A CARBOHYDRATE DIET FOR HYPERTHYROIDISM.

BY

HENRY MOLL, M.D., M.R.C.P.,

MEDICAL TUTOR AND REGISTRAR, UNIVERSITY OF LEEDS.

HITHERTO not enough importance has been given to diet in Graves's disease, treatment being focused mainly on the thyroid gland. There are, however, important reciprocal actions between the thyroid gland and the gastro-intestinal tract of which we have little knowledge yet, but which nevertheless demand a closer study of dietary in hyperthyroidism. Thus, various manifestations of nervous dyspepsia are not only common symptoms of hyperthyroidism, but may also precede for a variable time the other cardinal signs. This was borne out by a statistical study of the more important symptoms in a series of sixty-nine cases of Graves's disease, in which the symptoms were tabulated both in order of frequency and time of occurrence.

TABLE I.—Percentage Incidence of Symptoms in Hyperthyroidism.

Palpitation, shortness of breath, and weakness ...	100 per cent.
Mental excitability and loss of weight ...	95 "
Diarrhoea ...	75 "
Swelling of the neck, exophthalmos, and tremor ...	70 "
Indigestion ...	50 "

TABLE II.—Order of Occurrence of Symptoms in Hyperthyroidism.

A.—Early Symptoms:	B.—Later Symptoms:
1. Weakness.	5. Swelling of the neck.
2. Indigestion.	6. Mental excitability.
3. Shortness of breath.	7. Diarrhoea.
4. Palpitation.	8. Exophthalmos and tremor.

From these tables it will be seen that indigestion—and under this term are included nausea, vomiting, anorexia, gastric discomfort, and sometimes pain—not only occurs in half the cases of Graves's disease, but, when present, is usually a very early symptom, and accompanies other prodromal signs of hyperthyroidism such as loss of weight, weakness, shortness of breath, and palpitation. Moreover, in 50 cases of hyperthyroidism in which fractional gastric analyses were carried out it was found that gastric motility and secretion become profoundly modified in exophthalmic goitre.¹

The rate of gastric emptying is exceedingly rapid, often so rapid that very little is to be found in the stomach at the end of an hour, the average emptying rate being fifty-five minutes in cases of achlorhydria and ninety minutes in cases of hypochlorhydria. This rapid emptying of the stomach may be largely explained by the achylia so commonly found in Graves's disease, and which was actually present in 22 cases out of 50 examined. The hurrying of food which takes place in hyperthyroidism, together with the lack of secretion, must to a certain extent prevent digestion occurring in the stomach, and may afford an explanation of the frequency of digestive troubles in exophthalmic goitre. Some generalities may therefore be drawn from this with regard to dietary.

As the intestines probably have to act vicariously and digest food for the stomach, it is important to arrange the diet so that it can easily be acted on by the intestinal juices. Proteins, being largely digested by the stomach, should be given only in easily digestible forms—for example, brains, scraped beef, sweetbreads, chicken, and fish—and their amount reduced to a minimum, as they can be positively harmful by stimulating physical and mental activities and thereby increasing the strain on the thyroid gland.

The food must be broken up in fine particles and given as far as possible in liquid form. Six to seven small meals should be given at intervals of two to three hours rather than three full meals in the course of the day. The caloric value of the diet should be high, with a tendency towards supralimentation in order to keep pace with the raised metabolic rate.

Amongst the protein spacers to be substituted in the diet, fats are unsuitable. Hyperthyroid patients seldom tolerate fats and these should be given in the more digestible forms, such as the artificial fatty foods. Carbohydrates, on the contrary, may be freely given, as they are, as a rule, well tolerated by patients suffering from hyperthyroidism, and may be given in sufficiently large amount to raise the caloric value of the diet to a high level. As a matter of fact I have found carbohydrates act so favourably in hyperthyroidism that the following dietary containing a large excess of carbohydrates was adopted in conjunction with other forms of medical treatment of Graves's disease. The diet is of a caloric value of 2,700 calories, and contains roughly 80 grams of protein, 60 grams of fat, and 440 grams of carbohydrates.

Diet Sheet of a Full Carbohydrate Diet for the Treatment of Graves's Disease.

6 a.m. (Breakfast).—Oatmeal porridge 4 oz.; tea with cream; bread, two slices; butter 1/4 oz.; honey or jam 1/2 oz.; fruit 4 to 6 oz. (bananas, grapes, plums, prunes, apples, pears, cherries, apricots, oranges).

8 a.m.—Proprietary food 6 oz. (Benger's food, Mellin's food, Horlick's malted milk, bananina, Robinson's groats or patent barley, etc.); bread, two slices.

10 a.m.—Milk 8 oz. with Mead's dextrimaltose.

12 a.m. (Lunch).—Meat 2 oz. (scraped beef, brains, sweetbreads, chicken, pigeon, rabbit); or fish 2½ oz. (cod, haddock, whiting, hake, plaice, sole). Vegetables—Group 1, 4 to 6 oz. (potatoes, artichokes, parsnips, peas, carrots, onions, beetroots, turnips); Group 2, 3 oz. (broccoli, brussels sprouts, cabbage, cauliflower, leeks, spinach, tomatoes, celery, lettuce). Bread, two slices; milk 6 oz.; milk pudding 4 oz. (rice, arrowroot, cornflour, sago, tapioca).

