8

BPD and BPD-DS Concerns and Results

Francesco Saverio Papadia, Hosam Elghadban, Andrea Weiss,
Corrado Parodi and Francesca Pagliardi
Genoa University,
Italy

1. Introduction

Biliopancreatic diversion (BPD) is considered one of the most effective surgical procedures in the treatment of obesity since its introduction in clinical practice by Professor Nicola Scopinaro in 1976. Nonetheless, it is, up until now, still largely the preserve of a selected group of bariatric surgeons, and faces frequently unjustified prejudices in its clinical acceptance.

In fact, despite the complexity of the operation (which is more apparent than real), its perceived operative risk (which is on the contrary probably even lower than operations such as sleeve gastrectomy) and the concerns regarding late metabolic sequelae (which can be extremely severe, but, with an adequate and in reality not particularly close follow-up are also extremely infrequent), biliopancreatic diversion with or without duodenal switch represents a formidable weapon in the most challenging cases, such as superobesity, uncontrolled metabolic syndrome, especially insulin-dependent diabetes type 2, and revisional surgery, in which it yields results that are far superior to those of any other bariatric operation. For these reasons, we believe that this operation should be in the armamentarium of every bariatric surgeon, to the point that no bariatric surgeon should be defined as such if he/she is not familiar with BPD and its patho-physiology.

In this chapter, we will briefly describe the development of the operation up to its latest modifications, highlight surgical technical points, detail the follow-up patients with BPD should be submitted to, explore early and late morbidity of the technique and describe the management of the complications. Finally, we will address the importance and ease of BPD as a revisional procedure.

1.1 Clinical development of BPD

The reduction of nutrient absorption was the first approach to surgical treatment of obesity. The early weight loss results with jejunoileal bypass (JIB) led to more than 100,000 of these operations performed in the USA through the years 1960's and 1970's. However, the analysis of late results and complications of JIB caused a drastic coolness of the initial enthusiasm. In addition to its complications, essentially due to indiscriminate malabsorption and the harmful effects of the long blind loop, the main problem with JIB is its narrow "therapeutic interval". In fact, the total length of the small bowel left in continuity is restrained within the range of 40 to 60 cm, a shorter or longer bypass resulting in life-threatening malabsorption or no weight reduction, respectively. On the other hand, the

massive intestinal adaptation phenomena cause an increased absorptive surface leading out of the upper limits of the above range, with ensuing substantial recovery of energy absorption capacity (Scopinaro, 1974). This, in addition to the frequent need of restoration for major complications, ends in a high rate of failure with weight regain (Halverson et al, 1980; MacLean & Rhode, 1987). The high complication rate and the overall unsatisfactory weight loss results of jejunoileal bypass (JIB) during the years around 1980 led to general abandoning of the malabsorptive approach for obesity surgery, the gastric restriction procedures becoming those most frequently used.

Because of the absence of a blind loop and of the malabsorption essentially selective for fat and starch, biliopancreatic diversion (BPD) is largely free of many of the complications pertaining to JIB (Scopinaro et al, 1979a, 1979b). Moreover, BPD has a very wide "therapeutic interval" because by varying the length of the intestinal limbs, any degree of fat, starch and protein malabsorption can be created, thereby adapting the procedure to the population's or even the patient's characteristics, to obtain the best possible weight loss results with the minimum of complications (Scopinaro et al, 1996) . This extreme flexibility also allows us to neutralize the consequences of intestinal adaptation phenomena, which, on the other hand, are little effective in BPD.

BPD consists of a partial gastrectomy with a gastro-ileal anastomosis, which results in a temporary decrease of appetite and occurrence of postcibal syndrome, and thus a reduction of food intake during the early postoperative period. Gastrointestinal continuity is obtained by the construction of a long Roux-en-Y with an alimentary limb of variable length (usually between 200 and 250 cm) and a 50 cm common channel (Fig.1).
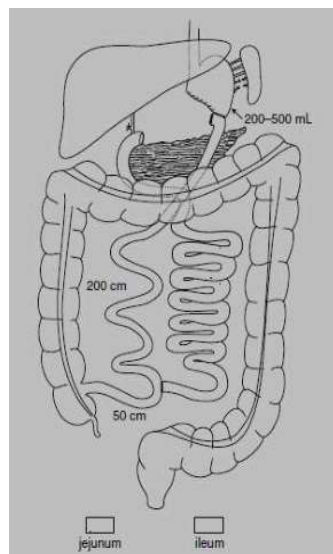

Fig. 1. Ad hoc stomach biliopancreatic diversion. Alimentary limb, from gastroenterostomy (GEA) to enteroenterostomy (EEA); biliopancreatic limb, from duodenum to EEA; and common limb, from EEA to ileocecal valve (ICV)

This anatomical arrangement creates malabsorption that is essentially selective for fat and starch, and ensures long-term weight maintenance. In our experience, the long-term results of this operation are represented by a mean reduction of approximately 75% of the initial excess weight (IEW), a weight loss which is maintained over 20 years.

2. BPD physiology

Through this part of the chapter we will try to explain the new physiology of the digestive apparatus after BPD and the specific effect of BPD on the metabolic syndrome.

2.1 Weight loss and maintenance

The initial weight loss is determined by the **temporary forced food limitation** that occurs immediately after operation. As a rule, the operated patient fully recovers appetite and eating capacity before the stabilization weight is attained. The final weight loss (the weight of stabilization) depends on **the amount of daily energy absorption** that the operation permits (a permanent mechanism), and influenced by the gastric volume, most likely because a smaller stomach, resulting in more rapid gastric emptying, accelerates intestinal transit, thereby reducing absorption (Scopinaro et al, 1999).

The original philosophy for limitation of digestion in BPD was to delay the meeting between food and biliopancreatic juice in order to confine the pancreatic digestion to a short segment of small bowel. The analysis of changes in weight loss and in protein intestinal absorption in the BPD models that followed each other in the evolution of the operation (Gianetta etal, 1980; Scopinaro et al, 1980; 1997) demonstrated that in the present model of BPD no pancreatic digestion occurs in the CL. Protein and starch digestion, which is only due to intestinal brush-border enzymes, occurs in the entire small bowel from the GEA to the ICV, while only fat absorption, which needs the presence of bile salts, is confined to the CL.

Some clinical-statistical observations on the modalities of this very long term weight maintenance indicate that body weight after BPD is essentially independent of individual and interindividual variations of food intake. This prompted us to investigate the relations between usual energy intake and energy intestinal absorption.

		Alimentary intake	Fecal loss	Apparent absorption	Apparent absorption (%)
energy (kcal/24h)	mean	3070	1329	1741	58
	range	1840-4060	210-2590	1012-2827	32-71
fat (g/24h)	mean	130	89	39	28
	range	88-185	22-251	13-94	12-59
nitrogen (g/24h)	mean	27	12	15	57
	range	15-48	2.5-36	6.7-20	25-82
calcium (mg/24h)	mean	1994	1443	551	26
	range	1037-3979	453-2565	251-1414	-24-69

Table 1. Energy, fat, nitrogen and calcium intestinal apparent absorption in 15 subjects (3 men) with stable body weight 2-3 years after BPD (mean ± s.d. body weight: at the time of the operation 119 ± 24 kg; at the time of the study 75 ± 14 kg).

An absorption study was carried out,(Scopinaro et al, 2000) the results of which are reported in Table –1, demonstrating that the BPD digestive/absorptive apparatus has a maximum transport capacity for fat and starch, and thus energy. Consequently, all the energy intake that exceeds the maximum transport threshold is not absorbed; therefore, assuming that daily energy intake is largely higher than the aforementioned threshold, daily energy absorption is constant for each subject. Therefore, in each BPD individual, the weight of stabilization cannot be modified by any increase or decrease of fat–starch intake, provided the intake is greater than the maximum transport threshold.

In conclusion, the original intestinal lengths and gastric volume being equal, the interindividual variability of the weight of stabilization in BPD subjects is accounted for by interindividual differences of; (1) Original energy intestinal digestive-absorptive capacity per unit of surface; (2) Intestinal adaptation phenomena; (3) Intestinal transit time (which, in addition to gastric volume, can be influenced by the intake of fluids); (4) Simple sugar intake; and (5) Energy expenditure per unit of body mass. In reality, since the intestinal carrier becomes rapidly desaturated after the passage of food, an increased number of meals per day can also increase energy absorption, and this is confirmed by clinical experience.

The aforementioned results were confirmed by **an overfeeding study**, where 10 long-term BPD subjects kept a strictly stable body weight when fed their usual diet for 15 days and the same diet plus 2,000 fat–starch kcal/day (without increasing the number of meals per day) for 15 more days (Table 2), without observing any increase in body weight, considering that, with a positive balance of 2000kcal/day for 15 days, the average increase in body weight should have been in excess of 2 kg (Forbes GB, 1987).

subjects	initial BW	BW on usual food intake	BW after overfeeding
1	77.7	78.0	78.0
2	90.0	90.5	89.2
3	97.0	96.5	95.7
4	73.0	72.7	73.4
5	89.1	88.8	90.3
6	68.5	68.0	68.5
7	102.8	103.5	103.0
8	87.0	87.0	86.5
9	66.5	66.0	66.0
10	70.5	70.0	71.0

Table 2. Overfeeding study in 10 subjects 3-9 years after BPD. Individual data of body weight (BW, kg) at the beginning of the study, after a 15 day period on usual food intake (mean: ~ 3800 kcal/day) and after a 15 day period of overfeeding (usual food intake plus 2000 fat/starch kcal/day).

3. Immediate morbidity and mortality

BPD is major abdominal surgery and, as for any other similar operation, its postoperative morbidity and mortality essentially depend on the frequency in its use. We do an average of about 100 open plus 100 laparoscopic operations per year, and our mortality rate is steadily

<0.5%. Similarly, general and major surgical complications tend to decrease with increasing surgical volumes and are reasonably low. Both the anastomoses are well vascularized and without tension, so leaks are exceptional.

3.1 Weight loss results

The 50-cm CL in BPD has proven to be the best compromise between fat absorption limitation and bile salt loss in the colon such as not to cause bile acid diarrhea. Lenghtening the common limb will increase fat and bile salt absorption; protein absorption needs an elongation of the total bowel length comprised between GEA and ICV. For the rest, the smaller the gastric volume and the shorter the small bowel between the GEA and the ICV, the lower the stabilization weight but the greater the risk of nutritional complications.
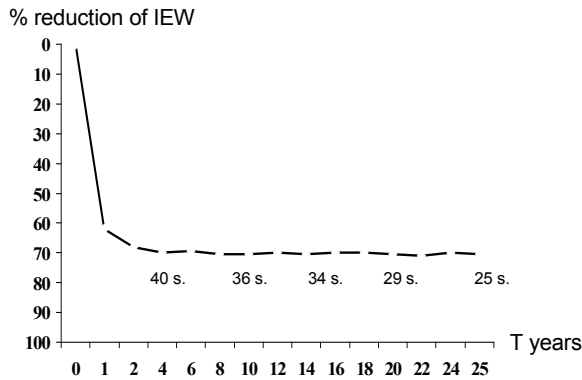

Fig. 2. Weight changes following "ad hoc stomach" BPD in operated subjects with minimum follow-up 5 years. Unpaired data: 2 years 2,371 patients; 20 years 427 patients.
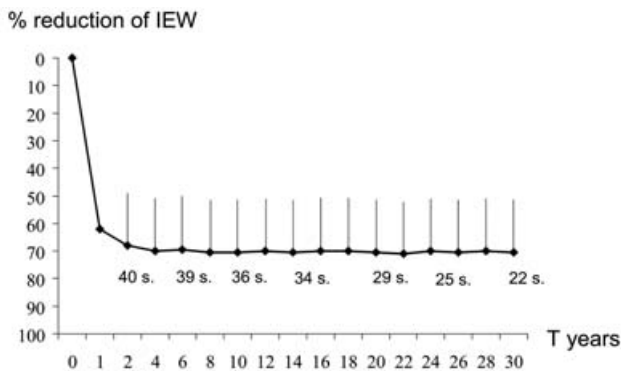

Fig. 3. Very long-term weight changes in a group of 40 subjects submitted to "half-half" BPD. Paired data.

