Egyptian Journal of Surgery Vol. (19), No. (3), July, 2000

PYLORIC FUNCTION AFTER PYLORUS-PRESERVING PANCREATO-DUODENECTOMY

By
Ayman Salah, Mohamed El-Sherif El-Sarky, Alaa A. Maher, Ashraf Salah, Ahmed Fahmy Omar and Adel M Taher
Department of Surgery, Faculty of Medicine, Cairo University

This work compares the outcome of pylorus preserving pancreatoduodenectomy (PPPD) for periampullary carcinoma after 2 reconstructive methods, Billroth-I (B-I) and Billroth-II (B-II). A special consideration was given to pyloric function after PPPD (gastric emptying and pyloric sphincter competence) as it has been frequently reported to be disturbed after the procedure. Of the studied 26 patients, 15 patients had the classical B-II reconstruction while 11 patients had B-I reconstruction. Patients were comparable for age and sex in both groups. Mortality was 11.5% and complications occurred in 38.5% of cases. Delayed gastric emptying (DGE) occurred in 69.9% of cases in the immediate postoperative phase. At follow-up, 69.5% of patients had GI symptoms and 30.4% of patients had DGE. Bile stasis was observed in 4 patients (17.4%) and biliogastric reflux in 3 of them (13%). Antral gastritis occurred in 3 patients (13%) and reflux esophagitis in one patient (4.3%). Mortality and morbidity showed no significant difference between the 2 groups. In the early phase, the incidence of DGE was comparable in both groups but delayed food tolerance was more observed in group B-II. In the late phase, the delay in gastric emptying in B-II patients involved the actual emptying phase rather than the lag period, suggesting dysfunction of proximal jejunal loop as an explanation for the delay. Biliogastric reflux, antral gastritis and reflux esophagitis were all limited to group B-II patients. In conclusion, both methods of reconstruction are comparable as regards general outcome but B-I reconstruction is probably superior functionally.

Key words: Pylorus preserving pancreatoduodenectomy – Billroth-I and Billroth-II reconstruction – pyloric function.

INTRODUCTION

The standard procedure for periampullary carcinoma is the classical pancreato-duodenectomy (CPD). This operation was first performed successfully by Codivilla in 1898[1]. In 1935, Whipple et al described a two-stage pancreaticoduodenectomy[2] where they preserved the distal stomach, pylorus and proximal duodenum and in 1941, Whipple reported the first successful one-stage pancreaticoduodenectomy in which the distal stomach, pylorus and duodenum were removed[3]. Since then, a number of technical modifications have been reported for the treatment of periampullary malignant diseases.

Pylorus-preserving pancreaticoduodenectomy (PPPD) was first described by Watson in 1944 but it did not gain popularity[4]. In 1978, Traverso and Longmire re-introduced the procedure in order to reduce dumping, postgastrectomy syndromes and marginal ulcers[5]. These complications were frequently observed in association with the classical pancreatoduodenectomy (CPD).

Several technical and functional advantages were described for PPPD over CPD. This encouraged the spread of the former in the last years[6,7,8,9].

On the other hand, PPPD still has its problems. Beside many of those known for CPD, PPPD is associated with disturbed function of the preserved pylorus. This is reflected by 2 disorders: 1) Delayed gastric emptying (DGE), and 2) Pyloric incompetence which results in biliogastric reflux (BGR).

Several modifications have been suggested to further

improve the functional and nutritional outcome of PPPD and reduce its complications. Many of these involved the method of reconstruction after resection. One modification is a Billroth-I (B-I) type of reconstruction that was first described by Imanaga in 1960 and has been spreading since then[10]. In this method, the duodenum, the pancreas and the bile duct are anastomosed to the proximal jejunal loop in this order, i.e. in their physiological order. This method was reported to have several advantages over the original Billroth-II (B-II) type of PPPD.

Until recently, very few attempts were made to compare the results of B-I and B-II methods of reconstruction in PPPD, particularly as regards subsequent pyloric function. This work compares the outcome of these 2 methods concerning early (immediate postoperative) and late pyloric function.