3.30 p.m. (Tea).—Same as breakfast, but without porridge.

6 p.m. (Supper).—Proprietary food 6 oz.; bread, two slices; one egg.

10 p.m.—Milk 8 oz. with malt extract or milk sugar.

Extras.—Cream up to half a pint, or fatty artificial foods (cod-liver oil, cremalto, etc.), 1 oz. thrice daily.

Fats in the form of cod-liver oil and cream are added later as extras, thus raising the caloric value of the diet to over 3,000 calories, while a liberal amount of dilute hydrochloric acid should be given in the form of a long drink to be sipped during mealtime to counteract the achylia so often present in exophthalmic goitre. Patients take the diet particularly well, and, barring cases in which it may cause or aggravate a flatulent dyspepsia, can usually be kept on it for quite a considerable time without tiring of it. Often the appetite improves remarkably.

In watching the progress of the case, the extreme variability of the appetite so often present in Graves's disease has to be taken into account, as periods of anorexia alternate with periods of hunger, vacillating with the emotional instability. The improvement is shown by the lessening of the nervous symptoms, by the fall of the pulse rate and of the pulse pressure, and by the rapid increase of weight. Some patients gain as much as 10 lb. in a fortnight. The patients should be kept on the diet for several months and afterwards revert to a normal diet by degrees, first by the addition of fats and then of proteins.

It may be objected that it is unsafe to give a full carbohydrate diet in a disease where there is often a lowered sugar tolerance. This is often revealed by the response which is observed in exophthalmic goitre to a sugar tolerance test, although there may be only a latent defect of sugar storage without glycosuria. Thus, in three cases of hyperthyroidism, taken at random, but sugar-free on a basal diet, one gave a normal response to a glucose tolerance test (50 grams), another showed a lag type curve, and the third had a high threshold and a high rising curve typical of diabetes mellitus. Apart from these cases, in which the disturbance of carbohydrate metabolism is only detected by a sugar tolerance test, there are other cases of hyperthyroidism with glycosuria of varying degrees, from severe forms with marked denutrition and ketosis to milder ones of intermittent glycosuria. In the light of these findings a full carbohydrate diet would at first appear to be inadvisable in Graves's disease. However, glycosuria very rarely occurs after a carbohydrate excess diet, and when it does the glycosuria very often soon disappears after pushing the diet still further.

While certain cases of hyperthyroidism with glycosuria have to be treated on true diabetic lines, others can be

given starchy food with impunity, and it is remarkable how glycosuria rapidly disappears on full carbohydrate diet, as is shown by the following case.

A woman, aged 22, had had an enlarged thyroid gland since the age of 15, but without symptoms of hyperthyroidism. Six months ago the thyroid gland increased in size, and for the first time signs of hyperthyroidism made their appearance—namely, breathlessness, sweating, exophthalmos, and tremor. Examination revealed a large adenomatous thyroid gland, ocular signs, exophthalmos, tremor, and a high pulse pressure. At this time there was no glycosuria. The patient while in hospital developed diphtheria and had to be removed to a fever hospital. On her return a month later the general symptoms of hyperthyroidism seemed somewhat intensified and glycosuria had appeared. Thereupon a glucose tolerance test (50 grams) was made and showed a lowered tolerance for sugar, the values being as follows: resting blood sugar normal, half an hour after glucose administration 0.18 per cent.; one hour later 0.20 per cent.; 1½ hours later 0.22 per cent.; two hours later 0.135 per cent. A trace of sugar (0.5 per cent. approximately) appeared after the second hour.

The patient was put on a full carbohydrate diet and the glycosuria disappeared thereafter.

This case is of particular interest because it illustrates two facts not infrequently found in Graves's disease—namely, that glycosuria made its first appearance with the intensification of hyperthyroid symptoms, following an acute throat infection; and, in the second place, that it disappeared on giving carbohydrates freely.

It is difficult to give a satisfactory explanation of the paradoxical response of thyroid glycosuria to a carbohydrate diet. That the mechanism of thyroid glycosuria is more complex than we imagine is revealed by the very contradictory evidence of the effects of feeding animals with thyroid extract. I have failed to produce glycosuria in four dogs after administering thyroid extract (Burroughs and Wellcome) equivalent to one gram of fresh gland per kilo of body weight daily for over a month. Cramer,² Kuriyama,³ and Burn and Marks⁴ have also reported similar failures to produce glycosuria by thyroid feeding. Cramer, however, has observed that experimental hyperthyroidism produced by thyroid feeding leads to a complete disappearance of glycogen from the liver, and Burn and Marks have made the interesting observation that this disappearance of glycogen only occurred at a later stage when thyroid feeding had been continued for some time. These experiments suggest that the action of the thyroid hormone is at first to stimulate the glycogenic function of the liver, and that later one may reach a stage in which this function may become exhausted. If such be the case it is reasonable to suppose that a diet rich in carbohydrates may compensate a defective glycogenic function of the liver, dependent on an excessive thyroid action, by reducing to a minimum the stimulating effect of proteins on the thyroid gland.