The 20-year weight loss curve in **Fig. 2** (patients with a minimum follow-up of 5 years) goes from little more than 70% loss of the initial excess weight at 1 year to about 80 IEW%L at 20

years. This of course does not mean that the weight loss increases with time. Simply, the one with unpaired data in the graph shows that, by slowly increasing the gastric volume and the alimentary limb length, we reached the other extremely important compromise, the one between weight loss and protein nutritional complications. To make a long story brief, 25 years ago we had a wonderful 90% EWL, at the unacceptable price of protein malnutrition in ~30% of patients, which was recurrent in ~10%, and thus necessitated revision of the BPD consisting of elongation of the CL (obviously, at the expense of the BPL; see revision).

The real weight maintenance after BPD can be appreciated in **Fig. 3,** where weight loss paired data is reported for a group of 40 patients submitted to the original "half-half " model of BPD, 22 of whom who could be followed-up until the 30th year.

In conclusion, after BPD the weight maintenance is ensured by the existence of an intestinal energy transport threshold. The weight of stabilization depends partly on that threshold and partly on the changes of body composition consequent to the operation.

3.2 Other beneficial effect

The other benefits obtained after BPD are listed in **Table 3**. The percents of changes observed after the operation were calculated for each complication in patients with a

	minimum follow-up (mo)	disappeared (%)	improved (%)	unchanged (%)	impaired (%)
Pickwickian syndrome* (2%)	1	100	-	-	-
Somnolence† (6%)	1	100	-	-	-
Hypertension‡ (39%)	12	81	13	6	-
Fatty liver§ (46%)	24	87	9	4	-
Leg stasis• (31%)	12	45	39	16	-
Hypercholesterolemia¶ (55%)	1	100	-	-	-
Hypertriglyceridemia (33%)	12	95	5	-	-
Hyperglycemia (14%)	4	100	-	-	-
DM (6%)	4	100	-	-	-
DM requiring insulin (2%)	12	100	-	-	-
Hyperuricemia (16%)	4	94	-	3	3
Gout (2%)	4	100#	-	-	-

(%) percent of patients with condition.
*Somnolence with cyanosis, polycythemia, and hypercapnia
†In absence of one or more characteristics of pickwickian syndrome.
‡Systolic ≥ 155, diastolic ≥ 95 mmHg, or both.
§More than 10%.
•Moderate or severe.
¶More than 200 mg/mL (21% more than 240 mg/mL).
#Serum uric acid normalized, no more clinical symptoms.

Table 3. Other beneficial effects of AHS BPD.

minimum follow-up corresponding to the postoperative time after which there was generally no further substantial modification.

Recovery and improvement were considered only when favorable changes were essentially maintained at all subsequent reexaminations. The observed beneficial effects are obviously not attributable to the BPD itself, but to the weight loss and/or the reduced nutrient absorption, the only two exceptions being the effects on glucose and cholesterol metabolism (Scopinaro et al, 1997, Marinari et al, 1997).

3.3 Glucose homeostasis and antidiabetic effects of BPD

One of the most impressive benefits of BPD other than weight loss is certainly represented by the disappearance of type 2 diabetes mellitus (T2DM) in nearly 100% of the previously diabetic morbidly obese patients. This effect was observed both at short term in Buchwald meta-analyses (Buchwald et al, 2004, 2009) and as long as 10 years (Scopinaro et al, 2005) and even 20 years after BPD in very long-term series (Scopinaro et al, 2008).

 It could be not surprising, considering that weight reduction, no matter how obtained, causes a decrease in insulin resistance (McAuley & Mann, 2006), thus explaining the beneficial effect on T2DM the greater and the more sustained depending on size and duration of weight reduction. Even the apparent resolution of T2DM occurring within days after operation could be easily explained keeping in mind that insulin resistance, which, due to the surgical stress, generally increases immediately after a surgical operation (Brandi et al, 1993, Thorell et al, 1999) is on the contrary postoperatively reduced in the diabetic patient (Adami et al, 2003, Wickremesekera et al, 2005). The early and late effect of BPD on T2DM could then be an aspecific consequence of weight loss and calorie deprivation, shared with all other bariatric operations. However, the normalization of insulin sensitivity after BPD is maintained after weight has stabilized around BMI 30 that is still in the obese range. Moreover, differently from other bariatric procedures, BPD has shown the ability to restore acute insulin response to intravenous glucose load (AIR) in morbidly obese patients, both at short (Briatore et al, 2008) and long term (Polyzogopoulou et al, 2003).

Furthermore, a group of preoperatively diabetic women after BPD never showed one single serum glucose value higher than normal during the whole pregnancy and delivered normal weight babies, thus demonstrating a beta-cell function adequate to the requirement (Adami et al, 2008). This restored insulin secretion capacity, which was never observed before, not only demonstrates that beta cell function is not irreversibly lost in T2DM patients but also indicates that BPD possesses a specific action, independent of weight loss and negative calorie balance, which, together with the normalized insulin sensitivity, fully accounts for the diabetes resolution after the operation.

Hypothetically, the BPD-specific action can be identified with the food-stimulated incretin GLP-1, produced by the distal ileum, where after BPD stomach directly empties, which was demonstrated able to improve beta-cell function (Doyle & Egan, 2007), stimulate beta-cell proliferation (Xu et al, 1999), and inhibit beta cell apoptosis (Farilla et al,2003). GLP-1 production was found increased after BPD both in rat (Borg et al, 2007) and in man (Valverde et al, 2005, Guidone et al, 2006). More than 90% of type 2 diabetic patients are not morbidly obese, being in the BMI range 25 to 35. Recently, with the aim to investigate if the BPD effect is maintained in the above BMI range, we submitted to BPD 30 T2DM patients belonging to that range, with the obvious rationale that, if the action of BPD is specific, and

thus independent of weight loss, it should be maintained also in the patients who, being only mildly obese or simply overweight, lose little or no weight after operation (Scopinaro et al, 2011). The reason why BPD does not entail risk of excessive or undue weight loss is that there is a maximum energy absorption capacity after the operation, which corresponds to a weight of stabilization of about 85 kg for men and 70 kg for women (Scopinaro et al, 2000). The more starting weight, with the related energy intake, approaches these values, the less calorie imbalance, and thus weight loss, is to be expected after BPD. Patients with initial weight equal to or lower than those should absorb all energy they eat and consequently have no energy malabsorption or weight reduction at all.

Recently, our group published the first results of BPD in diabetic, non/morbidly obese patients BMI 25-34.9 kg/m². The main finding of this study was the striking difference between the effect of BPD on T2DM in the morbidly obese patients (BMI > 35) and the patients with BMI 25-34.9 kg/m². One year after the operation, this latter group had a control (HbA1c≥7%) rate of 83%, normal HbA1c (≥ 6.5%) was found in 63% of patients, whereas full remission (FSG ≥ 110 mg/dL) was shown by only 30% of patients, vs. nearly 100% in the morbidly obese group. Diabetic patients with BMI >35 showed then a much better response to BPD than those with BMI 25-34.9 kg/m², considered together.

Contrary to expectations, in this study mean serum triglyceride values remained essentially unchanged during the first postoperative year, whereas the percentage of abnormally high values showed a remarkable increase until the eighth postoperative month, followed by a nonsignificant reduction, ending up with values at 1 year double than preoperatively. An increase of serum triglyceride concentration following interruption of enterohepatic bile acid circulation by means of bile-acid-binding resins (cholestyramine or colestipol) was demonstrated almost 30 years ago (Beil et al, 1980, Ast & Frishman, 1990). Since an important interruption of the enterohepatic bile acid circulation also occurs after BPD, our hypothesis is that, due to increased liver bile acid neosynthesis aimed at compensating for intestinal loss, cholesterol pool is depleted, and very low-density lipoprotein synthesis is stimulated, with consequent VLDL-triglyceride parallel increase. Indeed, an elongation of the common limb up to 100 cm, which entails a greater bile acid absorption in the distal ileum, led to normalization of postoperative serum triglyceride levels up until now in a small series of diabetic patients with BMI< 30. Incidentally, this elongation determines a significant amelioration in bowel habits.

3.4 Cholesterol homeostasis

Two specific actions of BPD account for the permanent serum cholesterol normalization in 100% of operated patients: the first is the calibrated interruption of the enterohepatic bile salt circulation (bile acids are electively absorbed by the distal ileum) that causes enhanced synthesis of bile acids at the expense of the cholesterol pool; the second specific action is the strongly reduced absorption of endogenous cholesterol consequent to the limitation of fat absorption.

The serum cholesterol level shows a stable mean reduction of approximately 30% in patients with normal preoperative values and 45% in patients who were hypercholesterolemic before the operation (Gianetta et al, 1985). High-density lipoprotein (HDL) cholesterol remains unchanged, the reduction being entirely at the expense of low-density lipoprotein (LDL) and very lowdensity lipoprotein (VLDL) cholesterol (Montagna et al, 1987). These results were

maintained at long term, the HDL cholesterol showing a significant increase, in 51 BPD subjects at 6 years (total serum cholesterol: preop. 210 ± 46 mg/dL, postop. 124 ± 25 mg/dL, Student's t test, $p < 0.0001$; HDL cholesterol: preop. 44±12 mg/dL, postop. 50±15 mg/dL, Student's t test, $p < 0.03$) and at very long termin the 10HHBPD subjects whose values were available 15–20 years after operation (Table 4).With the National Institutes of Health criterion of 200 mg/dL as the upper recommended limit for serum cholesterol, of the 2,888 (total series) obese patients submitted to BPD with a minimum follow-up of 1 month, 1,542 had hypercholesterolemia (612 had values higher than 240 mg/dL and 110 had values higher than 300 mg/dL). All of these patients had serum cholesterol values lower than 200 mg/dL 1 month after operation, and the values remained below that level at all subsequent examinations.

subjects	preoperative total serum cholesterol	serum cholesterol 15-20 years after HH BPD	
		total	HDL
1	205	116	47
2	140	125	38
3	150	140	52
4	210	158	65
5	280	158	73
6	230	127	61
7	180	118	35
8	285	120	36
9	189	130	59
10	260	171	33
mean	213	136 *	50

Table 4. Serum cholesterol (mg/dl) in ten subjects before and 15-20 years after HH BPD.

4. Patient selection and perioperative preparation

Patients who undergo either BPD or DS must be prepared for the consequences of a malabsorptive operation. In very simple words, it must be made clear to the patient that he/she will trade one illness (severe obesity with or without its complications) with another one, less morbid and easier to control, malabsorption. Once this clear, it is probably easier to understand the necessity for lifelong chronic follow-up.

There is no clear consensus on indications and contraindications to BPD in the bariatric surgical community. Absolute contraindications are chronic diarrhea, alcoholism, inflammatory bowel disease, uncontrolled psychiatric illnesses, chronic renal failure, severe liver cirrhosis, and endogenous protein loss (protein losing enteropathy, nephrotic syndrome, etc.).Other relative contraindications for the procedure are significant geographic distance from the surgeon, lack of financial means to afford supplements, inability or unwillingness to undergo lifelong chronic follow-up.

