

Current Status

Biliopancreatic Diversion: Mechanisms of Action and Long-Term Results

Nicola Scopinaro, MD

Professor of Surgery, University of Genoa School of Medicine, Genoa, Italy

Introduction

Biliopancreatic diversion (BPD) was conceived and experimented in dogs >30 years ago,¹ and first performed in humans in 1976,² 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,³ 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.⁴

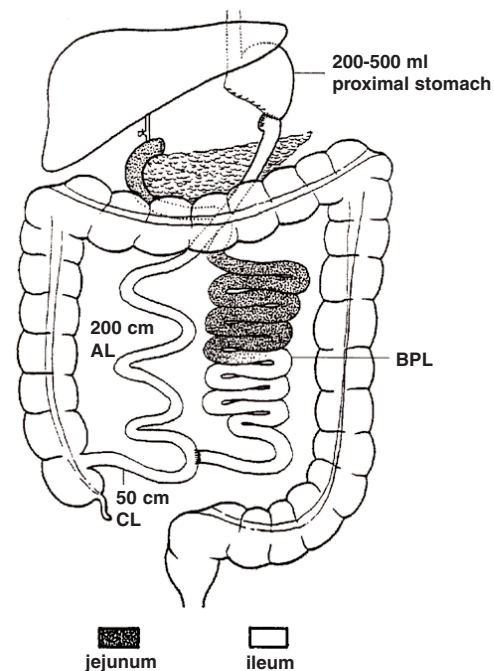


Figure 1. A distal gastrectomy is performed. Small bowel is divided 250 cm proximal to ileocecal valve, and is anastomosed to the stomach remnant. The biliopancreatic limb (BPL) is anastomosed to the side of the distal limb 50 cm proximal to the ileocecal valve, to form a 200-cm alimentary limb (AL) 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.

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Reprint requests to: Nicola Scopinaro, MD, DICMI - Semeiotica Chirurgica R, Università di Genova, Ospedale San Martino, 16132 Genova, Italy. Fax: +39-010-502-754; e-mail: scopinar@unige.it

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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 nitrogen⁵ 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 that 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.⁶

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,^{7,8} 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 school⁹ 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,¹⁰ and of course the reduced or annulled beta-cell fat toxicity.¹¹ 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.¹² The minimal fat absorption witnessed by our study⁵ 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 absorption⁵ 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,

and 39 (1.7%) had preoperative type 2 diabetes requiring insulin therapy. All but two of these patients 1 year after BPD and permanently thereafter have had normal serum glucose levels without medication on a totally free diet. There were another four cases of late mild relapse, but none of these subjects at long-term required any medication. Comprehensively, this is accompanied by normalization of serum insulin levels as demonstrated both in a longitudinal study,⁴ and in a cross-sectional one,¹² showing postoperative normal basal serum insulin levels and response to the test meal, as well as normal area under the curve, and by normalization of insulin sensitivity, which was explored in another study by means of the euglycemic hyperinsulinemic clamp.⁴ Interestingly, the preoperative insulin-dependent diabetic patients had normal serum glucose levels by 1 month after BPD, when the excess weight was still ~80%, thus again proving a specific effect of BPD independent of body weight changes. Also, the HOMA values, and thus again the insulin sensitivity, had normal values 1 to 2 months after BPD.¹³

Effect on Co-Morbidities

It is interesting to compare the beneficial effects on glucose metabolism of BPD and the other operations; RYGBP, besides very good long-term weight loss results, also has a specific action on glucose metabolism, but having only one of the specific actions of BPD, has an inferior impact on diabetes. The Greenville group^{1,15} presented the 10-year results of RYGBP in the treatment of type 2 diabetes in their 150 diabetic patients. Therefore, we considered the 1,576 AHS BPD patients who had a minimum 10-year follow-up at the end of 2003, 312 of whom had preoperative diabetes defined as fasting serum glucose >125 mg/dl. At 10 years after BPD, 42 (13%) of the operated patients were lost to follow-up. Of the remaining 270, 36 (13%) had died, so that 234 were available for evaluation. RYGBP patients had a reduction of mean fasting serum glucose from 187 to <140, maintained up to 10 years. The corresponding values for BPD patients were from 178 to <90. The percentage of patients cured, i.e. normoglycemic on a free diet and without medications, is 83% at 10 years in Pories' series,^{1,15} while it varies from 99 to 100% with 98% at 10

