BPD FROM BARIATRIC TO METABOLIC SURGERY

RETROSPECTIVE STUDY OF 443 MORBIDLY OBESE PATIENTS WITH >20 YEAR FOLLOW-UP

PILOT STUDY IN PATIENTS WITH SIMPLE OVERWEIGHT OR MILD OBESITY

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Churchill Livingstone
Evolution of Biliopancreatic Bypass

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ABSTRACT The original model of partial biliopancreatic bypass was safe but not effective enough to assure permanent reduction under 40% overweight for all patients. In three successive types the weight loss was increased by shortening the alimentary tract and by adding to malabsorption some restriction of food intake. This was effected by creation of a little stomach and a transient postcibal syndrome. Though long term results are unavailable, the combined use of the two latest modifications is likely to produce satisfactory results in all patients. The overall early complication rate (8%) did not increase in the latest type of biliopancreatic bypass. The incidence of stomal ulcer decreased to less than 1%. No additional specific late complications appeared, except for a 20% incidence of transient and easily manageable protein malnutrition.

INTRODUCTION

There is general agreement that obesity becomes a real disease with a significant increase of mortality when weight exceeds 40% above ideal [1]. The goal of obesity surgery, in terms of ‘recovery’ from morbid overweight, should be to reach and maintain a body weight under this risk limit.

also temporarily reduced by adding to malabsorption of the partial BPB the transitory postcibal syndrome generated by the creation of a little stomach.

DEVELOPMENT OF PARTIAL BILIOPANCREATIC BYPASS
### Table 3  Other beneficial effects of partial biliopancreatic bypass

<table>
<thead>
<tr>
<th>Condition</th>
<th>HH</th>
<th>SL</th>
<th>LS</th>
<th>VLS</th>
<th>Min follow-up</th>
<th>Disappeared</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pickwickian syndrome*</td>
<td>0</td>
<td>0</td>
<td>4%</td>
<td>9%</td>
<td>1 month</td>
<td>100%</td>
</tr>
<tr>
<td>Somnolence**</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7%</td>
<td>1 month</td>
<td>100%</td>
</tr>
<tr>
<td>Hypertension</td>
<td>10%</td>
<td>28%</td>
<td>25%</td>
<td>49%</td>
<td>12 months</td>
<td>90%</td>
</tr>
<tr>
<td>Liver abnormalities***</td>
<td>45%</td>
<td>37%</td>
<td>53%</td>
<td>56%</td>
<td>24 months</td>
<td>83%</td>
</tr>
<tr>
<td>Leg atasis</td>
<td>22%</td>
<td>28%</td>
<td>45%</td>
<td>38%</td>
<td>12 months</td>
<td>41%</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>18%</td>
<td>27%</td>
<td>18%</td>
<td>24%</td>
<td>1 month</td>
<td>100%</td>
</tr>
<tr>
<td>Hypertriglyceridemia</td>
<td>19%</td>
<td>27%</td>
<td>35%</td>
<td>39%</td>
<td>12 months</td>
<td>94%</td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>10%</td>
<td>7%</td>
<td>13%</td>
<td>14%</td>
<td>1 month</td>
<td>100%</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>2%</td>
<td>2%</td>
<td>4%</td>
<td>2%</td>
<td>4 months</td>
<td>100%</td>
</tr>
<tr>
<td>Diabetes mellitus, insulin</td>
<td>0</td>
<td>5%</td>
<td>2%</td>
<td>2%</td>
<td>12 months</td>
<td>50%</td>
</tr>
<tr>
<td>Hyperuricemia</td>
<td>8%</td>
<td>2%</td>
<td>19%</td>
<td>14%</td>
<td>4 months</td>
<td>81%</td>
</tr>
<tr>
<td>Gout</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3%</td>
<td>4 months</td>
<td>100%</td>
</tr>
</tbody>
</table>