On the other hand, BPD is a very useful tool for those patients with superobesity, severe metabolic syndrome especially uncontrolled T2DM, and as a surgical option for revision of

failed other bariatric operations. Finally, although still investigational, promising preliminary reports have been given by our group for BPD in the surgical approach to type 2 diabetes mellitus in patients with a BMI between 35 and 25 as mentioned above.

Patient selection begins in the outpatient office. Particular attention has to be put on the patients' eating habits and behavior, because a protein-rich postoperative diet is required. The postoperative daily minimum oral protein requirements are approximately 80 g/day, which are easily met in most European and North-American diets, but that could be challenging e.g. in vegetarians or in patients less prone to eating meat or protein in general. Therefore, some basic nutritional skills are required in the surgeons' hands, in order to discriminate patients with an obviously bad outcome. It is in fact to be expected that the patient will not change his/her habits, and therefore the operation will have to be tailored to the patient, and not the patient to the operation.

Furthermore, false expectations and realistic goals have to be discussed openly with the patient: mean postoperative % excess weight loss is 70%, and any greater degree of weight loss is likely to be associated with complications. Finally, bowel habits change have to be discussed very clearly. Bowel movements will be increased in the vast majority of patients, slight modifications of eating habits may be necessary to achieve the best results and minimize side effects: milk, simple sugars, large amounts of fruits and vegetables should be avoided in order to prevent weight regain and reduce flatulence and frequency of stools. It is nonetheless to be remarked that sweet-eaters and nibblers have bad outcomes with any operation.

Being a choice for superobese and bariatric revisional cases, BPD candidates usually present with co-morbidities that are much more severe than the usual bariatric candidate. A multi-disciplinary approach comprising sleep-apnea studies, pulmonary and respiratory function tests, arterial blood gases, cardiologic consultation should be easily available. In case of abnormal arterial blood gases or sleep apnea studies, outpatient C-PAP prescription is mandatory to improve respiratory function. Any coexisting co-morbidity should, if possible, be treated, always with the aim to reduce perioperative morbidity.

Preoperative weight loss is always desirable, nonetheless it is very difficult to achieve in practice. A two-step approach is feasible for BPD with duodenal switch, but it is impossible for standard BPD, where intragastric balloon placement and subsequent surgery can be offered.

On top of the routine preoperative laboratory investigations, we usually stress upon vitamin A, D and E and PTH determinations, in order to have a baseline value upon which to compare the subsequent follow-up values. Preoperative investigations should reasonably rule out pregnancy and chronic renal or liver disease, either by biochemical tests, or by ultrasound. Whereas frankly abnormal liver function tests may highlight an undiagnosed NASH, which benefits from the operation, patients with abnormal platelet counts, low albumin, high creatinine or any frankly abnormal finding on abdominal ultrasound should be put on hold or excluded from the operation.

Upper GI radiology and endoscopy are not routinely used, except for revisional cases, because the routine distal gastric resection eliminates the problem of the unexplored distal gastric stump, which is on the contrary of concern in gastric bypass.

After admission, routine antithrombotic prophylaxis is started, usually with 4000 to 6000 units of low-molecular weight heparin. No bowel preparation is necessary: the patient is kept on a liquid diet the day before the operation. A single dose of second-generation cephalosporin is administered as an ultra-short-term antibiotic prophylaxis half an hour before skin incision.

5. Surgical technique

5.1 Position of the patient and operating table

The operating table should be specific for bariatric surgery, and must allow tilting and extreme inclination in total safety. We must pay especially close attention to the pressure areas, since, due to the patient's weight there is a greater risk of ischemic, venous, and nervous injuries. For the laparoscopic approach, the patient is placed with legs wide apart; the operator stands between the patient's legs, the cameraman [assistant] generally to his right and 2nd assistant to his left. The laparoscopy monitor should be placed to the right of the patient, next to the head of the table.

An urinary catheter is routinely placed, and kept for the first 24 hours. A central venous line is not mandatory, but in practical terms useful especially for high-risk patients. Invasive pressure monitoring is not used routinely, but fiberoptic conscious sedation intubation is used in difficult patients, according to the anesthesiologists' preference.

5.2 Trocar position

The pneumoperitoneum is usually performed through Trocar 1 supraumbilical midline 10/12 mm port done through open procedure with Hasson technique [safe]. In morbidly obese patients, the umbilical scar should never be used as an anatomic point of reference for the introduction of the trocars. $CO2$ pressure should be maintained at 15 mm/Hg during the procedure. Once the 30 degree optical system has been inserted, an additional 4-5 trocars are placed under direct vision according to the following diagram (Fig.5.).

Trocar 2 (10/12 mm) along the left midclavicular line, about 6 cm below the costal margin.
Trocar 3 (10/12 mm) along the right midclavicular line, about 6 cm below the costal margin
Trocar 4 (10/12 mm) on the midline, 3 cm below the xiphoid.
Trocar 5 (5mm) on the left costal margin, along the left middle axillary line.

Occasionally, if the intestinal measurement or the entero-enterostomy appears difficult, an additional 5 mm trocar is introduced in the left iliac fossa. The procedure is entirely done with a 10-mm and 30∘ optical camera.

5.3 Operative steps (laparoscopic approach)

5.3.1 First phase: cholecystectomy and gastric resection

Cholecystectomy [prophylactic or therapeutic] is carried out first and the gallbladder is left in the right hypochondrium over the liver to be removed from the abdominal cavity at the end of the surgery. With the surgeon between the patient's legs and the operating table slight head up, the optical system stays in portal 1; the first assistant uses the grasping
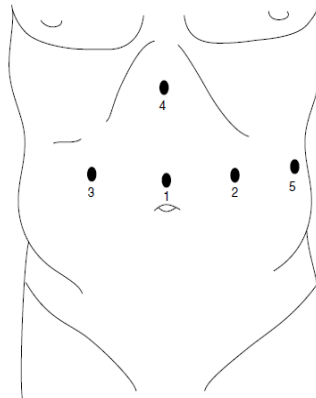
Fig. 5. Trocar position in laparoscopic standard biliopancreatic diversion. 1: supraumbilical (10–12 mm), on the midline, 3–4 cm above the superior margin of the umbilicus; 2: left hypondriac (10–12 mm), along the left midclavicular line, about 6 cm below the costal margin; 3 right hypondriac (10–12 mm), along the right midclavicular line, about 6 cm below the costal margin; 4: xiphoid (10–12 mm), on the midline, 3 cm below the xiphoid; 5: left subcostal (5 mm), on the left costal margin, along the left middle axillary line.

forceps in portal 5 to expose the stomach. The surgeon uses portal 2 for the harmonic scalpel and portal 3 for the grasping forceps, thus exposing and dissecting the stomach. The second assistant pushes the liver away by means of portal 4.

The gastrectomy is made in the caudo-cranial direction following the great curvature, starting in the middle and ending up dissection after division of the first two short gastric vessels. Aim of the dissection is to fully mobilize the gastric fundus, so that the stapler can be safely fired about 15 cm below the angle of His. This dissection is always made with the harmonic scalpel, and close to the gastric wall, aiming at reducing bleeding, and it extends down to 2 cm distal from the pylorus. Subsequently, the lesser omentum is incised, and the right gastric vessels are ligated close to the pylorus. Next, the duodenum is transected with a linear endostapler: we usually use a 45 or 60 mm stapler with a blue cartridge introduced through the port 3.

After the duodenal transection, the stomach is pulled caudally and to the left in order to facilitate the dissection of the lesser gastric curvature, which is made up to the level of the left gastric artery, always close to the gastric wall, so as to prevent bleeding. Once the level of the left gastric artery is reached, the gastric resection is carried out by repeated firing of endoGIA 45 or 60, blue and green cartridges, starting from the greater curve, at approximately 15 cm from the angle of His, towards the lesser curve, at the end of the dissection, about 7 cm caudally from the cardias. A landmark of 15 cm on the greater curve corresponds to a gastric volume of about 300mL; a distance of 20 cm corresponds to a volume of about 500mL. The divided stomach is left in the patient's left hypochondrium to be removed at the end of surgery together with the gallbladder.

5.3.2 Second phase: intestinal measurement and enteroenterostomy

The operating table is rotated approximately 15° degree to the left with a slight Trendelenburg position. The surgeon positions himself at the level of the patient's left flank, having the first assistant on his left and the second assistant on his right, at the level of the patient's head. The optical system is placed in portal 2 by the first assistant.

The surgeon uses portals 5 and 6 by grasping forceps for measuring the intestinal loops. Both forceps have a mark at 10 cm from the distal extremity. The small bowel is measured backwardws from the cecum, fully stretched, using the two forceps in alternating movements. A mark is left at 50 cm. The measurement of the loop continues up to 250 or 300 cm from the ileocecal valve, at which level it is divided using the linear endoGIA, which is introduced through portal 5. At this point the alimentary limb should be identified by a stitch, and the mesentery is sectioned in depth with the ultrasonic scissors. Care should be taken to arrange the alimentary limb to the patient's right side and the biliopancreatic limb to the left side to avoid twist.

Next, the stitch left 50 cm from the ileocecal valve is identified, and the biliopancreatic limb is brought next to it. The correct orientation and positioning of the limbs has to be checked before stapler firing to ensure that there is no twist in the mesentery. If The enteroanastomosis is performed in a laterolateral, isoperistatic technique. This is done first by opening a small orifice into both loops with the harmonic scalpel and passing a linear endostapler through them. The enterotomies are then closed in a running seromuscular suture.

5.3.3 Third phase: gastroenterostomy, liver biopsy and drain placement

The surgeons return back to the primary position, with the surgeon between the patient's legs, and the assistants to his left and right. The transverse mesocolon is elevated up until identification of Treitz's ligament, and a small opening is performed with the harmonic scalpel just above it. The surgeon then pulls the left angle of the gastric stump though it into the submesocolic space. The distal intestinal stump (alimentary limb) is identified and perforated with the ultrasonic scissors at a distance from the suture line equal to the operative length of the endoGIA 45 or 60. Of extreme importance at this pont is the final check of the correct orientation of the intestinal limbs. The alimentary limb should lie on the right of the patient,, adherent to the transverse mesocolon and the root of the mesentery. It should be traced from its most cranial stump up until the entero-enterostomy, and no additional bowel loops should be interposed: this is an unequivocal sign of twist.

Next, a laterolateral isoperistaltic gastro-enterostomy is performed on the posterior wall of the stomach, as close as possible to the distal angle and at midway between the suture line and the greater curve, with manual closure of the conjoined defect. A methylene blue test of the anastomosis is performed in the end.

Liver biosy is taken for baseline analysis of the liver condition. This data is useful in order to identify patients with severe liver pathology, in whom rapid weight loss may be hazardous.

One closed suction drain on top of the duodenal stump is placed. Stomach stump and gallbladder are extracted through the supra-umbilical port. Fascia is closed on the supra-umbilical port only, with interrupted slowly-absorbable 0-stitches.

5.4 Operative technique (open approach)

Through an upper midline incision and after abdominal exploration, the first step is the intestinal measurement. The small bowel is measured backwards from the cecum to the ligament of Treitz and marking stitches are placed at 50 and 300 cm. It is very important that the small bowel is measured fully stretched, to make intestinal measurements reproducible in all hands. The ratio between the same small bowel fully loose and fully stretched is approximately 1-2. The small bowel is then transected at the 250 or 300-cm from the ileo-cecal valve and the ileal mesentery is sectioned in depth. The EEA can be done with any technique, bringing the BPL to the left side, and the AL to the right of the abdomen.

The distal gastrectomy is done, the duodenal stump is closed, the gallbladder is removed, and a wedge liver biopsy is obtained. We are used to cutting the stomach on a TA 90 linear stapler placed as oblique as possible, in order to compensate for the shortness of the ileal mesentery. As with the laparoscopic technique, the first two short gastric vessels on the greate curve are ligated and sectioned, whereas the dissection reaches up to the left gastric artery on the lesser curve. If the transaction is performed along those landmarks, a gastric volume of around 400 mL will be obtained. In any case, actual measurement of the gastric volume may be useful during the surgeon's learning curve.

