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Mechanism and Management of the Dumping Syndrome

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MECHANISM AND MANAGEMENT OF THE DUMPING SYNDROME*

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An understanding of the behavior and misbehavior of an abbreviated stomach remnant following partial gastrectomy, the commonest basic procedure employed in the surgical treatment of peptic ulcer disease, is predicated on an appreciation of the physiology of the normal, whole stomach. In oversimplification, the stomach's job may be considered as 3 tasks: a) to act as a receptacle for the ingesta, as a reservoir for the food consumed at a given meal and delivered by the esophagus; b) to initiate digestion of this food within its own lumen and, just as important, to set in motion the digestive processes further in the alimentary canal; and c) to deliver the chyme to the duodenum in the proper amount, at the proper rate, and in suitable consistency. All or part of these important functions are destroyed or altered by the surgeon's knife as he transects the stomach.

Shorn of its greater distal part, the stomach remnant can serve as a very limited receptacle, if as a reservoir at all. It has been deprived of much of its acid bearing mucosa and of the inherent mechanism for stimulating those parietal cells which remain. As the acid activator goes, so goes the pepsin. There remains practically no intralumenal digestion. In the absence of acid (which state of anacidity was the purpose of the operation), the pancreas, liver, gall bladder, and small intestine are hampered in initiating their own important digestive functions. Finally, the material presented by the stomach remnant to the small intestine bears little resemblance to chyme in its consistency, its osmotic concentration, or in its volume or rate of delivery. Viewed in the light of these observations, it is remarkable that the gastrectomized patient exists, indeed often thrives, as well as he usually does.

In terms of the experience of this clinic, the statistics pertaining to which represent a respectable follow-up of almost 5 years, 84.8 per cent of gastrectomized patients were considered† to have emerged from partial gastric resection with good or excellent results as defined by (a) freedom from all gastrointestinal symptoms, (b) subjective well-being, and (c) return to gainful employment or usual activity. A minority (15.2 per cent) have failed to fulfill these criteria. Of these, the greater number consider themselves well by their own estimate and almost all have returned to work. Only 3.8 per cent are classified as "poor" results.

In the last group, 3 patients (1.9 per cent) have exhibited proven or presumed recurrent peptic ulceration. From the "fair" and "poor" categories, 9 have lost an individual average of 22.4 pounds. At the other end of the scale, even in this group, 7 have gained an individual average of 19.9 pounds.

Untoward Post-Prandial Symptoms. In former years, disagreeable post-prandial symptoms were attributed to hypoglycemia. That this erroneous concept was so long entertained is understandable inasmuch as (a) the symptoms often resembled those

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†By Dr. James I. Baltz, senior associate in gastroenterology, to whom I am indebted for these data.

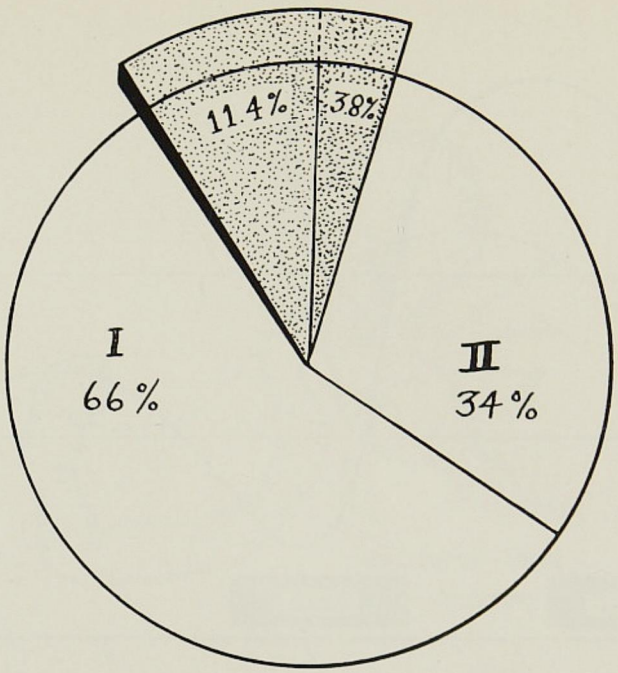

Figure 1

Among 158 gastrectomized patients followed an average of almost 5 years, gastroduodenostomy (Billroth I) was performed in 104 (66 per cent) and gastrojejunostomy (Billroth II) was performed in 54 (34 per cent). Less than "good" results were obtained in 15.2 per cent (strippled segment). A greater than proportional number of "fair" and "poor" results occurred following gastroduodenal anastomosis.

associated with excessive circulating epinephrine such as seen with hypoglycemia, and (b) levels of blood sugar in the post-prandial period were often depressed. With further study (Figure 2) it became apparent that the most intense distress occurred in the earliest post-prandial interval during which the blood sugar curve actually was rising, often to *hyper*glycemic levels. Only later and inconsistently was there "rebound" *hypo*glycemia.