PATIENTS AND METHODS

This study included 26 patients who had operable periampullary carcinoma. Diagnosis was based on clinical findings, ERCP, abdominal CT scan among other investigations. Preoperative pathological diagnosis was available in 7 patients by CT-guided biopsy.

The patients were 14 males and 12 females and ranged in age from 34 to 68 years (average 53.4 ±7.5). In addition, 8 volunteers shared in the study to provide control data for gastric radionuclide scan (GRS). They were 5 males and 3 females with an average age of 45 years (± 10.36). These volunteers had no abdominal complaints and were mainly workers and patients waiting for ambulatory minor procedures.

All patients were treated by pylorus-preserving pancreatoduodenectomy (PPPD). Reconstruction after resection was achieved by either: B-II reconstruction (group B-II, 15 patients) or B-I reconstruction (group B-I, 11 patients), (Fig. 1). The choice of the method was based on the preference of the surgeon, i.e. no randomization.

B-II B-I

Fig. (1): A diagram which compares the two methods of reconstruction after PPPD: B-II and B-I

In B-I reconstruction, the proximal jejunal loop (next to resection end) was passed through the transverse mesocolon to be anastomosed to the duodenal end (end-to-end) then 3-4 cm distally to the pancreatic end (end-to-side) and finally to bile duct (end-to-side). In 5 patients of B-II group and 4 patients of B-I group, the pancreatic duct was ligated.

Two pyloric functions were assessed: emptying and competence. They were studied by detecting: A) Two primary disorders: DGE and BGR, and B) Two secondary disorders: antral gastritis and alkaline reflux esophagitis.

Assessment of pyloric function was done in the early phase (postoperative period) and late phase (follow-up, at least 2 months after the procedure).

* ***Assessment in the early phase***

In the early phase, diagnosis of delayed gastric emptying was based on nasogastric output monitoring and patients' ability to tolerate oral intake. Nasogastric output was observed postoperatively for the daily amount. The postoperative day when the patient started to tolerate oral feeding was also marked. Delayed gastric emptying was defined as persistent nasogastric output of 1000 ml or more for 10 or more days, or inability to tolerate oral feeding for 14 or more days, or both[11].

* ***Assessment in the late phase***

In the late phase, diagnosis of pyloric dysfunction was based on the following:

1. Suggestive persistent symptomatology (nausea, vomiting, dyspepsia, fullness, abdominal pain, food intolerance, heartburn or dysphagia).

2. Confirmatory tests:
   - Gastric radionuclide scan for delayed gastric emptying (DGE).
   - HIDA scintigraphy for BGR.
   - Upper GI-endoscopy for: antral gastritis and reflux esophagitis.

- The severity of patients' symptoms, which suggested disturbed pyloric function, could be simply classified according to patients' own perception into 3 grades. These were: 1) mild (discomfort, not interfering with daily activity or producing continuous distress), 2) moderate (tolerable, slightly interfering with activities which are however maintained, and producing frequent but not continuous distress) and 3) severe (intolerable, incapacitating, blocking activities and causing continuous distress).

- Gastric radionuclide scan the patient was instructed to fast for 12 hours (overnight). The test diet consisted of corn flakes and skimmed milk. The meal was mixed with ^{99m}Tc labeled sulfur colloid (a dose of 250 MicroCi). Images were taken by gamma camera every 5 minutes for 20 minutes then every 10 minutes for 2-3 hours with the patient standing.

Two parameters were calculated: lag time (LT: the time interval from full administration of the meal to the beginning of gastric emptying) and half emptying time (T1/2: the time required by the stomach to empty half of its contents). Calculation of these parameters followed the mathematical method (i.e. based on equations). Results were compared with those of volunteers.

- Detection of BGR was based on HIDA-scan after i.v. administration of a bolus of 80MBq of ^{99m}Tc Trimetil, 3-Br iminodiacetic acid. The diagnosis of bile reflux was based on visualization of the stomach in the scan.

- Upper GI-endoscopy was done under general sedation and focused on signs of alkaline reflux esophagitis, antral gastritis, pyloric ring patency and any marginal pathology at the duodeno-jejunal junction.

