REFERENCES

- Banks, B. M. (1941). *New Engl. J. Med.*, **225**, 433.
 Clark, N. S. (1946). *Arch. Dis. Childh.*, **21**, 160.
 Deák, E. (1931). *Virchows Arch.*, **282**, 895.
 Friedmann, I., and Por, F. (1947). *British Medical Journal*, **2**, 494.
 Fox, R. A. (1943). *Arch. Path.*, **36**, 311.
 Hepp, P. (1887). *Berl. klin. Wschr.*, **24**, 297, 322.
 Karelitz, S., and Welt, S. K. (1932). *Amer. J. Dis. Child.*, **43**, 1134.
 Nellen, M. (1947). *Lancet*, **1**, 326.
 Ophüls, W. (1923). *Arch. intern. Med.*, **32**, 870.
 Rich, A. R. (1946). *Rev. méd. Argent.*, **60**, 816.
 Unverricht, H. (1887). *Z. klin. Med.*, **12**, 533.
 Wagner, E. (1887). *Dtsch. Arch. klin. Med.*, **40**, 241.

POST-PRANDIAL SYMPTOMS FOLLOWING PARTIAL GASTRECTOMY

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While the results following subtotal gastrectomy for ulcer are generally excellent, in a small proportion of cases there develop post-prandial symptoms which may persist for many months. Such post-prandial upsets after gastrectomy have been described in British, German, and American literature, and frequent attempts have been made to relate these symptoms to changes in sugar levels in the blood. Lawrence (1936) noticed that after such surgical procedures patients developed hyperglycaemic then hypoglycaemic sugar levels following ingestion of glucose. Evensen (1942) found that 34 out of 95 cases treated by partial gastrectomy developed unusually low blood-sugar levels after meals. Adlersberg and Hammerschlag (1947) divided such upsets into two groups—early and late. The early symptoms (sense of pressure and fullness in the epigastrium) they attributed to the rapid emptying from the stomach remnant. Later symptoms (palpitations, headache, dizziness, and perspiration) they believed to be due to chemical factors, especially hypoglycaemia. Gilbert and Dunlop (1947) believed the symptoms were due to hypoglycaemia, and they found that ephedrine administration lessened the upset. They suggested that the development of insulin sensitivity was a causal factor.

Present Investigation

Twenty-four cases were studied—23 men and one woman. All had had subtotal gastrectomy for duodenal or simple gastric ulcer. The type of operation performed in every case was a gastrectomy, removing three-quarters of the stomach and the first inch of the duodenum, with an antecolic anastomosis. No valve mechanism was formed.

The Nature of the Symptoms.—In mild cases (the majority) the patients complain of a heaviness and fullness in the epigastrium, accompanied by a curious sense of muscular or body fatigue, and want to sit down; in more severe cases they may have to lie down and may fall asleep. A flushing of the skin may occur and the patient often "feels hot" and may actively perspire. Giddiness and palpitations often occur. Such symptoms were present in all 24 cases. To this list of symptoms must be added bilious vomiting, which occurred in two of the cases. These two patients had all the other symptoms, but in addition after meals they felt very upset and wanted to vomit. They usually vomited about 30 to 45 minutes after meals, and thereafter felt very well and were able to go about their business. The vomited material consisted either of food heavily bile-stained or, as often, of 1½ pints (0.85 litre) of unmixed pure bile.

Time Interval between Operation and Symptoms.—In every case the symptoms started either while the patient was in hospital or as soon as he went home and began to

live a normal life. In no case was there a time interval of some weeks, such as has been described by those who believe the condition to be due to the development of insulin sensitivity.

Time of Onset of Symptoms in Relation to Meals.—In each case the symptoms started almost immediately after meals or, indeed, while the patient was still eating. The upset usually lasted 20 to 30 minutes. The rapidity of onset suggested a mechanical causation, and in nearly every case the symptoms could be brought on rapidly by eating more quickly, and by giving a larger meal the symptoms usually increased in severity. In no case did a group of late symptoms appear 1 to 1½ hours after meals.