The mesocolon is incised and the AL is brought into the supramesocolic space, checking for possible twist. Any technique can be used for the GEA. We prefer to do it end-to side, by cutting away the left corner of the gastric stump. The GEA is then anchored by two stitches to the mesocolic rent, to avoid intestinal kinkings and internal hernias. We always close the distal mesenteric defect and never the proximal.

The last maneuver is the final intestinal check, starting from the ICV, with the surgeon following the alimentary limb and the first assistant following the biliopancreatic limb. The fascia is closed with continuous suture. Drains are put as in the laparoscopic technique. A subcutaneous suction drain is controversial, and in any case used only for patients with very thick subcutaneous fat.

6. Postoperative care

Routine intensive care admission is not mandatory. Only very selected cases, with severe respiratory co-morbidities are sent to ICU in our practice, as discussed preoperatively with the anesthesiologist. Analgesia is performed via continuous infusion of opiates, NSAIDs and anti-emetics. Usually, 100 ml/h of fluids are administered in the first 24 hours.

The morning after the operation, bloods are checked, in particular, CPK and myoglobin, in order to detect rhabdomyolysis. With CPK values above 1000 U/dl, an aggressive protocol is started, with infusion of 200ml/h of saline, 100 mg bid fuorsemide, and 100 ml 8.4% HCO3-, with close monitoring of urine output, and daily check of CPK values. Hyperhydration is stopped when CPK values are steadily dropping and/or below 500 U/dl. The urinary catheter is usually kept until rhabdomyolysis is ruled out or resolved.

Preoperative antithrombotic prophylaxis is continued postoperatively up until 30 days from the operation. An upper GI radiology may be useful but is not mandatory. The naso-gastric tube is removed on the first postoperative day. Oral liquid diet and drain removal is performed on the third postoperative day. If tolerated, the diet is the progressed to soft diet first and, on the fifth or sixth postoperative day, to a free diet.

6.1 Patient instructions

Patients undergoing BPD must be aware that for the rest of their lives they will absorb minimal fat, (Gianetta et al, 1981; Scopinaro et al, 1987) little starch, sufficient protein, (Scopinaro et al, 1987;Gianetta et al, 1981) and nearly all mono- and disaccharides, short-chain triglycerides, and alcohol (i.e., the energy content of sugar, fruit, sweets, soft drinks, milk, and alcoholic beverages). They must also understand that when their body weight will have reached the level of stabilization the intake of these aliments may be varied as needed for individual weight adjustments.

All patients undergoing BPD have reduced appetite, early satiety and occasionally in association with epigastric pain and/or vomiting. These symptoms characterize the postcibal syndrome and are caused by rapid gastric emptying with subsequent distention of the postanastomotic loop and early food stimulation of the ileum. All these symptoms, which are more intense and last longer the smaller the gastric volume is, rapidly regress with time, most likely due to intestinal adaptation.

One year after operation, the appetite and the eating capacity are fully restored and the patient's mean self-reported food intake is one and a half times as much as preoperatively, independently of gastric volume.

Interestingly, the vasomotory phenomena characterizing the dumping syndrome are always absent after BPD, this indicating the lack of the specific receptors and/or the vasoactive gut hormones in the ileum that are thought to be implicated in the pathogenesis of dumping syndrome.

6.2 Home medication and diet program

Anti-thrombotic prophylaxis is continued up until 30 days after the operation. Patients are prescribed 30 to 60 mg/day of PPI for the first year. Oral multivitamin supplementation is started at discharge, whereas calcium and iron supplementation are begun one month after the operation, when appetite is starting to resume.

Patients are advised to assume a protein-rich diet, in small, frequent meals. In practice, little amounts of bread and pasta may be allowed, whereas milk, vegetables and fruit should be avoided for the first weeks. Frequent vomiting should be immediately reported, because it may be sign of gastric outlet obstruction due to early ulcer, and necessitate not only aggressive PPI treatment, but sometimes also endoscopic dilation, and, to prevent vitamin B1 deficiency and Wernicke's encephalopathy, aggressive vitamin B complex supplementation. In case of near-starvation, we administer intra-muscular vitamin B1 (as cocarboxylase) 38 mg, pyridoxin chloridrate (Vit. B6) 300 mg, and hydroxocobalamine (Vit. B12) 5000 mcg per day for five to ten days.

The first follow-up visit is scheduled 30-45 days after uncomplicated surgery. Although extremely rare, we encountered three cases of early postoperative porto-mesenteric vein thrombosis, which presented as severe, unexplained abdominal pain one week after an otherwise normal discharge. Unexplained severe, unremitting postoperative abdominal pain should therefore be investigated and not attributed to unspecific complaints.

6.3 Bowel habits

After full resumption of food intake, BPD subjects generally have two to four daily bowel movements of soft stools. Most have foul-smelling stools and flatulence. These phenomena, which can be reduced by modifying eating habits or by neomycin or metronidazole or pancreatic enzyme administration, tend to decrease with time along with a reduction of bowel movement frequency and increased stool consistency.

Diarrhea usually appears only in the context of postcibal syndrome, and then it rapidly disappears, being practically absent by the fourth month (Scopinaro et al, 1996). Sporadic acute gastroenterocolitis, generally lasting not more than a few days, may be observed, especially during the summer.

6.4 Follow/up care

Standard follow/up visits are scheduled at one, four and twelve months, and early afterwards. At each outpatient visit, the patient has to check blood exams according to Table. 5 Furthermore, any abnormal clinical condition is annotated and dealt with. In the long term, PTH and vitamins A, D, E are of particular importance, to prevent metabolic bone disease (with adequate oral calcium and parenteral vitamin D supplementation) and emeralopia (vitamin A deficiency)or peripheral neurpathy (vitamin E deficiency). Prompt detection of low values ensures early supplementation and absence of severe symproms.

Postoperative biochemical examination
Complete blood count
Total protein and differential
Na, K, Ca, Cl, P, Ma
Ferritin, Transferritin
Glycated haemoglobin
Serum cholesterol level, LDL, HDL, Triglycerides
Bilirubin (total and direct)
Fibrinogin
Transaminases
LDH, Gamma glutamile transferase
APTT
Amylase
Uricic acid
Creatinine
HBV and HCV marker only after 4th month
PTH, vitamine A, D, E one year after the operation and yearly after
Other exams; complete urine analysis

Table 5. Postoperative laboratory examinations and their frequency.

6.5 Standard supplementation would be

Oral calcium carbonate, 2g per day, increasing or decreasing the dose according to PTH values (and not, in the firs instance, serum calcium levels: Oral "over the counter" multivitamins.

Oral iron sulfate, 500 mg/day, and 1000 mg/day in menstruating women. Oral Vitamin A and E, 30.000 U and 70 mg per day, respectively. Vitamin D is administered on occasional basis when a deficiency is detected

7. Late complications

7.1 Specific complications

7.1.1 Anemia

The exclusion of the primary site for iron absorption in the alimentary tract causes this unavoidable complication. More rarely, the anemia is due to folate deficiency and, exceptionally, to vitamin B12 deficiency (Schilling test gives normal results short term after BPD (Scopinaro et al, 1987, Civalleri et al, 1982). Anemia appears only in BPD patients with chronic physiologic (menstruation) or pathologic (hemorrhoids, stomal ulcer) bleeding. Reflecting the cause of the anemia, most cases are **microcytic**, fewer are **normocytic**, and a few are **macrocytic**. The general incidence of anemia after BPD in our population would probably be around 40%, but chronic supplementation with iron, folate, or both can reduce its occurrence to less than 15%.

7.1.2 Stomal ulcer

BPD is a potentially ulcerogenic procedure. Since the beginning of experimental work in dogs,(Scopinaro et al, 1979) distal gastrectomy was preferred to gastric bypass (Mason & Ito, 1967) because it was thought to be more effective in preventing stomal ulcer (Storer et al, 1950) and because of the concern for the fate of the bypassed stomach (Scopinaro et al, 1992). The incidence of stomal ulcer was initially rather high (12.5% with the HH BPD) because of the large residual parietal cell mass. Considering only the ulcers diagnosed in the first two postoperative years in order to allow comparisons among groups, the incidence was successively reduced to 9.1% in the first 132 consecutive patients submitted to AHS BPD, simply due to the reduced stomach size (Civalleri et al, 1986).

Some changes of surgical technique, namely preserving as much as possible of the gastrolienal ligament with its sympathetic nerve fibers (Scopinaro et al, 1982) and shifting from end-to-end to end-to-side GEA, the latter being better vascularized and less prone to stenosis,(Gianetta et al, 1987) led to further progressive reduction (5.8% in the subsequent 650 cases). In the following group of 640 AHS patients operated on from January 1991 to March 1999 with a minimum follow-up of 2 years, thanks to H2-blockers' oral prophylaxis (Adami et al, 1991) during the first postoperative year in patients at risk (see below), started at the beginning of 1991, the incidence of stomal ulcer in the first 2 years was further reduced to 3.3%.

If the totality of stomal ulcers in the first two groups are considered, they were significantly more frequent in men (14.4%) than in women (5.2%). Differently than what was reported in

previous articles, (Scopinaro et al, 1992, 1996, 1998) the incidence of stomal ulcer appeared *unaffected* by alcohol consumption, increased in men (though not significantly) by cigarette smoking, and significantly increased, more in women than in men, by the association of alcohol and smoke. Stomal ulcers responded well to medical treatment (100% healing with PPI) and they showed no tendency to recur, provided the patient refrained from smoking. Endoscopic evidence of stomal ulcer was obtained in 52% of cases within the first postoperative year, in 26% of cases within the second year, and in 22% of cases, with progressively decreasing frequency, between the third and the tenth year.

However, it must be considered that (1) most patients diagnosed in the second and the third year were symptomatic already in the first one; (2) most patients diagnosed at a greater distance from the operation had been treated (one or more times) previously because of specific symptoms; (3) many patients once or repeatedly treated because of specific symptoms had refused endoscopy at all instances; (4) in some cases operated patients with no endoscopic diagnosis had received PPI therapy from their family doctors; and (5) with few exceptions, all patients with specific symptoms appearing after the second postoperative year were smokers, or smokers and drinkers. The consideration of all the above facts leads to the conclusions that (1) for BPD patients, not smokers or smokers/drinkers, the risk of developing a peptic ulcer is essentially confined to the first postoperative year and (b) the real incidence of stomal ulcer after BPD is certainly higher than that reported above.

For all these reasons, prophylactic PPIs should be given to all patients for the first year, and probably discontinued in the non/smokers only.

7.1.3 Bone demineralization

The duodenum and proximal jejunum are selective sites for calcium absorption. However, our study on calcium intestinal absorption showed a more than sufficient mean apparent absorption in the 15 subjects on a free diet. Moreover, intestinal absorption as an absolute value was positively correlated with the intake (Kendall rank test: $p < 0.03$), which means that, unlike fat and energy and similarly to protein, an increase of calcium intake results in increased absorption. Therefore, all of our patients are encouraged to maintain an oral calcium intake of 2 g/day (with tablets supplementation, if needed), while the daily requirement of vitamin D, as well as of all other vitamins and trace elements, is contained in a multi-integrator that all patients are recommended to take for all life.