years in our series. In both series, these results are seen within a few days after the surgery, thus confirming the specific actions of the operations. In MacDonalds' group,¹⁵ patients being treated with insulin or oral hypoglycemics fell from 32% prior to surgery to 9% at last contact after RYGBP, while no one of our operated patients, not even the very few with incomplete recovery or mild relapse, has ever taken any medication in the 10-year follow-up.

Finally, mortality in MacDonalds' control group was 28% versus 9% in the surgical group. We do not have a control group thus far, and our overall mortality is 12%, including 4 cases of protein malnutrition, which probably represents the higher price to be paid for an operation with possible late life-threatening complications. There were only 4 cardiovascular deaths, i.e. slightly >1% compared to the 15% in the controls.

It is impossible to compare the effect of BPD on serum cholesterol, as RYGBP (not different to pure gastric restrictive operations) has no effect on cholesterol other than that consequent to the weight loss. In our series, mean reduction of serum cholesterol after BPD is ~30% in the patients with normal preoperative values, and ~45% in the preoperatively hypercholesterolemic patients. In particular, out of the 2,800 (total series) operated patients with a minimum follow-up of 1 month, 1,486 had serum cholesterol >200 mg/dl, 609 >240, and 102 >300. All of them had serum cholesterol <200 mg/dl 1 month after BPD, and no value >200 was ever observed during the 30-year follow-up. If we consider only patients for whom HDL cholesterol values were available, these results were maintained at the medium-term in 51 paired patients, while HDL cholesterol showed a slight but significant increase, and at very long-term in the 10 subjects with the original "half-half" model of BPD whose values were available at 15 to 20 years.

The effect of BPD on arterial hypertension is less impressive but still comprehensively very good. Considering a systolic blood pressure <140 and a diastolic pressure <85 to be normal, and considering only the 73 operated patients whose 10-year pressure is available, the normalization of blood pressure percentually increased during the 10 years of follow-up, reaching 80% at 10 years, despite aging and body weight stability, which is exactly the opposite of what the SOS study demonstrated after gastric restrictive procedures.¹⁶

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Some important inferences can be made at this point. As we saw, the specific metabolic effects of BPD are totally independent of weight changes, and this is demonstrated by the changes observed already at 1 month in serum glucose, insulin sensitivity, intramyocellular lipids, and also by the normalization of blood pressure which continues to increase at long-term despite the stability of body weight. So why not do BPD in non-morbidly obese or even in non-obese patients with metabolic syndrome? Not certainly for fear of an undesired weight loss. In fact, let us imagine a BPD patient with an intestinal energy absorption threshold corresponding to a body weight of 80 kg. We know that this is the weight that the patient will reach no matter how high the starting weight is. What would happen if, on the contrary, the starting weight was 70 kg? The obvious answer is, the patient would not lose weight. Thus, in order not to take any risk, let us place the threshold at a much higher level. We need now to anticipate that in BPD only fat absorption is limited to the common limb, while both protein and starch are absorbed both in the common and alimentary limb (see below). Therefore, the intestinal energy absorption threshold can rise considerably by elongating the alimentary limb, which does not affect the specific effects of the operation. This is not only a hypothesis, because it has been verified in two young lean sisters with familial chylomicronemia who did not lose any weight when submitted to BPD with a longer alimentary limb.¹⁷

The obvious conclusion is that BPD is the most powerful treatment now available for hyperlipidemia and type 2 diabetes, and that it could be effectively and safely used for treatment of severe metabolic syndrome in non-obese subjects also.

Certainly, this has a price, like all things in life, especially the good ones. Practice and experience led us out of the learning curve with an operative mortality steadily reduced to <0.5%, along with near disappearance of general complications and major surgical complications.