Type of Meal which Caused the Symptoms

In all 24 cases a careful study was made of the type of food and the amount taken at each meal in the day, and a rough measure of the total bulk of each meal and its total carbohydrate content was made. In 23 cases the symptoms appeared only after "the big meal"—i.e., the most bulky meal of the day. In almost all of these cases the carbohydrate content of "the big meal" was considerably less than the carbohydrate content of another meal in the same day which produced no symptoms. Indeed, the patients themselves in most cases clearly realized that it was the bulkiness of the meal which troubled them, and many noted that by missing the soup one day, and doing without pudding the next, they could avoid such symptoms altogether. In other words, the incidence of symptoms depended on the total amount of food eaten and not on its carbohydrate content.

In order to clarify still further the question whether the symptoms were due to bulk or to carbohydrate content each patient was given 50 g. of glucose in water and the severity of the symptoms and their time of onset, etc., were recorded; the blood-sugar levels were estimated by MacLean's method. On the following day the patient was given a bulk meal containing scarcely any carbohydrate (about 6 g.) and the severity and time of symptoms were again recorded. The blood-sugar levels were again estimated to check the fact that there was no rise in the blood sugar. In 16 of the 24 cases the symptoms were more pronounced after this bulk meal than they were after the ingestion of 50 g. of glucose in water. In four cases the severity of the upset was equal, and in four cases the 50 g. of glucose caused more upset than the bulk meal. In short, it was again shown that it was the bulk and not the carbohydrate content of the meal which mattered. It is, however, interesting to note that after these bulk meals the blood-sugar level rose to a moderate extent in most cases. Indeed, blood-sugar levels were recorded which were higher than one would expect after a meal of such small carbohydrate content. It will be suggested that this may be due to stimulation of the sympathetic system caused by distension of the gut, bringing about an adrenaline effect.

Relation of Symptoms to Blood-sugar Level

It has already been stated that each patient had blood-sugar estimations done after the ingestion of 50 g. of glucose in water, and the time during which the symptoms were present was carefully noted. The results are shown in the accompanying Table. By graphing the blood-sugar levels and marking on the graph the period of upset the following points were noted.

The symptoms appeared long before the sugar level in the blood had reached its peak. The symptoms were usually over before the blood-sugar level started to fall or before it fell below normal limits. In short, the symptoms appeared during the period of hyperglycaemia and the symptoms had

Blood-Sugar Levels after 50 g. of Glucose by Mouth
(The Readings in bold type occurred during the symptoms)

Case No.	Blood-Sugar Readings in mg. per 100 ml.								
	Before Meal	5 Min. After	10 Min. After	1 Hr. After	1 Hr. After	1 Hr. After	1 Hr. After	1½ Hr. After	1½ Hr. After
1	88	95	115	130	173	199	106	104	112
2	100	110	110	175	286	156	100	88	98
3	89	128	136	150	248	187	123	100	80
4	103	166	180	200	282	120	80	93	86
5	120	100	160	179	248	147	70	90	89
6	86	90	100	121	126	130	196	58	82
7	48	50	70	92	116	50	62	186	64
8	90	100	116	136	210	256	198	110	90
9	100	106	130	172	211	190	100	90	86
10	102	90	112	156	191	148	80	100	92
11	86	92	126	180	243	110	120	97	106
12	108	102	130	162	224	160	111	126	108
13	117	127	142	155	198	133	60	50	46
14	112	115	140	182	200	180	158	138	112
15	84	92	100	154	186	64	100	103	107
16	78	84	116	162	171	100	87	103	82
17	104	100	115	158	180	82	106	104	109
18	68	71	86	173	210	70	162	58	89
19	96	102	120	186	80	112	80	94	90
20	106	100	141	195	166	84	92	110	96
21	86	92	112	138	173	184	101	106	100
22	105	116	165	179	267	143	70	83	66
23	95	106	173	182	226	61	102	98	93
24	80	109	127	158	194	69	105	98	86

passed off before low levels of blood sugar were recorded. Furthermore, exactly the same symptoms were produced, often of greater intensity, with the bulk meals, where the blood-sugar levels showed a less marked rise and fall. An illustrative case, with typical blood-sugar curves, is noted below.

A man, aged 40, had a subtotal gastrectomy for duodenal ulcer 18 months ago. Since operation he has been complaining of fullness in the epigastrium for 20 minutes after meals, with weakness and sweating. Palpitations are common and giddiness is often present. This comes on after his dinner at midday, which usually consists of soup, then fish or meat. If he takes any pudding of any sort his symptoms are much worse.