It is now appreciated that there may occur both immediate and delayed post-prandial symptoms. The former are more commonly observed, and only the latter are associated with low blood sugar levels. For an elucidation of the mechanism of the immediate post-prandial symptoms, generally known as the "dumping syndrome", we are indebted to the researches of Machella[1] and, more recently, of Roberts and her associates[2].

Mechanism of the Dumping Syndrome. As we know only too well from the vivid descriptions provided by an unhappy minority of gastrectomized patients, the dumping syndrome may include the following symptoms: a disagreeable sense of upper abdominal fullness and pressure, nausea, vomiting, lightheadedness or near-syncope, palpitation, profuse diaphoresis, abdominal rumbling, and an intense urgency to stool with watery diarrhea. Under varying circumstances and in differing patients, the syndrome may be more or less complete. Elements of the dumping syndrome

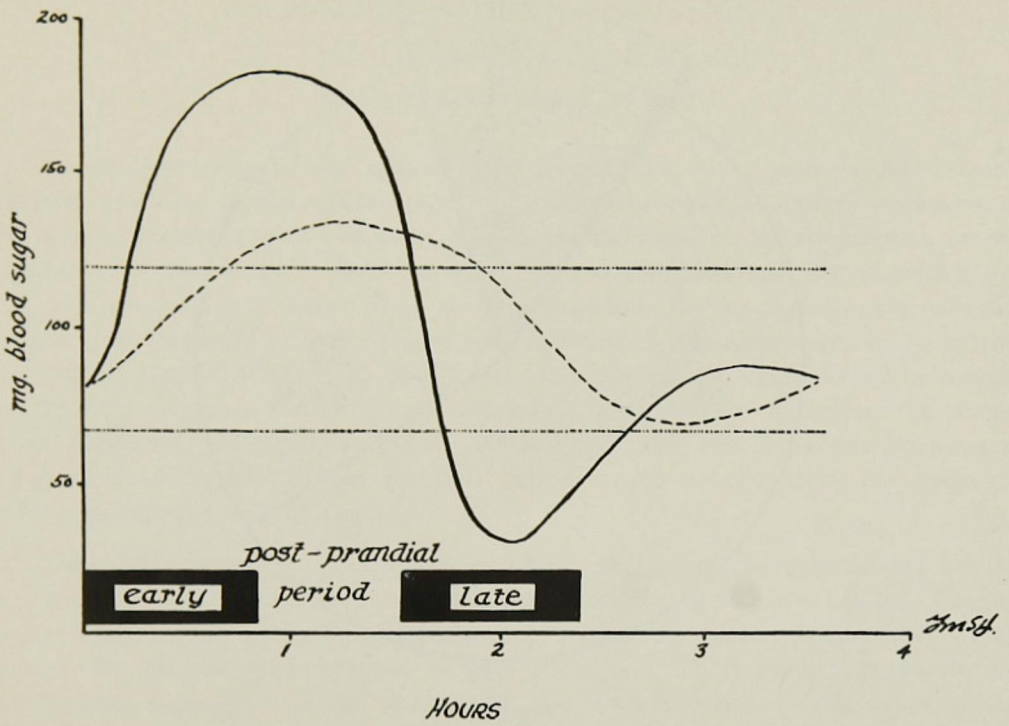

Figure 2

Temporal relationship between immediate and delayed post-prandial symptoms and the concentration of blood sugar in the gastrectomized patient (the usual curve of blood sugar for the individual with an intact stomach is depicted by the broken line).

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occur transitorily in many gastrectomized patients in the early post-operative period, particularly as the diet is first liberalized. Fortunately in only a small minority do symptoms continue unabated; in these few instances the syndrome may persist for months or years.

As the name implies, the dumping syndrome is initiated by the precipitate delivery of ingesta to the proximal small intestine. As such, "dumping" may be observed in the intact stomach from which emptying is unduly rapid and also with intraduodenal or intrajejunal tube feeding in which the rate of flow is excessive. The gastric remnant, following partial gastrectomy, has neither the reservoir capacity nor the pyloric mechanism to restrain the egress of fluid or food.

Machella demonstrated that jejunal distention alone, as by an inflated ballon, often reproduced features of the immediate post-prandial syndrome. It was postulated that such distention served as a stimulus to various autonomic (largely sympathetic) reflexes which, in turn, produced the undesirable symptoms. Such a mechanism does pertain; however, this explanation alone is incomplete.