Specific Late Complications of BPD

Anemia, occurring only in patients with chronic bleeding, can be reduced to <5% with adequate supplementations.

Bone demineralization also requires calcium supplements to be prevented and cured. Interestingly, a significant percentage of the obese population already have moderate to severe bone disease preoperatively, which tends to improve with the weight loss. What likely happens is that in the first postoperative year the adverse effect of reduced calcium absorption prevails so that the percentage of bone disease increases, but subsequently the beneficial effect of the weight loss prevails. This general phenomenon is more evident in the populations with the most severe preoperative alterations, i.e. the older and the heavier patients.¹⁸⁻²⁰

The incidence of stomal ulcer, essentially confined to the first postoperative year, is strongly influenced, especially in women, by alcohol and, above all, cigarette smoking. Although ulcer is easily curable by antisecretory drugs, in smokers it can relapse indefinitely until the patient stops smoking.⁴

Peripheral neuropathy is an early complication which is very easily preventable by ensuring thiamin supplementation.²¹

Protein malnutrition, with its classic symptoms, is the most serious complication of BPD. It consists generally of a single early episode, because of lack of compliance with the alimentary rules to be observed during the initial postoperative period. Rarely it is recurrent, because of protein intake lower than protein requirement, which requires surgical correction by elongation of the common limb.^{22,23} The battle against this complication has been the most important one in the development of BPD.

To understand how technique, indications and postoperative management evolved until when we eventually reached the control of this complication, we need to consider principles of the physiology of BPD nutritional complications. The gastric volume greatly influences energy and protein absorption, both in the early postoperative period by causing reduction of appetite and occurrence of postcibal syndrome, and in the long-term by influencing intestinal transit time. These effects are more intense the smaller the gastric volume and the more distal the intestinal segment anastomosed to the stomach. Moreover, it is very important to understand that very few, if any, pancreatic enzymes reach the common limb, so that no pancreatic digestion occurs in the common limb. The only available digestive capacity is the brush border intestinal

enzymes. Consequently, protein and starch are digested and absorbed in the entire intestinal segment between the gastroenterostomy (GI) and the ileocecal valve (ICV), while, due to the need for bile salts, only fat absorption is strictly confined to the common limb.²⁴ As we know, BPD entails a maximum threshold absorption for fat and starch, and thus energy, which, with the standard lengths in our populations, corresponds to an average of 40 g/day for fat and 225 g/day for starch; this explains both the constancy of weight reduction and the long-term weight maintenance after BPD. The length of the common limb is also important because it must allow sufficient bile salt absorption to prevent excessive bile salt loss into the colon, and thus diarrhea with consequent reduction of colonic absorption. Interestingly and fortunately, contrary to fat and starch absorption, protein absorption after BPD depends on the alimentary intake and, with our standard lengths, corresponds to about 70%. Consequently, the greater the alimentary intake the greater the protein absorption, and the longer the alimentary limb the greater the protein absorption, but, due to the concomitant greater starch absorption, also the lesser the weight loss. Finally, a five-fold increase of endogenous nitrogen loss has been demonstrated after BPD. With all calculations made, this brings the average protein requirement up to about 90 grams per day, and this explains the risk of protein malnutrition after BPD.

In the early postoperative period, due to the forced reduced food intake, the prevalence of protein in the small amount of food consumed favors the development of the marasmic form of the protein-energy malnutrition, which is exactly the aim of the operation. However, if a preference is given to carbohydrate intake, the hypoalbuminemic form may develop which is the real complication, requiring rehospitalization and parenteral nutrition.

Because the operation functions for the entire lifetime, sporadic protein malnutrition can occur at any time after the operation, generally due to prolonged diarrhea from viral infection or to reduced food intake for any reason.

