aggravation of catarrhal illness. A consideration of some of the results which may follow the habitual absorption of an excess of glucose into the blood seems justifiable, even though at present it is largely speculative.

EFFECTS UPON THE PANCREAS

Habitual excessive absorption of glucose must be met by habitual hyperinsulinism.⁸

(a) In these circumstances any sudden reduction in glucose absorption (as, for example, at the onset of an acute febrile illness) will result in a comparative hypoglycaemia, with the production of diacetic and oxybutyric acids from incomplete combustion of fats. May not the habitual over-consumption of carbohydrates be responsible for the increasing frequency of acidosis among modern children?

(b) The undoubted benefit which many modern children derive from glucose feeding can only be explained by some degree of hypoglycaemia, indicating (since in starvation hypoglycaemia does not occur) over-activity of the islets of Langerhans.

(c) Many cases of severe spontaneous hypoglycaemia are now being reported in adults. In some of these the symptoms, which were temporarily relieved by glucose or adrenaline administration, have disappeared when a partial pancreatectomy was performed.

(d) Actual enlargement of the islets has⁹ been demonstrated in many cases.

(e) Since over-activity of an organ is apt to be followed by failure of function, and hypertrophy by atrophy, the increasing frequency of diabetes may be subject to a similar explanation. Each of these states may be traced to over-stimulation of the islets of Langerhans as a result of habitual excessive glucose absorption.

EFFECTS UPON THE SUPRARENAL AND PITUITARY SECRETIONS

(a) The chief physiological antagonist of insulin is suprarenal secretion. Habitual hyperinsulinism will thus be associated with habitual hyperadrenalism. A second antagonist is pituitary secretion. In addition to their actions upon blood sugar both of these secretions have pressor effects. May not the increasing frequency of hyperpiesia be attributable to over-activity of these glands?

(b) Adrenaline encourages the production of abnormal lipids from cholesterol. Recent work indicates that the earliest change in arteriosclerosis is a deposit of lipoids in the vessel walls. This suggests that arteriosclerosis, too, may have some relation to excessive carbohydrate ingestion.

EFFECTS ON FAT STORAGE

Apart from endocrine disturbances, we know that when glucose is absorbed in excess of the normal capacity of the liver and muscle to store glycogen, that excess is deposited as fat. In America, where the consumption of sugar (112 lb. per head per annum several years ago) is considerably higher than our own, the increasing frequency of obesity is causing alarm.

Thus there is a group of diseased conditions—obesity, arteriosclerosis, and diabetes—which have long been associated clinically, and which are increasing in modern times, although the general death rate is falling. Is it not possible that this increase is directly associated with excessive carbohydrate intake, which is largely, if not entirely, due to the prodigious increase in the use of sugar?

EFFECTS ON DIGESTION

A further result of excessive ingestion of carbohydrate food is the occurrence of indigestion. Not only may those processes fail which are concerned with the conversion of

starch into glucose and sucrose into invert sugar, but, when active, they may give rise to abnormal fermentative reactions in the intestine. Substances are thus produced and absorbed which are capable of toxic effects such as the capillary disturbance described by Maitland Ramsay. If the failure of digestion is sufficiently severe only a fraction of the ingested carbohydrate becomes available for absorption, and in the midst of plenty there is famine. In such cases, as the following will show, the patients, though large eaters, are undernourished.

The patient was a girl, aged 15, listless, pale with a muddy complexion, and a frequent sufferer from febrile catarrhal attacks. She was thin, and failed to gain weight though a heavy eater, especially of starches and sweets. In 1931 she developed renal glycosuria. As this persisted to an unusual extent she was seen by a specialist, who greatly reduced her starchy intake and prohibited sugar. He warned me that the amount of carbohydrate he had permitted was probably quite insufficient for her school activities, and that the latter would have to be restricted to prevent loss of weight. On the altered diet, however, she began to gain weight rapidly, became more alert and active, and was able to play all games. Her appearance and complexion rapidly improved, and in the last year she has only suffered from one slight cold and has been free of glycosuria. As I interpret it, she had suffered from carbohydrate indigestion with its accompanying toxæmia, and, when her digestion improved as a result of carbohydrate restriction, her malnutrition disappeared.