When natural history of bone disease was investigated by us in obese patients and operated subjects not taking any supplementations 1–10 years after BPD, histomorphologic signs of mild to severe bone demineralization (cross-sectional study on 252 transiliac bone biopsies after double-labeling with tetracycline, 58 of which preoperatively) were present in 28% of the obese patients and 62% of the operated subjects. Slightly low levels of serum calcium and high levels of alkaline phosphatase were found in about 20% of the subjects in that study, with no significant differences between obese patients and operated subjects or between operated subjects with and without bone alterations.

Serum magnesium, phosphorus, and 25-hydroxyvitamin D levels were essentially normal both prior to and after operation. The prevalence and severity of metabolic bone disease

(MBD) increased after BPD until the fourth year [prevalence: preop. 16/58, at 4 year 15/21, chi-square test p < 0.001; severity (subjects with moderate or severe MBD): preop. 7/58, at 4 year 8/21; chisquare test p < 0.01], at which point they tended to regress.

Long-term (6–10 year) mineralization status was not significantly worse than that observed before operation. Patients with the most severe preoperative alterations, i.e., the older and the heavier patients, showed a sharp improvement in bone mineralization status compared to their preoperative status (prevalence of moderate or severe MBD in patients over 45-year-old: preop. 25%, at 1–2 year 29%, at 3–5 year 33%, and at 6–10 year 11%; in patients with an IEW greater than 120% these values were, respectively, 24%, 28%, 53%, and 14%) (Scopinaro et al, 1987, Compston et al, 1984, Adami et al, 1987). The histomorphology data were in total agreement with the clinical findings. Bone pain attributable to demineralization (with prompt regression after calcium, vitamin D only when needed, and diphosphonate therapy) was observed in 6% of patients, generally between the second and fifth postoperative years (maximum prevalence: 2.4% during the fourth year) and more rarely on long term(10–20 years).

The pathogenesis of bone demineralization in obese patients is unclear. The bone problems caused by BPD do not seem to differ substantially from those reported in 25%–35% of postgastrectomy subjects with duodenal exclusion for peptic ulcer (Williams, 1964, Eddy, 1984, Fisher, 1984) and in one-third of patients with gastric bypass for obesity (Crowley et al, 1986). The mechanism is very likely a decreased calcium absorption causing an augmented parathyroid hormone (PTH) release which is generally sufficient to normalize serum calcium level at the expense of bone calcium content. During the first postoperative years, the adverse effect of reduced calcium absorption seems to prevail over the beneficial one of the weight loss, whereas the opposite happens at long term, this being more evident in the subjects with the most severe preoperative alterations.

Recently, it has been suggested that low albumin level is also implicated in the pathogenesis of MBD after BPD (Marceau et al, 2002). In our experience, oral calcium supplementation seems to be able both to prevent and to cure bone alterations caused by BPD, monitored by computerized bone mineralometry. Still, great differences in calcium requirement and metabolism exist among populations and individuals in the same population. Vitamin D synthesis in the skin at different latitudes probably also plays a major role. It is important to remember that parenteral vitamin D supplementation should not be used in the treatment of MBD unless low serum levels have been documented. In fact, an excess of vitamin D can cause bone damage similar to that caused by its deficiency.

7.1.4 Neurological complications

Peripheral neuropathy and Wernicke's encephalopathy, early complications caused by excessive food limitation(Primavera et al, 1987) have now totally disappeared (none in the last consecutive 1,969 operated subjects of the total series with a minimum follow-up of 1 year) because of prompt administration of large doses of thiamin to patients at risk, i.e., those reporting a very small food intake during the early postoperative weeks.

A more insidious cause of peripheral neuropathy is vitamin E deficiency, which can lead to various degrees of impairment, up to ataxia in the most severe cases. Patients reporting

paresthesia, especially if long-term after the operation and not well supplemented, should be checked for vitamin E levels and promptly corrected in necessary.

7.1.5 Protein malnutrition

Protein malnutrition (PM), with its classic symptoms, is the most severe possible complication of BPD, and it may require hospitalization with parenteral nutrition. It can be early episodic (1st postoperative year) due to patient non-compliance with alimentary rules to be followed during the first postoperative months, or late recurrent due to insufficient protein intake or intestinal absorption, generally requiring elongation of the CL. Protein absorption after BPD corresponds to ~70% of a meal containing 60 grams of protein.

Unfortunately, our study on protein absorption also showed a five-fold increase in endogenous nitrogen loss, which doubles daily protein requirement and accounts for possible occurrence of PM. This complication in our hands was frequent many years ago, when, with the aim of obtaining greater weight loss, we explored the formidable power of the small stomach volume. The evolution of BPD, essentially consisting of increasing the stomach volume and the length of the AL, eventually led to near disappearance of PM, both in the early and recurrent form. However, as the operation is active for life, sporadic PM can occur at any time after BPD, if there is prolonged diarrhea or reduced food intake for any reason.

Therapy for early or late PM must be aimed at eliminating PEM and restoring normal nutritional status, with parenteral feeding that includes both the nitrogen and the energy necessary to restore the amino acid pool, reestablish the anabolic condition, and resynthesize deficient visceral protein.

In addition to the increased endogenous nitrogen loss, with its impact on daily protein requirement, important phenomenon acting in the same direction is the overgrowth of colonic bacterial flora. Overgrown bacterial flora, the synthesis of which partly or totally occurs at the expense of alimentary protein escaped to absorption in the small bowel, reduces protein absorption by the colonic mucosa, thus increasing protein malabsorption and protein requirement.

Correlation between stomach volume (gastric restriction) and protein malnutrition, At a remote phase of BPD development, in an attempt to accelerate and increase the weight loss, we drastically reduced mean gastric volume to about 150 mL, obtaining, in addition to excellent weight reduction (near 90% of the IEW at 2 years), a catastrophic approximate 30% incidence of PM with 10% recurrence rate .With the aim of decreasing the PM incidence without losing the benefit of the small stomach, the gastric volume was adapted to the patient's initial EW (AHS BPD, June 1984). In fact, the original philosophy of the AHS was to confine the risk of PM to patients who required greater weight loss. It resulted in a 17.1% incidence of PM with 8.3% recurrence, and the mean weight reduction remained at a very satisfactory 77% of the IEW (initial 192 AHS BPD patients with a minimum follow-up of 2 years), the higher weight of stabilization being evidently due to the larger mean stomach volume (about 350 mL) with the consequent slower intestinal transit and greater energy absorption.

Further mean increases in gastric volume to about 400 mL, and of alimentary limb length (up to 250 or 300 cm) led to a further decrease of incidence in protein malnutrition to the present 3% (1% recurrent cases). Interestingly, there is a strong correlation between stomach volume, stabilization weight and protein malnutrition incidence. The smaller the gastric volume, the lower the stabilization weight, and the higher the likelihood of protein malnutrition. These data are an explicit warning against trying to combine gastric restriction with malabsorption.

7.2 Minor or rare late complications

Among the 2756 AHS BPD patients with a minimum follow up of 2 years, the following minor or rare complications were observed or reported: 284 (9%) cases of impairment or appearance of hemorrhoids, 110 (4%) cases of anal rhagades, 55 (2%) cases of perianal abscess, 88 (3.2%) cases of acne, 61 (2.2%) cases of inguino-perineal furuncolosis, 207 (7.5%) cases of night blindness, 4 cases of lipothymias from hypoglycemia, 2 cases of transient dumping syndrome, 1 case of bypass arthritis, and 1 case of gallstone ileus.

These complications showed a decreasing incidence in our population of operated patients. Anyway, they occur more rarely as time passes and tend to disappear in the long term. Halitosis after BPD could be due either to food stagnation in a virtually achloridric stomach, which can be avoided by correct execution of the GEA, or to pulmonary expiration of ill-smelling substances resulting from malabsorption, the oral administration of pancreatic enzymes being of use in these cases. This unpleasant side effect has also become less common in our series, currently affecting less than 5% of the operated patients.

BPD causes oxalate hyperabsorption, but not hyperoxaluria, though oxalate urinary excretion in the operated patients is significantly higher than in controls (Hofmann et al, 1981). The procedure can then be considered a remote cause of kidney stone formation, keeping in mind that not even hyperoxaluria can cause this complication in the absence of cofactors, the first of which is decreased urinary volume from dehydration. The incidence of kidney stones in our series (5/1,804 or 0.3%) does not differ from that of the general population. Thirty-two needle kidney biopsies obtained at long-term relaparotomy in BPD patients failed to demonstrate any microscopic or ultrastructural alterations (unpublished data: study in cooperation with Dr. Thomas Stanley, VA Hospital, Los Angeles, CA, 1984).

7.3 Late mortality

Specific late mortality is essentially attributable to the consequences of untreated protein malnutrition in lately presented or inadequately treated cases.

8. Revision of BPD

8.1 Options available

All specific late reoperations after biliopancreatic diversion (BPD) consisted of;
Elongations of the common limb (CL) which can be done either:
Along the biliopancreatic limb or
Along the alimentary limb
Restorations of intestinal continuity with either

Sparing the duodenum from the alimentary continuity
Putting the duodenum in the alimentary continuity

8.2 Indications to revision of BPD are

1. Recurrent protein malnutrition with or without poor protein intake
2. Excessive Weight loss with normal food intake
3. Excessive Foul Smelling of the stool and Flatulence
4. Diarrhea
5. Intractable severe bone demineralization
6. Occurrence of a disease whose consequences would be worsened by malabsorption
7. Intolerance of the operation

The revision is generally implemented to correct an excess of effect of the original operation, and, because it entails a permanent modification of intestinal absorption, it is critical to ensure that intestinal adaptation mechanisms have been substantially completed, which require at least 1 year. If the problems persist and reoperation is then indicated, the risk of having a premature reoperation with a consequent overcorrection and undue weight regain is minimal.

8.3 Post-revision physiology

Protein, simple sugar and starch absorption depend on the total bowel length between the gastro-enterostomy and the ileo-cecal valve. Fat and bile acid absorption depend on the length of the common limb. Bile acid absorption also depends on the length of ileum which is exposed to bile acid transit.

a. Elongation of the common limb along the biliopancreatic limb leads to an increase of the total intestinal length from the gastroenterostomy to the ileocecal valve, which in turn increases protein, energy and water absorption.
b. Elongation of the common limb along the alimentary limb leads to an increase of bile salt absorption without a direct increase in protein, energy and water absorption, because the total bowel length between the gastroenterostomy and the ileo-cecal valve remains unchanged. Energy absorption increase is also minimal, because only fat absorption is increased.
c. Restoration of intestinal continuity with a bypass of the duodenum allows the resumption of normal protein-energy absorption, still partially preserving both the specific effects of BPD on glucose and cholesterol metabolism.
d. Restoration of the intestinal continuity with re-canalization of the duodenum in the alimentary pathway will sacrifice the effect on glucose metabolism; the effect on cholesterol metabolism is preserved if the alimentary limb is kept interposed between the stomach and the duodenum, because it still entails a reduction of the ileum exposed to bile acid transit (Figure 34–5).

8.4 Problem solving

A recurrent Protein malnutrition (PM) (with or without additional problems) is the condition to be cured in the vast majority of cases. In this case, PM is due to insufficient protein intestinal absorption, either *absolute* (insufficient absorption capacity per unit of intestinal

length, too rapid intestinal transit due to excessively little stomach) or *relative* (insufficient protein content of ingested food, excessive loss of endogenous nitrogen).