It is a common observation that certain items of diet are incriminated by the patient more often than others. Among the frequent offenders are concentrated sweets

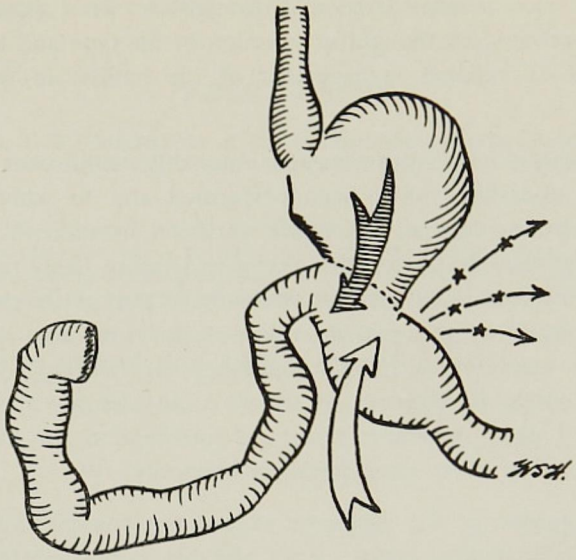

Figure 3

The precipitate and unrestrained "dumping" of food and fluid (shaded arrow) from the gastric remnant into the jejunal lumen results in rapid distention abetted by the inflow of fluid from circulating plasma (white arrow). Symptoms derive from the sympathetic reflexes initated by the distention and the acute fall in plasma volume.

which are actually highly hypertonic in relation to blood. The same effect is produced by the introduction of any substance which may be subject to rapid enzymatic hydrolysis and hence a high osmolarity within the jejunal lumen. Indeed it was demonstrated that the distention of the proximal small bowel resulted, not alone from the ingesta, but also from an outpouring of diluting fluid from the bowel wall itself as a means of inducing isotonicity in the lumenal contents. The only source for this fluid could be, of course, the circulating plasma.

Roberts and her co-workers have amply shown that an acute fall in plasma volume is regularly associated with the dumping syndrome. Further, they have demonstrated that *hypo*volemia ensues within 10 to 15 minutes after the introduction of any hypertonic solution into the jejunem, that it reaches its maximum in 30 to 40 minutes, and that it is spontaneously corrected after 80 to 120 minutes. Hypovolemia invokes sympathetic mechanisms mediated through the pressoreceptor centers in response to a fall in blood pressure and diminution in cardiac output. Although with rapid glycogenesis there is often depression of serum potassium and phosphate, Roberts and her associates found no correlation between the symptoms of the post-prandial period and the levels of these electrolytes.

Associated with the acute hypovolemia are perceptible and reproducible electrocardiographic changes which include tachycardia, flattening of T-waves, alteration in S-T segments, and the occasional appearance of U-waves.

Correction of Post-prandial Symptoms. Quite properly, the avoidance of the dumping syndrome may be considered in 3 categories: a) a discriminating selection of patients for gastrectomy, b) thoughtful selection of the type and technic of operation to the patient, and c) rational management of the patient in whom dumping has been observed.

Because dumping occurs by no means invariably within any group of patients in whom identical operations have been performed and to whom identical stimuli are offered, it is obvious that an individual variation in susceptibility must exist. I share with many observers the notion that dumping is more likely to be manifest in the asthenic, sensitive, nervous, and anxious individual than in the more stable citizen. I am particularly wary of the patient whose history includes past indications of unstable vasomotor reactions as habitual giddiness or fainting. Indeed, I speculate that objective tests might be constructed which could identify these individuals prior to gastrectomy. If I must recommend surgical intervention for these persons, I will try to select an operation other than partial gastrectomy.

With full recognition of the surgeon's skill, I am convinced that the technic of the operation is a contributing factor. Roth and his associates[3] have found that the frequency with which dumping is encountered is proportional to the extent of resection although admittedly the difference is not great. Although no man-made anastomosis can approximate the function of the intact pylorus, I believe that just the right size of stoma and the precise manner in which it is constructed are important in controlling, in part, the rapidity with which the ingested material is presented to the small intestine. It is the majority opinion that the Billroth I (gastroduodenal) anastomosis is less often attended by dumping than the Billroth II (gastrojejunal) anastomosis. Such has not been my own personal observation or the experience of this clinic.

Finally, selection of the operation and the skill of the surgeon notwithstanding, the problem of the patient who has exhibited dumping must be confronted. Sympathetic reassurance and explanation are often helpful, but ultimately the solution must be largely, if not exclusively, by manipulation of the diet. Such diet advice is based squarely on an understanding of the mechanism by which symptoms are produced. From the quantitative view, frequent, small, and relatively dry feedings are required. Qualitatively, the feedings must avoid concentrated sugars or other carbohydrates which may be rapidly hydrolyzed. Protein foods are tolerated well and fats may be helpful in retarding motility and providing maximum calories per unit volume. Anticholinergic medications are traditionally prescribed but are of limited usefulness. The judicious use of mild sedatives is indicated.

SUMMARY

Partial gastrectomy deprives the stomach, in whole or in part, of its normal function in the physiologic scheme of digestion. Precipitate dumping of ingested food and fluid from the stomach remnant produces duodenal or jejunal distention aggravated by an outpouring of fluid from the bowel wall in an attempt to render the lumenal contents isotonic. The resultant acute depression of plasma volume and sympathetic reflexes initiate the complex of subjective sensation which we know as the dumping

syndrome. Control of this untoward sequence requires discriminating selection of the patient, skillful surgery, and rational management of diet.

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