Current Status

Biliopancreatic Diversion: Mechanisms of Action and Long-Term Results

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Introduction

Biliopancreatic diversion (BPD) was conceived and experimented in dogs >30 years ago, and first performed in humans in 1976, always keeping in mind that weight reduction is useless unless it is followed by an indefinite weight maintenance. The operation consists of a distal gastrectomy with a long Roux-en-Y reconstruction where the enteroenterostomy is placed at a distal ileal level (Figure 1). As a general surgery operation that a good general surgeon can do, the important technical detail in open surgery as well as in laparoscopy, consists of measuring the small bowel, fully stretched in order to obtain reproducible measurements.

Mechanisms of Action

BPD can be considered a combined procedure, with a temporary mechanism for weight loss and a permanent one for weight maintenance. The temporary mechanism is based on a reduced gastric volume which, through a wide gastroenterostomy, rapidly empties into a relatively distal intestinal segment. This provokes temporary decrease of appetite and occurrence of postcibal syndrome, and thus food intake limitation which is responsible for the initial weight loss. As these effects are more intense and lasting with a smaller gastric volume, since 1984 the gastric volume has been adapted to the patient’s individual characteristics, including initial excess weight and other variables. The permanent mechanism of action of BPD is the delayed meeting of food with biliopancreatic juices, with consequent permanent reduced digestion and thus absorption of fat and starch.

Figure 1. A distal gastrectomy is performed. Small bowel is divided 250 cm proximal to ileocecal valve, and is anastomosed to the stomach remnant. The biliopancreatic limb (BPL) is anastomosed to the side of the distal limb 50 cm proximal to the ileocecal valve, to form a 200-cm alimentary limb (AL) and a 50-cm common limb (CL) where the major digestion occurs. The proximal gastric pouch initially restricts intake, which is maintained by the reduced absorptive area.
Our series now approaches 3,000 operations, 126 of which were done laparoscopically. There are three characteristics of weight loss that make BPD the most effective bariatric operation ever proposed. The first characteristic is its weight loss magnitude, which is >70% of the initial excess weight, both in obese and super-obese patients. The second characteristic of BPD weight loss is its constancy. The third and by far the most important characteristic of the BPD weight loss is the consistent long-term maintenance, with 70% loss of the initial excess weight maintained up to 25 years in a group of subjects undergoing the original type of BPD.

A study on intestinal absorption of fat, energy and nitrogen gave us the explanation for this almost unbelievable indefinite maintenance of the weight loss, demonstrating that the digestive-absorptive apparatus of BPD has a maximum transport capacity for fat and starch, and thus energy, which corresponds to ~1,250 Calories per day. All the energy intake that exceeds this maximum transport threshold is not absorbed, and because daily energy intake of operated patients is largely higher than the aforementioned threshold, daily energy absorption is constant for each subject. Thus, the body weight must also remain constant indefinitely. This was confirmed by an overfeeding study, where 10 long-term BPD subjects kept an absolutely stable body weight when fed 2,000 more calories of fat and starch than their usual intake, for 15 days.

Besides the obvious benefits due to the weight loss, some specific actions of BPD account for the permanent normalization of serum cholesterol and serum glucose in 100% of subjects with abnormally high preoperative values, which, in conjunction with the 90% and >80% normalization of triglyceridemia and blood pressure respectively, means an extraordinary effect on the metabolic syndrome.

The effect of gastric restrictive operations on glucose metabolism is simply consequent to weight loss, so that if the weight is regained the effect disappears. To understand what happens after Roux-en-Y gastric bypass (RYGBP) and BPD, let us remember that according to the Randle hypothesis, diabetes in obesity would be due to the increased free fatty acid (FFA) oxidation which in turn inhibits glucose oxidation, thus causing insulin resistance. More recently, the Swedish school suggested that hyperinsulinemia was due to decreased hepatic clearance of insulin secondary to increased FFA portal concentration, with consequent competition with the insulin receptors. In either case, the normalization of insulin sensitivity after BPD could simply be due to decreased lipid absorption and reduction of intraabdominal adipose tissue. Thus, it would not be a specific action and it would not explain the recovery from diabetes also in the ~20% of patients who still require insulin therapy after weight normalization by dieting. If we consider that, independent of its pathogenesis, type 2 diabetes mellitus is characterized by the vicious cycle in which hyperinsulinemia causes insulin resistance and vice versa, it is clear that any factor influencing that vicious cycle would have a beneficial result on diabetes. This is the case of the virtual annulment of the entero-insular axis resulting from bypass of the duodenum and proximal jejunum, which causes reduction of insulin production and then increased insulin sensitivity.

BPD adds to this a specific action, consisting of extremely reduced lipid absorption with consequent intramyocellular fat depletion and reversal of insulin resistance, and of course the reduced or annulled beta-cell fat toxicity. The nearly annulled entero-insular axis is proved by the extreme reduction of the gastric inhibitory polypeptide (GIP) which is considered the most important incretin, which after BPD results in sharply reduced basal levels, a flat curve in response to the meal, and a very reduced integrated response. The minimal fat absorption witnessed by our study demonstrates an average daily absorption of only ~40 grams of fat after BPD.

Unlike RYGBP, BPD has two specific actions on cholesterol metabolism, represented by the calibrated interruption of the enterohepatic bile salt circulation with the consequent increase of bile acid synthesis at the expense of the cholesterol pool, and the greatly reduced absorption of endogenous cholesterol due to the minimal fat absorption. These actions are also proved by a study which demonstrated a daily fecal excretion of bile salt of 750 mg versus an upper normal value of 400 mg, and again the extremely limited fat absorption which permits absorption of only a minimal part of the endogenous cholesterol.

Out of the 2,266 ad hoc stomach (AHS) BPD patients with a minimum follow-up of 1 year, 317 (14%) had preoperative simple hyperglycemia, 140 (6.2%) had type 2 diabetes manageable with oral hypoglycemics,