In both cases the aim of the surgical revision is to increase protein absorption, and this (keeping in mind the physiology of the operation), would not be obtained by elongating the CL along the alimentary one. Since, as said above, protein absorption after BPD substantially depends on the total intestinal length from the GEA to the ICV, the elongation of the CL for correction of a recurrent PM must be performed at the expense of the biliopancreatic limb, the length which in our experience has proven effective in all cases being 150 cm, with the result of a total of 400 cm of small bowel in the food stream (Fig 5).
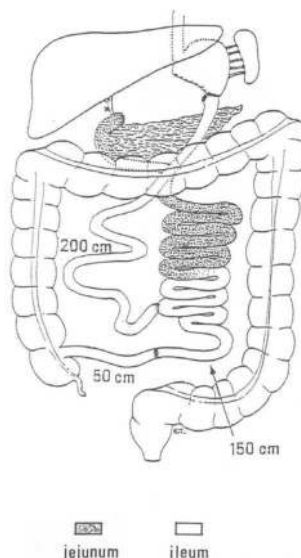
Fig. 5. Elongation of the CL along the BPL.

Excessive weight loss in presence of a normal food intake, problems of *foul-smelling stools and flatulence and diarrhea* due to excessive fluid intake are conditions which can be corrected by elongation of the common limb along the biliopancreatic limb.

Rarely, *diarrhea* is due to excessive reduction of ileal bile salt absorption. This condition can be easily diagnosed by cholestyramine administration, and it represents the only indication to the elongation of the CL along the alimentary one, 100 cm being sufficient in our experience (Fig. 6).

Restoration of the intestinal continuity is specifically indicated in presence of a recurrent PM and/or an excessive weight loss due to permanence of the food limitation effect with or without poor protein intake. The goal in these cases is to restore a normal intestinal absorption capacity in a subject who will maintain his/her weight reduction because of the permanently reduced food intake. This can be obtained with different operations, ranging from a simple high side-to-side enteroenterostomy to the complete reconstruction of the gastrointestinal tract, with a 50-cm ileal loop being interposed between the stomach and the duodenum (Fig. 7).
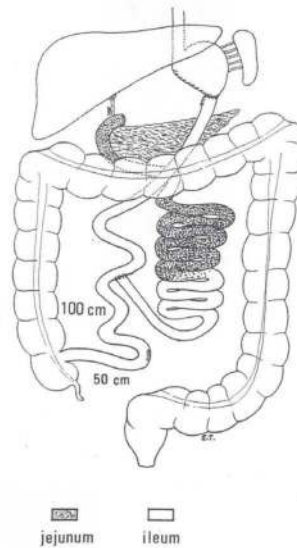
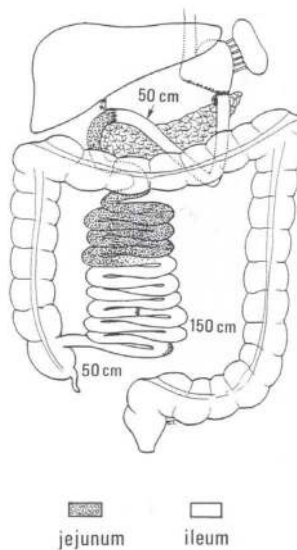Fig. 6. Elongation of the CL along the AL.

Fig. 7. Full restoration of intestinal continuity.

We prefer to section the alimentary limb (AL) immediately proximal to the enteroenterostomy (EEA) and join the ileal stump to the jejunum, immediately distal to the ligament of Treitz. This type of restoration allows the resumption of a normal protein-energy absorption, still partially preserving both the specific effects of BPD on glucose and cholesterol metabolism (Fig. 8).
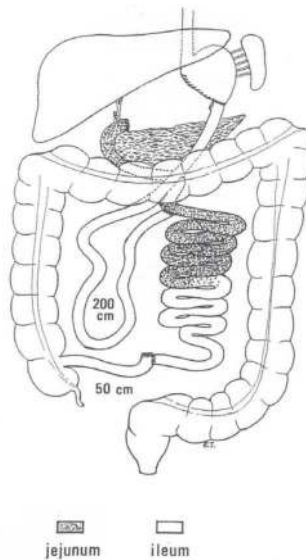
Fig. 8. Restoration bringing the AL to the ligament of Treitz.
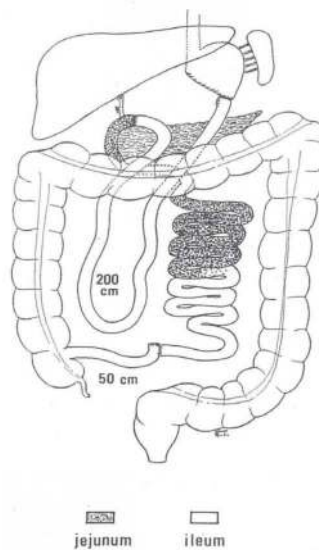
Fig. 9. Restoration bringing the AL to the duodenum.

Restoration may be necessary if **a disease occurs whose consequences would be worsened by malabsorption**, e.g., liver cirrhosis, nephrotic syndrome, chronic inflammatory bowel disease, malignancy, or psychosis. It may also be requested by the BPD subject instead of the elongation after a long period of recurrent PM, or for different reasons, e.g., intolerance of the stool/gas problems, or psychological intolerance of the environmental problems

originated by the changed body shape, or what we simply call **"intolerance of the operation."**

Only in case of **intractable severe bone demineralization**, or of moderate bone demineralization associated with a condition that indicates the restoration, we accept the sacrifice of the effect on glucose metabolism by putting the duodenum in the alimentary continuity, which can be accomplished as shown in Figure 34–3, or, if the effect on cholesterol metabolism is to be preserved, by keeping the entire AL interposed between the stomach and the duodenum (Fig. 9).
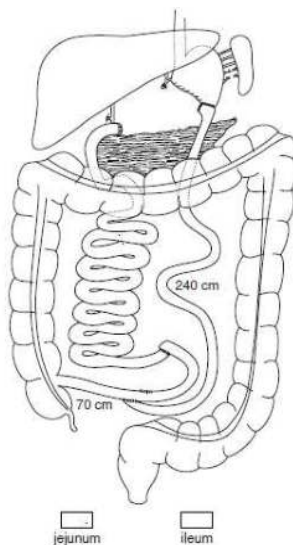
Fig. 10. Shorting of both alimentary and the common.

Exceptionally, a shortening of the intestinal limbs may be considered. The most common cause for **weight regain** after BPD is excessive intake of simple sugar. Only if this type of **patient's noncompliance** can be reasonably excluded, an excess of the adaptive phenomena leading to late energy absorption increase may be suspected. A preliminary measurement of alimentary protein intestinal absorption is, in our opinion, mandatory. More than 90% absorption on the one hand confirms the excessive adaptation, on the other hand it means that there is a wide margin for shortening without causing excessive protein malabsorption.

In two cases, both with 370-cm AL, we resected the proximal part of the AL exceeding the 240cm which, as a mean of the intestinal measurements taken at late reoperations for any cause, result after adaptation, with redo of GEA. Since these two patients failed to lose weight, we thought it could be due to the highly consuming small bowel removed, which compensated for the decreased absorption. In a third case, with 520-cm AL and 80-cm CL, we sectioned the AL 240 cm distal to the GEA, detached the biliopancreatic limb (BPL) from the distal ileum, and then anastomosed the BPL to the distal stump of the AL section and the proximal stump to the distal ileum, thus creating a CL of 70 cm (mean CL length after

adaptation), as shown in Figure 9. The patient slowly lost all the excessive weight previously regained. The same operation was subsequently successfully used in another case that had been submitted to BPD elsewhere and had had minimal weight reduction. Both the CL and the AL were excessively long, and this most probably was due to wrong intestinal measurement, even if a rapidly occurring excessive intestinal adaptation could not be ruled out.

8.5 Timing

The total number of revisions in the last consecutive 1000 AHS-AHAL BPD patients with a minimum follow-up of 1 year was 10 or 1.0%, 4 (0.4%) being elongation of the CL and 6 (0.6%) restorations of intestinal continuity. The latter cases presented with ensuing medical problems, unrelated to the operation, which necessitated resumption of normal protein absorption

8.6 Secondary effects

All problems of recurrent PM, excessive weight loss, diarrhea, stool/gas, and bone demineralization permanently disappeared after revisions. While no recurrences of hypercholesterolemia were ever observed, there were two cases of mild hyperglycemia in previously diabetic patients after elongation.

8.7 Weight regain

Since protein and starch digestion/absorption occur in the entire small bowel between the GEA and the ICV, and fat absorption in the intestinal segment between the EEA and the ICV, the elongation of the CL along the BPL which is necessary to increase protein absorption also causes an increase of starch and fat absorption. This obviously results in a higher energy absorption threshold, which explains the restabilization at a higher body weight of the subjects undergoing elongation of the CL for recurrent PM and/or excessive weight loss.

The subjects who were elongated along the AL because of diarrhea from excessive bile salt malabsorption had and maintained a food intake equal to the preoperative one, and they did not gain any weight in a 6- and 11-year follow-up, respectively.

The weight changes after BPD revisions indicate that, when the indication was correctly given by us according to the physiology of the operation, the revised subjects had a moderate weight regain with restabilization, the success being maintained, while in the other cases they regained weight progressively toward failure.

A final consideration regards the mean weight loss at the time of revision, which was in most cases lower than the average in our series. Any degree of weight reduction can be obtained with BPD, but, as we learned at our expenses, the greater the mean weight loss, the greater the number of problems. Unless a surgeon has an experience sufficient to enable him to tailor the operation on each single subject, any mean reduction of the IEW greater than 70% should be considered potentially dangerous. Weight maintenance, not weight loss, is the real magic of BPD.

9. Revision to BPD

The superior results of BPD both in weight maintenance and control of component of metabolic syndrome make this operation a good choice for revision of failed other types of bariatric procedures. Furthermore, revision to BPD often enables the surgeon to avoid scarred tissue high up in the stomach, thus simplifying reoperation and reducing perioperative morbidity.

Revisional Bariatric surgery is technically challenging and becoming more common due to the rapid increase in patients undergoing surgery for morbid obesity. Unfortunately, there is no sufficient evidence to help the surgeon deciding which revisional procedure to choose based. There are several factors that need to be considered when unsatisfactory outcomes after bariatric surgery are obtained.

The extent of weight loss may not be the only factor to be considered while evaluating the results of a bariatric restrictive procedure, because the major goal of bariatric procedures is the cure or control of comorbidities along with the weight loss. With this idea in mind, a borderline weight loss with a successful control of comorbidities (eg, easier hypertension or diabetes control, disappearance of articular pain or swelling, cure of sleep apnea, etc.) in a high-risk patient without surgery-related complications should probably be considered a good outcome, and the patient could avoid additional surgery. These issues should be clarified with the patient when discussing risks and benefits of the reoperation.

Both patient and surgeon need to be aware that a new operation will be a difficult task, and realistic goals have to be presented to the patient. There is a higher risk of complications, and a possibility that a new bariatric procedure may not be completed in one intervention as planned owing to inflammation, scarring, and bleeding. Patients must know that more than one intervention may be required. Unlike gastric restrictive revisional surgery, revision to BPD usually does not impair final weight loss results.

9.1 Indications for revision

1. Insufficient weight loss.
2. Weight regain.
3. Complications of previous the procedure.

9.2 Patient evaluation

Surgeons should consider that failure of specific restrictive bariatric procedures is owing to many factors. One of these factors is patient's compliance with the dietary program, and when required, with behavioral modifications after the primary procedure. Patients may not need a second surgery but a close follow-up and behavior counseling before deciding on a more drastic intervention.

9.3 Approach to the patient

Detailed evaluation of the preoperative data of the these patients, operative notes and postoperative BMI and complications through a Pre-operative check-list;

1. History and Physical examination;
   BMI
   Symptoms of dysmotility
   Symptoms of acid hypersecretion
   Screening for eating disorders
   Psychological evaluation if positive
2. Review old operative notes and videos if possible
3. Barium swallow to document anatomy
4. EGD if patient has symptoms of GERD

Imaging and endoscopic studies are necessary to study the problem and give an accurate map of the existing anatomy. As restrictive bariatric procedures are practiced following different techniques with modifications of some of the originally described steps, it is advisable to read the operative note or to discuss the case with the surgeon who carried out the index surgery.