* Somnolence with cyanosis, polycythemia and hypercapnia.
** In absence of one or more of the above.
*** More than 10% fatty metamorphosis.
**** Insulin no longer required.
***** Serum uric acid normalised, no more clinical symptoms.
genetically jeopardized pancreas

insulin resistance

TYPE 2 DIABETES
all type 2 diabetic patients have a genetically compromised endocrine pancreas

the endocrine pancreas weakness consists of impaired beta cell function and increased beta cell apoptosis

as beta cells in type 2 diabetes must counteract the insulin resistance, it can be assumed that the smaller the amount of extra body fat, and thus insulin resistance, the more severe genetic damage of the pancreas
ENVIROMENT

excessive fat availability

fat penetration into the muscular cell

the muscular cell uses fat instead of glucose as the energy source

insulin resistance

hyperinsulinemia
all bariatric operations have a beneficial effect on diabetes which is strictly depending on weight loss.

Biliopancreatic diversion (BPD) has specific actions on diabetes which are independent of weight changes.
BILIOPANCREATIC DIVERSION
permanent limitation of digestion

permanent limitation of intestinal absorption of fat and starch

WEIGHT LOSS AND MAINTENANCE
duodenal exclusion from the alimentary stream and food stimulation of distal small bowel cause gut hormonal changes which, together with the reduced or annulled beta cell fat and glucose toxicity, result in improvement of beta cell function and progressive trophic action on the beta cell organ.
the other very important BPD specific action consists of the extremely reduced fat absorption to only about 40 g/day, not chronically obtainable by any other means, causing fat depletion of the muscular cell, which is obliged to return to glucose utilization as energy source, with the consequent restoration of normal insulin sensitivity.
SPECIFIC ACTIONS OF BPD ON CHOLESTEROL METABOLISM

• calibrated interruption of the entero-hepatic bile salt circulation with the consequent increase of bile acid synthesis at the expenses of the cholesterol pool

• strongly reduced absorption of endogenous cholesterol
thanks to these specific actions BPD has proven able to fully resolve the metabolic syndrome in all the 443 morbidly obese type 2 diabetic patients operated on during the last over 30 years, and to maintain this normalization for more than 20 years.
237/237 type 2 diabetes patients with BMI > 35 kg/m² 10 years after AHS BPD

1. normal serum glucose 
2. normal serum cholesterol 
3. normal serum triglycerides 
4. normal arterial pressure in 80% of cases
237 type 2 diabetes patients with BMI > 35 kg/m² 10 years after BPD
237 type 2 diabetes patients with BMI > 35 kg/m² 10 years after BPD
237 type 2 diabetes patients with BMI > 35 kg/m² 10 years after BPD

BARIATRIC SURGERY AND TYPE 2 DIABETES IN PATIENTS WITH BMI >35
EFFECT OF BPD

Nicola Scopinaro, MD, FACS(Hon)

Diabetes Surgery Summit, Rome, March 2007
237 type 2 diabetes patients with BMI > 35 kg/m² 10 years after BPD

serum triglycerides (mg/dl)

prior to 1 yr 2 yr 3 yr 5 yr 10 yr

Nicola Scopinaro, MD, FACS(Hon)

Diabetes Surgery Summit, Rome, March 2007
### morbidly obese patients with full diabetes resolution after BPD

<table>
<thead>
<tr>
<th></th>
<th>N.o</th>
<th>%</th>
<th>follow-up rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>preoperatively</td>
<td>443/443</td>
<td></td>
<td>443/443 (100 %)</td>
</tr>
<tr>
<td>at 1-2 months</td>
<td>320/425</td>
<td>75.9</td>
<td>425/443 (96 %)</td>
</tr>
<tr>
<td>at 1 year</td>
<td>381/383</td>
<td>99.5</td>
<td>383/443 (82 %)</td>
</tr>
<tr>
<td>at 10 years</td>
<td>292/293</td>
<td>99.7</td>
<td>293/407 (72 %)</td>
</tr>
<tr>
<td>at 20 or more years</td>
<td>32/34</td>
<td>94.1</td>
<td>34/56 (64 %)</td>
</tr>
</tbody>
</table>
morbidly obese patients with type 2 diabetes
serum cholesterol >200 mg/dl after BPD

