well to give two consecutive egg and vegetable days once a fortnight, preceded by two days of fasting once a month should return of sugar make this necessary. If the carbohydrate tolerance exceeds 100 grams it is only necessary on two consecutive days once a fortnight to halve the amount of carbohydrate taken. Having in this way established standard diet for the individual, I provide for some variety in the diet by giving them a table with some comparative carbohydrate values:

Carbohydrate Values.

1 oz. of bread	...	...	...	=16 grams
2 oz. of potato	...	...	...	=16 grams
2 oz. of green peas	...	...	...	=10 grams
3 oz. of artichoke	...	...	...	=12 grams
3 oz. of baked apple	...	...	...	=15 grams
5 oz. of milk	...	...	...	= 6 grams

Any permutations and combinations of these can be allowed as long as the total does not exceed the limit arrived at as above. Say this is 60 grams: then, for example, 3 oz. of bread and 3 oz. of artichoke = 60 grams of carbohydrate.

If, on the other hand, sugar does not disappear on this scheme it is best to repeat the process once or, if necessary, twice more. But cases which do not become free from sugar on the first fast seldom de well. I have not often used the more prolonged last recommended by Allen

because, though it may render the urine free from sugar more quickly, it has sometimes done so at the expense of the patient's general condition. As far as possible I avoid the use of special diabetic breads, preferring to work the patient's tolerance up to a limited amount of ordinary bread. In this connexion it is worth while to take advantage of the fact that a patient's carbohydrate tolerance is highest at breakfast, no doubt because of alimentary rest during the night.

One of the greatest advantages gained by this method of treatment is that we are no longer on the horns of this dilemma—rapid restriction of carbohydrate on the old plan produced toxic acidosis, while failure to restrict adequately allowed the disease to progress. While it is true that sudden restriction of the carbohydrate intake will lead to acetonuria if a liberal diet of protein and fat takes its place, it is also true that cutting down the whole of the diet practically to zero will be followed by a diminution of acetonuria as well as glycosuria. The normal individual who fasts develops some acetonuria because he has to live on his fat; but this is not enough to produce toxic symptoms. In the diabetic this slight fasting acetonuria develops, but this is more than counterbalanced by the drop in toxic acetonuria produced by a diet poor in carbohydrates but rich in fats. It is certainly true that coma occurs much less frequently on this treatment than on the older methods. This plan of re-educating the patient's metabolism from zero, as it were, has yielded in most observers' hands far better results than any previous methods. Nearly 75 per cent. of all cases admitted to hospital are rendered free from sugar for longer or shorter periods, and the average mortality rate is 7.7 per cent., as against the former 23 per cent. (Poulton.) But one has still the unfortunate experience of watching the level of carbohydrate tolerance continue to fall in a good many cases. For, after all, we are not yet attacking the underlying cause, but only the exaggerated metabolism which results from that cause. Not until the sympathetic disturbance can be controlled shall we acquire a mastery over the disease.

EIGHTY-EIGHTH ANNUAL MEETING

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PROCEEDINGS OF SECTIONS.

SECTION OF VENEREAL DISEASES.

Mr. E. B. TURNER, F.R.C.S., President.

DISCUSSION ON

VENEREAL DISEASES IN WOMEN AND CHILDREN,

OPENING PAPERS.

I.—A FEW POINTS IN RELATION TO THE TREATMENT OF VENEREAL DISEASES IN WOMEN,

BY

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I THINK we are all agreed that this is one of the most urgent medical problems of the day. It is concerned with mother and child welfare. It affects the health of the people, the economic standard of the nation, in that every individual worker affected by this disease is economically less efficient in output, whether intellectual or industrial.

The eugenic problem is greater still, but I must confine myself to the particular aspect of the subject allotted to me—namely, the treatment of these diseases in women.

I propose in the short time at my disposal not to touch on the so-called routine treatment of syphilis about which everyone has heard so much, but to deal with special