A similar explanation may be extended to the effect of a carbohydrate diet on hyperthyroidism generally, thus supporting the view that careful dieting should play an important part in the treatment of exophthalmic goitre, whether surgical or medical, and that carbohydrates should be freely given, as they appear to have a soothing action on the thyroid gland.

Summary.

1. Attention is drawn to the importance of careful dietetic treatment in hyperthyroidism, from the following observations: (a) in a study of 69 cases of hyperthyroidism it was found that dyspepsia was both a common and an early symptom in hyperthyroidism; (b) in fractional gastric analyses made on 50 cases of Graves's disease rapid emptying of the stomach and achylia or hypochlorhydria were an almost constant feature.

2. A diet rich in carbohydrates is advocated in the treatment of exophthalmic goitre.

3. Mention is made of the paradoxical response of thyroid glycosuria to carbohydrates.

I have to thank Professor Telling for affording me facilities for carrying out these observations on patients under his care.

REFERENCES.

¹ Moll, H., and Scott, R. A. M.: *Lancet*, 1927, i, 68. ² Cramer, W.: *Brit. Journ. Exper. Path.*, 1924, v, 128. ³ Kuriyama, S.: *Amer. Journ. Physiol.*, 1917, 43, 481. ⁴ Burn, J. H., and Marks, H.P.: *Journ. Physiol.*, 1925, 60, 131.

JAUNDICE CAUSED BY LARGE EXTRAVASATIONS OF BLOOD.

BY

C. E. NEWMAN, M.D.CANTAB., M.R.C.P.LOND.,
MEDICAL REGISTRAR, KING'S COLLEGE HOSPITAL.

IN spite of the general trend of recent work on the pathology of jaundice there are still many who feel that a definite clinical jaundice is impossible without the intervention of the liver. The study of fluids aspirated from haematomata has for some time suggested its possibility, and the two cases I propose to describe seem to put the matter almost beyond question.

Haematoma Fluids.

When blood is extravasated into the tissues it is converted into a thick brown mucoid fluid, whose pigments consist of unchanged blood pigments and haematoidin, a substance which has the same composition as bilirubin, and which reacts to van den Bergh's test in the same manner as "delayed reaction" bilirubin. The direct van den Bergh test is often difficult, because the mucoid fluid may be impossible to separate by spinning, but the quantitative estimation is simple, as the alcohol used to precipitate the serum protein carries down the haemoglobin at the same time, leaving a clear extract containing the bilirubin. The following table gives the results of the van den Bergh test on several haematoma fluids.

No.	Source of Haematoma.	Time since Extravasation.	Direct Test.	Quant. (Units).	Notes.
1	Bruise of thigh	14 days	—	4.0	
2	Breast operation	27 hours	Delayed	2.5	
3	Nej hrectomy	9 days	Delayed	12.0	
4	Breast operation	5 days	—	5.0	
5	Breast operation	12 days	Delayed	4.0	
6	Breast operation	2 days	Delayed	2.0	
7	Bruise	3 hours	Delayed	0.2	Unaltered blood.
8	Pleural effusion	5 days	Delayed	0.75	Blood introduced by previous aspiration.
9	Cyst of breast	3 days	Delayed	3.0	After previous aspiration.
10	Traumatic hydrocele	4 months	Delayed	1.2	
11	Haemothorax	20 days	Delayed	4.2	
12	Haematoma auris	6 days	Delayed	4.2	
		5 days later	Delayed	4.5	Had filled up again.

None of these patients was jaundiced or passed bile in the urine. The blood bilirubin was estimated in Cases 1, 3, 8, and 12, and was found to be normal. It appears that in between three and twenty-four hours the local reticulo-endothelial cells start to convert the haemoglobin into "delayed reaction" bilirubin, and that thereafter the fluid contains a higher concentration of bilirubin than the circulating blood. It does not seem likely that the liver plays any part in this pigment formation. Many other investigators have already arrived at these conclusions.

Jaundice from Haematoma.

CASE 1.

A male, aged 62, had a large hypernephroma removed through a lumbo-dorsal incision on February 10th, 1925. The wound was closed without drainage. The anaesthetic used was ether. On February 12th a good deal of bruising was visible round the wound, and on the 14th the conjunctivae were yellow, with a great deal of urobilin in the urine, but no bile. Next day the patient was jaundiced all over and there was the most intense local jaundice round the wound. The van den Bergh test on February 16th, on the blood, gave a delayed reaction (after half an hour) with a quantitative figure of 2.7 units. The levulose tolerance test gave the following result:

Fasting blood sugar	...	0.103 per cent.
50 grams of levulose taken fasting.	...	
Half an hour after	...	0.122 "
One hour after	...	0.147 "
One and a half hours after	...	0.137 "
Two hours after	...	0.116 "