<table>
<thead>
<tr>
<th>Time</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preoperatively</td>
<td>280/443</td>
<td>63</td>
</tr>
<tr>
<td>At 1 year</td>
<td>1/363</td>
<td>0.3</td>
</tr>
<tr>
<td>At 10 years</td>
<td>0/293</td>
<td>0</td>
</tr>
<tr>
<td>At 20 years</td>
<td>0/36</td>
<td>0</td>
</tr>
<tr>
<td>Time</td>
<td>N.o</td>
<td>%</td>
</tr>
<tr>
<td>-----------------</td>
<td>----------</td>
<td>----</td>
</tr>
<tr>
<td>preoperatively</td>
<td>181/443</td>
<td>41</td>
</tr>
<tr>
<td>at 1 year</td>
<td>36/363</td>
<td>10</td>
</tr>
<tr>
<td>at 10 years</td>
<td>6/293</td>
<td>2</td>
</tr>
<tr>
<td>at 20 years</td>
<td>1/36</td>
<td>3</td>
</tr>
</tbody>
</table>
293 patients with type 2 diabetes and BMI > 35 kg/m² 10 years after BPD
CONCLUSION

BPD causes full resolution of type 2 diabetes and metabolic syndrome at short, long, and very long term, independently of the body weight changes, on free diet and with no medication.
if it works in the morbidly obeses, why not to try in the lower ranges of BMI, which include more than 90 per cent of type 2 diabetic patients?
GI SURGERY AND TYPE 2 DIABETES IN PATIENTS WITH BMI <35 kg/m²

EFFECT OF BPD

seven type 2 diabetes patients with BMI < 35 kg/m² (mean: 33.4)

fasting serum glucose (mg/dl)

Nicola Scopinaro, MD, FACS(Hon)

Diabetes Surgery Summit, Rome, March 2007
GI SURGERY AND TYPE 2 DIABETES IN PATIENTS WITH BMI < 35 kg/m²

EFFECT OF BPD

seven type 2 diabetes patients with BMI < 35 kg/m²

BMI (kg/m²)

Nicola Scopinaro, MD, FACS(Hon)

Diabetes Surgery Summit, Rome, March 2007
no patient at any postoperative time had excessive weight loss, the minimum observed BMI having been 22
A PILOT STUDY ON 27 TYPE 2 DIABETIC PATIENTS WITH BMI BETWEEN 25 AND 35 KG/M2 SUBMITTED TO BILIOPANCREATIC DIVERSION

started on May, 2007
120 kg corresponding to an intake of ~2300 Cal/day – expected loss: ~ 35 kg

95 kg corresponding to an intake of ~ 1950 Cal/day – expected loss: ~ 10 kg

Maximum energy absorption of ~ 1700 Cal/day corresponding to a body weight of ~ 85 kg

70 kg corresponding to an intake of ~ 1450 Cal/day – expected loss: none
as the BPD actions on T2DM are specific, and thus independent of weight changes, they should work also in the diabetic patients who, being close to the stabilization weight, or even having a weight lower than that, will lose little or no weight after the operation.
PILOT STUDY

effects of BPD on 27 patients

type 2 diabetes lasting at least 3 yrs
BMI between 25 and 34.9
medical therapy
HbA1c minimum 8.0%
PILOT STUDY

preoperatively and 1, 4, 8, and 12 months after BPD determination of

fasting serum glucose level

glycosylated hemoglobin (HbA1c)

acute insulin response to intravenous glucose load

HOMA

body weight
27 patients (9 F) operated on

mean age: 57 yrs (44-69)
mean BMI: 30.9 kg/m² (25.3-34.9)
mean diabetes duration: 13 yrs: (3-28)
insulin therapy: 9 c.