9.4 General considerations

Revisional bariatric surgery is technically demanding. There are technical aspects common to any reoperation after gastric surgeries that need to be taken into account to minimize complications.

Adhesions are likely to be present and they may be anywhere. Therefore, access to the cavity should be attained by open technique, or alternatively using an optical trocar, trying to prevent visceral injuries.

Once in the cavity, following ports should be placed in an order that allows dividing the adhesions from the best point of view. In addition, liver retraction should be done delicately, and only after its adhesions have been addressed, because any sudden movement or excessive force could produce a tear, and bleeding will obscure the surgical field, reducing visibility and causing delays.

Division of gastric vessels should only be carried out after the gastric vascular supply has been assessed. Restrictive procedures usually preserve lesser curvature vessels, and therefore these should be preserved at all costs. If the left gastric artery or its branches need to be cut at some stage, the condition of the stomach must to be carefully observed to rule out ischemia leading to gastric resection.

Intraoperative endoscopy is a valuable tool when during the procedure it is difficult to accurately characterize the anatomic changes seen during the laparoscopy. It should be scheduled beforehand to avoid delays in a possibly long intervention.

9.5 Open versus laparoscopic approach

Laparoscopic surgery has had a major impact on obesity surgery the last decade. All bariatric procedures have been proven to be technically feasible via laparoscopy (Buchwald et al, 2004). There is also evidence that the laparoscopic approach is advantageous, since it is associated with less perioperative morbidity and faster recovery (The Society of American Gastrointestinal and Endoscopic Surgeons (SAGES) Guidelines Committee , 2008; Weller

Rosati, 2008) . Laparoscopic approach to malabsorption procedures, such as the BPD or the duodenal switch operation (DS), is more complex and technically difficult (Scopinaro et al, 2002; Baltasar et al, 2002; Weiner et al, 2004). Several reports have demonstrated the efficacy of laparoscopic revisional bariatric surgery (Gumbs et al, 2007). However, laparoscopic re-do surgery, after failed primary bariatric procedures, should be handled cautiously. Van Dessel et al, 2008 reported recently that the threshold for conversion to open approach should be low.

In patients after failed or complicated bariatric operations, it is the surgical management that eventually matters and not the approach.

9.6 Which type of revision? And why BPD?

Surgeons differ in their management of failed bariatric procedures, depending on their individual experience and resultant choice of surgical operation or operations. Revision of failed gastric restrictive operation is a common problem today, and is likely to increase in the near future (Sarr, 2007). In our opinion, the conversion of a restrictive operation to a malabsorptive one appears for some patients as the only alternative to cope with poor weight loss results. Such conversions, after unsuccessful weight loss, have been previously described (Fox, 1991; Keshishian et al, 2004; Di Betta et al, 2006). Since gastric bypass relies primarily on restriction, a different surgical approach, based essentially on malabsorption was employed as the final option in this cohort of patients with multiple previous bariatric interventions. In a recent report, (Topart et al, 2007) demonstrated that BPD with duodenal switch resulted in greater weight loss compared to Roux-en-Y gastric bypass after failed gastric banding. However, BPD-DS resulted in a higher early complication rate. When compared to RYGB, BPD and BPD-DS procedures are suggested to create superior weight loss and more accurately, superior weight loss maintenance (DeMaria, 2004).

In a recent analysis, it was demonstrated that obesity surgery results depend on the performed technique. BPD was proven to be the only operation that kept excellent weight results in time but unfortunately, with increased morbidity (Gracia et al, 2009). So far, no adequate prospective trial has been done to adequately answer the question of "which revisional bariatric procedure to do" in the setting of inadequate weight loss or excessive weight regain (Gumbs et al, 2007).

Furthermore, failed RYGB represent a subset of particularly challenging cases, in which only BPD can provide adequate results. Conversion from RYGB to biliopancreatic diversion with duodenal switch (BPD-DS) might provide the most durable weight loss of all revision procedures currently available. Revision to BPD-DS can be done laparoscopically in 1 or 2 stages and involves 4 anastomoses: gastrogastrostomy, duodenoileostomy, ileoileostomy, and jejunojejunostomy (to reconnect the old Roux limb).

10. Duodenal switch

Duodenal Switch (DS), is the malabsorptive procedure that is usually performed in North America for treatment of morbid obesity. In 1987, DeMeester, in some experimental studies conducted on biliary gastritis, showed that keeping even a short duodenal limb could

significantly reduce the incidence of anastomotic ulcers. By transferring these observations to bariatric context, the goal of the Duodenal Switch (DS) variation of Biliopancreatic Diversion (BPD) proposed by Douglas Hess and Picard Marceau was to reduce the complications correlated with Scopinaro's original procedure while maintaining its long-term effectiveness on weight loss and co-morbidities resolution.

10.1 Modifications

The modifications adopted in DS are: modifying the distal gastrectomy of the BPD with a vertical gastrectomy along the greater curvature (sleeve gastrectomy). Further modification foresaw the lengthening of the common channel to 100cm, thus doubling the length of the absorbent common ileal segment, for greater control of the number of daily bowel movements. Some procedures that are consensual to BPD, like cholecystectomy, appendectomy and liver biopsies were routinely performed by different authors even in the DS variation. These modifications have led, according to the same authors, to the reduction in the incidence of dumping syndrome, anastomotic ulcer, and proteins malabsorption.

Analysing the results of open BPD vs. DS, Marceau reports fewer daily evacuations, less diarrhea, vomiting and bone pain following DS associated with higher serum levels of ferritin, calcium and Vitamin A.

Whether this report is simply the result of a progressive learning curve (standard BPD had been adopted in the early experience, and BPD with DS had been further developed in later years), or if the longer length of the common channel, by increasing bile acid absorption, and thus lowering the degree of steatorrhea, leads to fewer bowel movements per day, and, consequently, to less endogenous nitrogen loss, has never been studied in a prospective fashion. In fact, most recent publications report very similar results and side effects for both operations. Just as for standard BPD, BPD- DS primarily induces malabsorption of fats due to the diversion of bile and pancreatic enzymes to the common limb. Therefore, the length of the alimentary limb and the common limb is of the fundamental importance; if the common channel is too long, the malabsorption of fats might not be sufficient to guarantee an adequate weight loss over the long term; an excessively long alimentary limb leads to insufficient weight loss for insufficient malabsorption of starch.

Duodenal switch might nonetheless be a better option in revision of failed sleeve gastrectomies, as the latter operation had in fact been introduced in clinical practice as the first stage of BPD-DS in super-superobese patients.

10.2 Peri- and postoperative complications

10.2.1 Fistula

Suture line dehiscence can occur at the gastric suture after sleeve gastrectomy, at both anastomoses and at the duodenal stump. The incidence of suture-line leak after sleeve gastrectomy ranges between 1.3% and 4.6% (Hess, 1998, Lee et al, 2007). The critical areas for leak are the top of the suture line and the transition point between sequential cartridges. To prevent leak many authors suggest reinforcing the long suture line with buttress material, a running suture or fibrin glue. The suture line fistula can be managed by percutaneous

drainage plus total parenteral nutrition and antibiotics associated, in selected cases, with endoscopic stenting.

Anastomotic leak seems to have the same incidence rate in LapBPD-DS as in the open series (2.5%) (Marceau et al, 1998, Ren et al, 2000, Lee et al, 2007). The clinical presentations involves tachycardia (heart rate >120 beats /minute), fever, abdominal pain, hypotension and mental deadness. To make the correct diagnosis an upper GI X-ray can be useful, but a negative contrast X-ray study does not exclude a fistula.

Therefore, a spiral angioCT scan is the most accurate diagnostic tool in doubtful cases. In case of low output fitulas and when there are no signs of haemodynamic instability, management could be conservative. Failure of nonoperative management or signs of peritonitis, a laparoscopic or laparoscopic reoperation is indicated. Options includes suture of the anastomotic dehiscence, wide drainage and/or with the creation of a jejunostomy in the biliopancreatic limb for decompression and for enteral nutrition and supportive care. Compared to the most recent series of standard BPD, fistula incidence after BPD-DS seems higher.

11. Conclusions

BPD yields the best results obtained in the treatment of obesity and metabolic syndrome. The reasons why, although in use for more than 35 years, this operation still has relatively minimal diffusion in the world are many, and they have little to do with its effectiveness and safety. However, this is to be considered a favorable event: BPD would cause dramatic damages if improperly used, and this would certainly happen if the operation had a rapid diffusion among the average bariatric surgeon. For the same reason, being able to master BPD and its complications, and understanding its pathophysiology, is extremely important for a committed bariatric surgeon, because, *at the very least*, it represents a very powerful and effective weapon in difficult revisional cases and in the metabolically highly compromised patient. To be limited to a slowly increasing number of good hands is the main guarantee of safety for biliopancreatic diversion.

12. References

Adami GF, Compston JE, Gianetta E, et al.(1987) Changes in bone histomorphometry following biliopancreatic diversion. In: *Proceedings of the III International Symposium on Obesity Surgery,* Genoa, Italy, September 1987.

Adami GF, Gandolfo P, Esposito M, et al. (1991). Orally-administered serum ranitidine concentration after biliopancreatic diversion. Obes Surg, 1, 293–294.

Adami GF, Cordera R, Camerini G, et al. (2003) Recovery of Insulin Sensitivity in Obese Patients at Short Term after Biliopancreatic Diversion.J Surg Res.113, 217–221.

Adami GF, Murelli F, Briatore L, et al. (2008). Pregnancy in Formerly Type 2 Diabetes Obese Women Following Biliopancreatic Diversion for Obesity. Obes Surg.18, 1109–1111.

Ast M, Frishman WH. (1990) Bile acid sequestrants. J Clin Pharmacol.30, 99–106.

Baltasar A, Bou R, Miró J, et al. (2002)Laparoscopic biliopancreatic diversion with duodenal switch: technique and initial experience. Obes Surg.12, 245–248.

Beil U, Crouse JR, Einarsson K, et al. (1982) Effects of interruption of the enterohepatic circulation of bile acids on the transport of very low density-lipoprotein triglycerides. Metabolism.31, 438–444.

Borg CM, le Roux CW, Ghatei MA, et al. (2007) Biliopancreatic Diversion in Rats is Associated with Intestinal Hypertrophy and with Increased GLP-1, GLP-2 and PYY levels. Obes Surg.17, 1193–1198.

Brandi LS, Santoro D, Natali A, et al. (1993) Insulin Resistance of Stress: Sites and Mechanisms. Clin Sci (Lond).85, 525–535.

Briatore L, Salani B, Andraghetti G, et al. (2008) Restoration of Acute Insulin Response in T2DM Subjects 1 Month after Biliopancreatic Diversion. Obesity.16, 77–81.

Buchwald H, Avidor Y, Braunwald E, et al. (2004)Bariatric surgery: a systematic review and meta-analysis. JAMA. 292, 1724– 1737.

Buchwald H, Estok R, Fahrbach K, et al. (2009) Weight and Type 2 Diabetes after Bariatric Surgery: Systematic Review and Meta analysis. Am J Med.122, 248–256.

Civalleri D, Scopinaro G, Gianetta E, et al. (1982)Assorbimento della vitamina B12 dopo bypass biliopancreatico per l'obesit`a. Min Diet Gastroenterol. 28, 181–188.

Civalleri D, Gianetta E, FriedmanD, et al. (1986): Changes of gastric acid secretion after partial biliopancreatic bypass. Clin Nutr, 5 (Suppl), 215– 220.