CONCLUSION

I submit that our carbohydrate intake is excessive, and that such excess is mainly due to the prodigious increase in the consumption of sugar in the last half-century. Clinical data have been advanced with regard to catarrhal illness, but I must leave it to others to supply them for or against the association between sugar consumption and the diabetic group of diseases referred to. It is from the general practitioners that such data must be obtained.

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PATHOLOGICAL CHANGES IN ACUTE PANCREATITIS SEEN DURING LIFE

BY

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The following three cases of acute pancreatitis are described because the appearances in the abdomen seen at operation differed from those which have so far been seen at necropsy in the Bernhard Baron Institute of the London Hospital, and also from any given in the literature to which I have had access.

CASE I

Female, aged 52. Complete removal of right breast for carcinoma five years before. For three weeks before admission she had been suffering from vague indigestion and flatulence, and had noticed that the stools were pale and bulky. Two days before admission she was seized with violent upper abdominal pain, referred to the back and to the left shoulder, which caused her to collapse. She vomited twice at the onset. On admission she appeared to be very ill.

Temperature 101° F., pulse 140, and respiration 32. The tongue was dry and furred. The abdomen was held rigid, with no respiratory movement whatsoever. On palpation there was board-like rigidity of the upper abdomen, with great tenderness. Palpation revealed slight increase of liver dullness. The diagnosis of a posterior perforation of a gastric ulcer into the lesser sac was made, and immediate laparotomy was decided upon. Upper abdominal section was performed, and the peritoneal cavity was found to contain a moderate amount of clear blood-stained fluid, some of which was collected for pathological examination. The gall-bladder was normal, but emptied slowly, and there was no ulcer on the stomach or duodenum. The great omentum and transverse mesocolon contained large distended areas of an opaque, greyish, jelly-like material, and the peritoneum of the posterior abdominal wall was bulged forward with a large collection of a similar mucinoid substance. The pancreas was greatly enlarged and oedematous, and was entirely surrounded by this jelly-like substance. The gland itself gave a peculiar granular and at the same time "soggy" feeling on palpation. Two small areas of fat necrosis were found, one in the gastro-hepatic omentum and the other in the great omentum. No drainage was performed, and the patient was sewn up and put back to bed. The following day the urine showed 200 units of diastase. Within a week the diastatic index had dropped to 100, and on discharge on the seventeenth day it had dropped to 30. Recovery was completely uneventful.

CASE II

Female, aged 36. For three weeks she had been suffering from pain in the epigastrium referred to the back and to the point of the right shoulder, with frequent attacks of vomiting. On the day of admission she had a very severe attack of pain similar in situation, but enough to cause collapse. Her temperature was 98°, pulse 120, and respiration 32. There was no jaundice. The abdomen exhibited extreme tenderness and rigidity all over, most marked in the hypogastrium and right hypochondrium. The liver dullness was normal. At operation there was a very grossly enlarged gall-bladder containing a number of calculi. The omentum exhibited areas of distension with a greyish mucinoid material, which separated the layers into large blebs. The peritoneum of the posterior abdominal wall was similarly affected, and the pancreas lay in a mass of the same jelly-like material. There were no areas of fat necrosis. The gall-bladder was emptied of stones and muco-purulent fluid, and drained. The common duct was patent. The lesser sac was drained by a large tube passing through the transverse mesocolon, and the abdomen was closed. The tube in the lesser sac was removed on the third day as no discharge was being evacuated through it. The gall-bladder tube was removed on the sixth day. Recovery was uneventful, the patient being discharged well on the fifteenth day. Unfortunately the urine diastase was not estimated.