12 patients (2 F) with BMI <30 (mean: 27.9)
PILOT STUDY

ACCESS: 16 laparotomic, 11 laparoscopic

EARLY COMPLICATIONS
1 intraperitoneal bleeding (relaparoscopy)
4 gastroplegia (all resolved within 2 weeks)

LATE COMPLICATION
none
PILOT STUDY

all patients were discharged with no specific therapy

27 cases reached the 1° month control
21 cases reached the 4° month control
10 cases reached the 8° month control
FASTING SERUM GLUCOSE (mg/dl)

preop (27 c.): 222 (129-361)
1-2 months (27 c.): 181 (85-367)
4 months (21c.): 153 (89-312)
8 months (10 c.): 117 (80-166)
HbA1c
(mean glycemic control during the last 2-3 months in T2DM patients undergoing BPD)

preop (27 c.): 9.7 (8.0-13.5)
1-2 months (27 c.): 7.7 (5.8-10.4)
4 months (21 c.): 6.9 (5.7-10.4)
8 months (10 c.): 5.3 (4.3-6.2)
serum Hb1Ac concentration in T2DM patients with preoperative BMI < 35 kg/m² undergoing BPD
HbA1c level prior to and after BPD

- Preop
- 1 month
- 4 months
- 8 months

- HbA1c > 7%
- HbA1c > 6.1 - 7%
- HbA1c ≤ 6%
<table>
<thead>
<tr>
<th>Time After BPD</th>
<th>Mean (mg/dl)</th>
<th>Abnormal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prior to BPD</td>
<td>185</td>
<td>8/27 (31%)</td>
</tr>
<tr>
<td>1 month after BPD</td>
<td>125</td>
<td>0/27 (0%)</td>
</tr>
<tr>
<td>4 months after BPD</td>
<td>138</td>
<td>0/21 (0%)</td>
</tr>
<tr>
<td>8 months after BPD</td>
<td>147</td>
<td>0/10 (0%)</td>
</tr>
</tbody>
</table>

Type 2 diabetes patients with BMI <35 serum cholesterol values after BPD
<table>
<thead>
<tr>
<th></th>
<th>mean (mg/dl)</th>
<th>abnormal values</th>
</tr>
</thead>
<tbody>
<tr>
<td>prior to BPD</td>
<td>201</td>
<td>9/27 (33 %)</td>
</tr>
<tr>
<td>at 1 month after BPD</td>
<td>143</td>
<td>8/27 (30%)</td>
</tr>
<tr>
<td>at 4 months after BPD</td>
<td>177</td>
<td>10/21 (48%)</td>
</tr>
<tr>
<td>at 8 months after BPD</td>
<td>177</td>
<td>6/10 (60%)</td>
</tr>
</tbody>
</table>

type 2 diabetes patients with BMI <35
serum triglycerides values after BPD
V.R. (preop. BMI: 28.9) – Acute insulin response (mcU/ml) to intravenous glucose load (35 g in 2 min) preoperatively and 1 month after BPD
P.D. (preop. BMI: 33.9) – Acute insulin response (mcU/ml) to intravenous glucose load (35 g in 2 min) preoperatorively and 1 month after BPD.
acute insulin response (AIR) in morbidly obese patients with T2DM preoperatively and one months after BPD
acute insulin response (AIR) type 2 diabetic subjects preoperatively and one month after BPD
acute insulin response (AIR) in type 2 diabetic subjects preoperatively and four months after BPD
HOMA
(index of insulin-resistance)

preop (27 c.): 8.1 (0.6-20.4)
1-2 months (27 c.): 3.3 (1.2-12.9)
4 months (21 c.): 3.2 (0.52-10.8)
8 months (10 c.): 1.9 (0.3-3.2)
T2DM patients with BMI between 25 and 34.9 kg/m$^2$ after BPD
individual BMI changes

kg/m$^2$

prior to at 1-2 months at 4 months at 8 months
CONCLUSION

BPD seems able to ensure total remission of T2DM also in the range of BMI between 25 and 35 kg/m², without causing any excessive or undue weight loss.
THANK YOU FOR YOUR ATTENTION