Curve A shows blood-sugar levels after 50 g. of glucose by mouth. Symptoms were slight. Curve B shows blood-sugar levels after a bulk meal with almost no carbohydrate. Symptoms were pronounced after this meal. The period of symptoms is shaded. During this period blood-sugar estimations were made every five minutes.

Discussion

Post-prandial symptoms following partial gastrectomy are not likely to be due to hypoglycaemia. In all 24 cases they occurred in the hyperglycaemic stage. The symptoms were quite often over before the sugar level stopped rising. Even when hypoglycaemic curves were produced the symptoms did not coincide with that period, and a similar set of symptoms of at least equal intensity could be produced in the same patient with a bulk meal with a much smaller rise and fall in the sugar levels in the blood.

That these symptoms are due to mechanical distension of the gut is suggested by the following facts: (a) The rapidity

of onset of symptoms following the meal suggests a mechanical cause. (b) It was the major meal of the day which gave the trouble in 23 cases, and patients usually knew that they could avoid or lessen the upset by taking fewer courses. As has been said, this major meal in most cases had a smaller carbohydrate content than at least one other meal of the day which produced no symptoms. (c) In two-thirds of the cases the bulky low-carbohydrate meal produced more symptomatic upset, with only a moderate change in blood-sugar level, than did the 50 g. of glucose which caused a marked rise and fall in the sugar levels. Further, the very fact that the symptoms were the same in both cases suggests a mechanical basis.

It should be added that in the four cases in which the 50 g. of glucose caused more upset than the bulk meals no satisfactory explanation was found. These patients had no marked hypoglycaemia, and again the symptoms occurred in the hyperglycaemic stage. These four patients had insulin-sensitivity tests carried out. No hypersensitivity was found.

If the post-prandial upset is due to mechanical distension of the gut it is likely that the afferent pathway for this is in the sympathetic-nerve fibres and not the vagi. This is suggested by the work of Andrus (1947), who performed a preliminary gastrectomy on a patient with carcinoma of the oesophagus. At a later operation he did a bilateral vagotomy. By passing a cystoscope through the gastrectomy tube he was able to test the stomach wall for different types of sensation before and after vagotomy. He observed that most sensations, such as feelings of distension, heat and cold, pain, etc., were unaffected by vagotomy. Moore *et al.* (1947) also observed such post-prandial symptoms following vagotomy.

Finally, the symptoms themselves suggest stimulation of the sympathetic system. The weakness, giddiness, perspiration, etc., are not unlike the effects produced by adrenaline and ephedrine. In this respect it is interesting to note that the pulse rate increased by 10 to 20 beats a minute in all 24 cases during the upset. Furthermore, the rise in the blood-sugar level after a meal containing very little carbohydrate might be explained by an adrenaline effect following stimulation of the sympathetic nerve by mechanical distension of the gut.

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REFERENCES

- Adlersberg, D., and Hammerschlag, E. (1947). *Surgery*, **21**, 720.
- Andrus, W. de W. (1947). *J. Amer. med. Ass.*, **133**, 748.
- Evensen, O. K. (1942). *Acta med. scand.*, Supp. 126.
- Gilbert, J. A. L., and Dunlop, D. M. (1947). *British Medical Journal*, **2**, 330.
- Lawrence, R. D. (1936). *Ibid.*, **1**, 526.
- Moore, F. D., *et al.* (1947). *J. Amer. med. Ass.*, **133**, 741.

In Circular 150/48 (dated Aug. 20, 1948) the Ministry of Health has given some advice to County and County Borough Councils about their functions under the National Assistance Act, 1948. There are not to be any changes in the system of registration of blind persons or of the arrangements under which the Regional Associations for the Welfare of the Blind maintain regional registers. The certification of blindness will be part of the local authority's duty under the National Assistance Act and not under the National Health Service Act, and the Form B.D.8 will continue to be used. Local authorities are to pay the regional boards for certificates provided by specialists employed by the board, but where the specialist does the work outside his employment by the board the local authority will pay him direct. In addition to persons registered as blind within the meaning of Section 64 of the National Assistance Act, local authorities are now urged to keep an "observation register" of those who are substantially and permanently handicapped by defective vision ("partially sighted persons"). Local authorities should keep these patients under review and make sure that they are obtaining the treatment they may need through the National Health Service.