CASE III

Male, aged 54. For one month he had been suffering from vague dyspepsia, with pain in the abdomen referred through to the back. Two days before admission the pain had become much more severe, and was accompanied by vomiting. On the day of admission all the symptoms were increased, and there was slight jaundice present. The patient's temperature was 99.6°, pulse 130, and respiration 40. Abdominal respiration was absent. The abdomen evinced board-like rigidity in the upper half, with marked tenderness all over. On laparotomy the gall-bladder was found to be distended, and there were several small calculi present. The common duct was dilated, and one small stone, with a large amount of biliary mud, was found. As in the two other cases the great omentum was distended into blebs with the greyish mucinoid material. The pancreas itself was surrounded by similar material, and the peritoneum of the posterior abdominal wall was also raised by a collection of the jelly-like material behind it. There were three small areas of fat necrosis. The gall-bladder was drained, the common duct opened and cleared, and also drained. No drainage of the lesser sac was carried out. The diastatic index in this case was 180, which fell in eight days to normal. Recovery was uneventful, the patient being discharged on the sixteenth day.

REMARKS

Only two small fat necroses of typical appearance were seen in Case I, and three in Case II. In all three cases the retroperitoneal tissue round the pancreas, in the great omentum, and upon the posterior abdominal wall showed large, focal, or diffuse expansions occupied by an opaque, greyish jelly-like substance. In Case I the mesocolon was similarly affected, whilst the pancreas was greatly swollen and oedematous. Professor Turnbull tells me that he and his colleagues have not observed this gelatinous substance at necropsy, but have found in similar positions masses of an opaque pultaceous or putty-like substance containing fibres, suggesting a soft plaster mixed with hair. The colour has been in general white, yellow, or grey, but to a greater or less extent dirty grey, brown, black, or brown-red from altered blood. This putty-like substance containing fibres is obviously digested adipose tissue in which the digestion of the fibrous stroma is incomplete. This substance found at necropsy might be described as butter, but not as jelly. It is possible that the appearances shown during life alter after death. This, however, appears to be an unlikely explanation, because Professor Turnbull remembers cases in which masses of pultaceous substance were removed from the lesser sac during dressings after laparotomy. It is more probable that the gelatinous condition seen in these three cases during life represents a preliminary stage in digestion. Although the symptoms had come on acutely, and there was severe interference with pancreatic function, all three patients recovered. It is impossible, therefore, to know whether complete digestion ever occurred.

My thanks are due to Professor H. M. Turnbull for his kind help with the post-mortem description of this condition, and to Mr. Russell Howard for permission to publish the details of these cases.

TOTAL HYSTERECTOMY FOR NON-MALIGNANT CONDITIONS

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The very valuable paper on total abdominal hysterectomy for myoma of the uterus by Dr. Herbert Spencer in the *British Medical Journal* of June 25th, 1932, prompts me to make an addition to it which I consider of importance. No one has done so much as Dr. Spencer in calling attention to the real danger of cancer attacking the cervix which is left after subtotal hysterectomy. In the *Medical Journal of Australia* of June 23rd, 1917, I published a short series of such cases, and have known of many others. I think it is generally admitted all over the world that 2 per cent. would be a conservative estimate of such untoward happenings. Notwithstanding Dr. Herbert Spencer's irrefutable arguments in favour of total hysterectomy, I am convinced that it will never be as popular as the subtotal method until the Doyen technique (which I have seen both Doyen and Dr. Spencer perform, and which Dr. Spencer still advocates) gives way to one infinitely simpler, safer, and with much better ultimate results. Such a method I published in the *American Journal of Obstetrics and Gynecology* for December, 1917.

My first operation by this method was performed on June 14th, 1914, and my 532nd on July 29th, 1932. There have been three deaths—two due to accidents from the disorganization of the Sydney Hospital staff during the war. The third was due to acidosis, in a drug addict. This patient lived out of the city, and her case was