Of much greater importance is the recurrent form of protein malnutrition. A number of different factors, alone or in combination, can lead to this condition. Patient-dependant factors are insufficient protein intake, insufficient protein absorption per unit of intestinal surface, or excessive loss of endogenous

nitrogen, and operation-dependant factors are too small stomach (causing too rapid emptying and too rapid intestinal transit with permanently reduced absorption) or too short alimentary limb (causing insufficient protein absorption). One or more of these factors can be responsible for the complication. The concept is that that specific operation as it is, in that specific subject as he/she is, is incompatible with good protein nutritional status. After two or more episodes of protein malnutrition, the decision for a surgical revision is made, consisting of elongation of the common limb. Because, as already stated, protein absorption depends on the length of the small bowel from the GEA to the ICV, the elongation must not be done along the alimentary limb, but along the biliopancreatic one. The standard which in our hands guarantees the solution of all protein nutritional problems is to add 150 cm, for a final total length of 400 cm. This obviously entails the restabilization of body weight at an average level which corresponds to about 25% regain of the excess weight.

In order to better understand how we changed the operation in order to minimize the risk of protein malnutrition, let us summarize the causes of protein malnutrition (PM) after BPD. On the one hand, BPD itself, by decreasing protein absorption and increasing endogenous nitrogen loss, increases the protein requirement, which in turn influences both early episodic and recurrent PM. On the other hand, the reduced stomach volume temporarily decreases food intake and influences early episodic PM; it also permanently decreases intestinal transit time and thus reduces protein absorption, influencing both early episodic and late recurrent PM. We had initially been attracted by the extraordinary weight loss obtainable by reducing the gastric volume, and we drastically reduced the gastric volume to a mean of 150 ml, obtaining, in addition to a spectacular 90% loss of the initial excess weight, a catastrophic 30% incidence of PM with a 10% recurrence rate; the importance of the gastric volume was proved by the fact that the stomach of the PM patients was significantly smaller than that of the non-PM patients.

With the aim of decreasing the incidence of PM without losing the benefits of the small stomach, the gastric volume was then adapted to the patient's characteristics. The original philosophy of the "ad hoc stomach" BPD was to confine the risk of PM to the patients who required greater weight loss, by adapt-

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ing the gastric volume only to the initial excess weight. The mean gastric volume was reduced to ~300 ml, with an overall PM incidence of 15%, and a still very good 79% excess weight loss. Obviously, the patients with gastric volume <300 ml had a significantly greater incidence of PM. We decided then to adapt the gastric volume also to other patient characteristics, namely age, sex, eating habits and expected degree of compliance. The result was an overall 11% incidence of PM, with a 4.3% recurrence rate and the initial excess weight loss reduced to 75%. At that point, the absence of a significant difference in the gastric volume between PM and non-PM patients, demonstrated that the latter had no more influence on the incidence of PM. Therefore, in order to further minimize this complication, since 1992 we began to adapt also the length of the alimentary limb to the individual characteristics of the patients, because the main social-behavioral characteristics involved with the risk of PM are the protein content of customary food, the capacity of modifying eating habits according to the needs, and the financial status.

The resulting “ad hoc stomach ad hoc alimentary limb” BPD, in the last 300 patients with a minimum follow-up of 10 years, caused recurrent protein malnutrition in only 1%, which means reoperations in only 1% for late specific complications – and still a very good permanent reduction of 71% of the initial excess weight. The fact that this last policy yields the best compromise between effectiveness and safety is confirmed by a recent BAROS evaluation of >800 patients,^{25,26} which showed only a 2% failure rate in patients operated after 1992, with >90% of the results being from good to excellent.

Conclusion

A good knowledge of BPD physiology allows the adaptation of both the gastric volume and the length of the alimentary limb to the patient’s individual characteristics. The resulting only 1% revision rate for late specific complications makes BPD in our hands not only the most effective but also the safest operation now available for obesity treatment.

Therefore, the learning curve also beneficially affected our specific late complication rate, protein malnutrition dropping from 15 to <3%, stomal ulcer

from 9 to 4%, while the neurological complications simply disappeared.

BPD is a powerful weapon, and, as such, is potentially very dangerous if used improperly. Therefore, it should not be done sporadically. Its use should be confined to the hands of those surgeons who, by guaranteeing a certain number of operations per year and a very careful lifelong follow-up, can acquire the experience necessary to adapt the operation to their patient population, thus obtaining the best possible results with the minimum of complications.

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