Compston JE,Vedi S, Gianetta E, et al. (1984) Bone histomorphometry and vitamin D status after biliopancreatic bypass for obesity. Gastroenterology, 87, 350–356.

Crowley LV, Seay J, Mullin GT Jr, et al. (1986). Long term hematopoietic and skeletal effects of gastric bypass. Clin Nutr, 5(Suppl),185–187.

DeMaria EJ. (2004) Is gastric bypass superior for the surgical treatment of obesity compared with malabsorptive procedures? J Gastrointest Surg, 8, 401–403.

Di Betta E, Mittempergher F, Di Fabio F, et al. (2006) Duodenal switch without gastric resection after failed gastric restrictive surgery for morbid obesity. Obes Surg, 16, 258–261.

Doyle ME, Egan JM. (2007). Mechanisms of Action of Glucagone-like Peptide 1 in the Pancreas. Pharmacol Ther,113, 546–593.

Eddy RL: (1984) Metabolic bone disease after gastrectomy. AmJMed, 8, 293–302.

Farilla L, Bulotta A, Hirshberg B, et al. (2003) Glucagon-like Peptide 1 Inhibits Cell Apoptosis and Improve Glucose Responsiveness of Freshly Isolated Human Islets. Endocrinology,144, 5149– 5158.

Fisher AB: (1984). Twenty-five years after Billroth II gastrectomy for duodenal ulcer.World J Surg 8:293–302,.

Forbes GB. (1987). Human Body composition; Growth, aging and nutrition and activity. Springer Verlag New York, 1987.

Fox SR. (1991) The use of biliopancreatic diversion as a treatment for failed gastric partitioning in the morbidly obese. Obes Surg, 1, 89–93.

Gianetta E, Civalleri D, Bonalumi U, et al. (1980). Studio dell'assorbimento proteico dopo bypass bilio-pancreatico per l'obesit`a. MinDiet e Gastr, 26, 251–256.

Gianetta E, Civalleri D, Bonalumi U, et al. (1981). Studio dell'assorbimento lipidico dopo bypass biliopancreatico per l'obesit`a. Min Diet e Gastr, 27, 65–70.

Gianetta E, Friedman D, Adami GF, et al. (1985). Effects of biliopancreatic bypass on hypercholesterolemia and hypertriglyceridemia. In: Proceedings of the Second Annual Meeting of the American Society forBariatric Surgery, Iowa City, IA, June 1985.

Gianetta E, Friedman D, Adami GF, et al. (1987). Present status of biliopancreatic diversion (BPD). In: Proceedings of the Third International Symposium on Obesity Surgery. Genoa, Italy, September, 1987.

GriffenWO, Printen KJ (eds.). (1987) Surgical Management of Morbid Obesity. Marcel Dekker, New York.

Guidone C, Manco M, Valera-Mora E, et al. (2006) Mechanisms of Recovery from Type 2 Diabetes after Malabsorptive Bariatric Surgery. Diabetes,55, 2025–2031.

Gumbs AA, Pomp A, Gagner M. (2007) Revisional bariatric surgery for inadequate weight loss. Obes Surg, 17, 1137–1145.

Halverson JD, Scheff RJ, Gentry K, et al. (1980). Jeunoileal bypass. Late metabolic sequelae and weight gain. Am J Surg, 140, 347–350.

Hess DW. (1998) Biliopancreatic Diversion with a Duodenal Switch, Obes Surg, 8, 267-282.

Hofmann AF, Schnuck G, Scopinaro N, et al. (1981) Hyperoxaluria associated with intestinal bypass surgery for morbid obesity: Occurrence, pathogenesis and approaches to treatment. Int J Obes, 5 , 513–518.

Keshishian A, Zahriya K, Hartoonian T, et al. (2004) Duodenal switch is a safe operation for patients who have failed other bariatric operations. Obes Surg, 14, 1187–1192.

Lee CM, Cirangle PT, Jossart GH; (2007), Vertical gastrectomy for morbid obesity in 216 patients report of two years. Surg Endosc, 21 ,1810-1816.

Marceau P, Hould FS, Simart S, et al.(1998). Biliopancreatic Diversion with Duodenal Switch, World J Surg, 22 , 947-954.

Marceau P, Biron S, Lebel S, et al. (2002). Does bone change after biliopancreatic diversion? J Gastrointest Surg, 6 , 690–698.

Marinari G, Adami GF, Camerini G, et al.(1997). The effect of biliopancreatic diversion on serum cholesterol. Obes Surg, 7 , 297.

Mason EE, Ito C: (1967) Gastric bypass in obesity. Surg Clin North Am, 47 ,1345–1352.

McAuley K, Mann J. (2006) Thematic Review Series: Patient Oriented Research. Nutritional Determinants of Insulin Resistance. J Lipid Res, 47, 1668–1676.

Montagna G, Gianetta E, Elicio N, et al.(1987).Plasma lipid and apoprotein pattern in patients with morbid obesity before and after biliopancreatic bypass. Atheroscl Cardiovasc Dis, 3 ,1069–1074.

Polyzogopoulou EV, Kalfarentzos F, Vagenakis AG, et al.(2003). Restoration of Euglycemia and Normal Acute Insulin Response to Glucose in Obese Subjects with Type 2 Diabetes Following Bariatric Surgery. Diabetes, 52 ,1098–1103.

Primavera A, Schenone A, Simonetti S, et al.: (1987) Neurological disorders following biliopancreatic diversion. In: Proceedings of the Third International Symposium on Obesity Surgery, Genoa, Italy, September, 1987.

Ren CJ, Patterson E, Gagner M. (2000) early results of biliopancreatic diversion with duodenal switch; a case series of 40 consecutive patients. Obes surg, 10 , 514-523.

SAGES Guidelines Committee. SAGES guideline for clinical application of laparoscopic bariatric surgery. Surg Endosc. 2008; 22:2281–300.

SarrMG. (2007) Reoperative bariatric surgery. Surg Endosc, 21 ,1909–1913.

Scopinaro N: (1974) Intervento in Tavola rotonda su: Trattamento medicochirurgico della obesit`a grave. Accad Med, 88–89 ,215–234.

Scopinaro N, Gianetta E, Civalleri D, et al. (1979) . Bilio-pancreatic by-pass for obesity, I: An experimental study in dogs. Br J Surg , 66 , 613–617.

Scopinaro N, Gianetta E, Civalleri D, et al. (1979) . Bilio-pancreatic by-pass for obesity, II: Initial experience in man. Br J Surg , 66 , 619–620.

Scopinaro N, Gianetta E, Civalleri D, et al. (1980) . Two years of clinical experience with biliopancreatic bypass for obesity. Amer J Clin Nutr, 33 , 506–514.

ScopinaroN, Gianetta E, FriedmanD, et al. (1992) . Biliopancreatic diversion for obesity. Probl Gen Surg , 9 , 362–379.

Scopinaro N, Gianetta E, Adami GF, et al. (1996) . Biliopancreatic diversion for obesity at eighteen years. Surgery, 119 , 261–268.

Scopinaro N, Adami GF, Marinari G, et al. (1997) . The effect of biliopancreatic diversion on glucose metabolism. Obes Surg, 7 , 296–297.

Scopinaro N, Marinari GM, Gianetta E, et al. (1997) . The respective importance of the alimentary limb (AL) and the common limb (CL) in protein absorption (PA) after BPD. Obes Surg, 7 , 108.

Scopinaro N, Adami GF, Marinari GM, et al. (1998) . Biliopancreatic diversion. World J Surg, 22 , 936–946.

Scopinaro N, Marinari GM, Adami GF, et al. (1999) . The influence of gastric volume on energy and protein absorption after BPD. Obes Surg , 2 , 125–126.

Scopinaro N, Marinari GM, Camerini G, et al. (2000) . Energy and nitrogen absorption after biliopancreatic diversion. Obes Surg , 10 , 436–441.

Scopinaro N, Marinari GM, Camerini G. (2002) . Laparoscopic standard biliopancreatic diversion: technique and preliminary results. Obes Surg , 12 , 241–244.

Scopinaro N, Marinari GM, Camerini GB, et al. (2005) . Specific Effects of Biliopancreatic Diversion on the Major Components of Metabolic Syndrome: A Long-term Follow-up Study. Diabetes Care , 28 , 2406–2411.

Scopinaro N, Papadia F, Camerini G, et al. (2008) . A Comparison of a Personal Series of Biliopancreatic Diversion and Literature Data on Gastric Bypass Help to Explain the Mechanisms of Resolution of Type 2 Diabetes by the Two Operations. Obes Surg ,18 ,1035–1038.

Scopinaro N, Adami GF, Papadia FS, et al. (2011) . Effects of biliopancreatic diversion on type 2 diabetes in patients with BMI 25–35. Ann Surg , 253 , 699–703.

Storer EH,Woodward ER, Dragstedt LR: (1950) . The effect of vagotomy and antrum resection on the Mann-Williamson ulcer. Surgery , 27 , 526– 530.

Thorell A, Nygren J, Ljungqvist O. (1999) Insulin Resistance: A Marker of Surgical Stress. Curr Opin Clin Nutr Metab Care , 2 , 69–78.

Topart P, Becouarn G, Ritz P. (2007) . Biliopancreatic diversion with duodenal switch or gastric bypass for failed gastric banding: retrospective study from two institutions with preliminary results. Surg Obes Relat Dis , 3 , 521–525.

Valverde I, Puente J,Martín-Duce A, et al. (2005) Changes in Glucagon-like Peptide-1 (GLP-1) Secretion after Biliopancreatic Diversion or Vertical Banded Gastroplasty in Obese Subjects. Obes Surg ,15 , 387–397.

Van Dessel E, Hubens G, Ruppert M, et al. (2008). Roux-en-Y gastric bypass as a re-do procedure for failed restricive gastric surgery. Surg Endosc , 22 , 1014–1018.

Weiner RA, Blanco-Engert R, Weiner S, et al. (2004) . Laparoscopic biliopancreatic diversion with duodenal switch: three different duodeno-ileal anastomotic techniques and initial experience. Obes Surg , 14 ,334–340.

Weller WE, Rosati C. (2008) . Comparing outcomes of laparoscopic versus open bariatric surgery. Ann Surg , 248 , 10–5.

Wickremesekera K, Miller G, Naotunne TD, et al. (2005) Loss of Insulin Resistance after
          Roux-en-Y Gastric Bypass Surgery: A Time Course Study. Obes Surg ,15 , 474–
          481.
Williams JA: (1964) Effects of upper gastro-intestinal surgery on blood formation and bone
          metabolism. Br J Surg , 51 , 125–134.
Xu G, Stoffers DA, Habener JF, et al. (1999) . Exendin-4 Stimulates Both Beta-cell Replication
          and Neogenesis, Resulting in Increased Beta-cell Mass. Diabetes ,48 , 2270–2276.
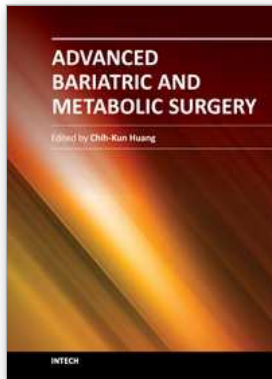
Advanced Bariatric and Metabolic Surgery

Edited by Dr. Chih-Kun Huang

Bariatric surgery has gained importance in the last 20 years because of the high prevalence of global obesity, and the vast understating of the physiological and pathological aspects of obesity and associated metabolic syndromes. This book has been written by a number of highly outstanding authors and pioneering bariatric surgeons from all over the world. The intended audience for this book includes all medical professionals involved in caring for bariatric patients. The chapters cover the choice of operation, preoperative preparation including psychological aspect, postoperative care and management of complication. It also extends to concept and result of metabolic surgery and scarless bariatric surgery.

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

INTECH
open science | open minds