RESULTS

**** Perioperative data***

A comparison between patients of both groups is presented in (Table 1). Complications occurred in 10 patients (38.5%), including the three patients who died. Five patients required reoperation including 2 of those who did not survive, (Table 2). Three patients did not survive (11.5%). Operative mortality was defined as death within one month of the operation[12]. One patient was of group B-I and died of intraabdominal bleeding (reoperated) while the other two were of group B-II and died of cardiac complications and anastomotic leak (reoperated), (Table 2).

Table (1)*: General comparison between the two groups of patients*

	B-II	*B-I*
Number	15	11
Age (years)	52.7 ± 7.9	54.3 ± 7.3
Male-to-female ratio	(8:7) 1.1	(6:5) 1.2
Mortality	2/15 (13.3%)	1/11 (9.1%)
Complications	7/15 (46.7%)	3/11 (27.3%)
Stay in hospital (days)	28.0 ± 10.0	26.5 ± 7.9

Table (2)*: complications, mortality and causes of death (Reop.: reoperation, C: complications, M: mortality and DOS: duration of stay in hospital in days).*

No	*Sex*	*Age*	*Procedure*	*Outcome*	*Reop.*	*DOS*	*Details*
3	F	62	B-I	C	+	44	Abdominal collection, no leak
5	F	43	B-II	C		33	Pancreatitis
8	M	53	B-II	C	+	48	Wound dehiscence
9	M	47	B-I	C		29	Upper GI bleeding
10	M	58	B-II	M		6	Cardiac complications
14	M	53	B-II	C		42	Cholangitis (?)
15	F	57	B-II	M	+	17	Anastomotic leak
18	M	68	B-I	M	+	14	Extensive intra-abdominal bleeding
19	M	59	B-II	C		29	Wound infection
24	F	57	B-II	C	+	37	Abdominal collection, no leak

**** Pyloric function in the early phase***

According to the predetermined definition, DGE was experienced in 16 patients in general (69.9%). (Table 3) presents a list of all patients and basis for diagnosis of early DGE.

Table (3): ***Full data about gastric delay in the early phase and duration of hospital stay in all patients.***
N/G: days of nasogastric suction. Feeding: day mark of tolerating oral feeding. DOS: duration of stay in hospital. X: mortality.

No	*sex*	*age*	*procedure*	*N/G*	*Feeding*	*DOS*
1	M	34	B-II	5	11	19
2	F	54	B-I	6	9	18
3	F	62	B-I	16	23	44
4	M	57	B-I	9	12	22
5	F	43	B-II	9	17	33
6	F	61	B-I	12	17	28
7	F	56	B-II	11	14	22
8	M	53	B-II	6	16	48
9	M	47	B-I	13	19	29
10	M	58	B-II	X	X	6
11	F	49	B-II	8	16	27
12	M	44	B-I	11	15	28
13	M	56	B-II	7	17	32
14	M	53	B-II	9	18	42
15	F	57	B-II	X	X	17
16	M	49	B-I	13	18	31
17	M	55	B-I	5	8	18
18	M	68	B-I	X	X	14
19	M	59	B-II	6	15	29
20	F	48	B-I	6	16	19
21	F	52	B-I	11	17	28
22	M	43	B-II	4	8	17
23	F	49	B-II	6	9	19
24	F	57	B-II	8	15	37
25	M	61	B-II	7	14	23
26	F	63	B-II	6	10	16

The distribution of patients with early DGE in both groups is presented in (Table 4), which also shows the basis for diagnosis of early DGE in each patient. All survivors who had postoperative complications (7 patients) had DGE (100%). The incidence of DGE in the others was 56.3% (9/16 patients).

Table(4)*: Early DGE, incidence and the deciding parameter in each group.*

Group	*Incidence*	*Deciding parameter*		
		N/G delay	Feeding delay	Both
B-II	9/13 (69.2)	0	8 (89%)	1
B-I	7/10 (70.00)	0	1 (14%)	6

**** Pyloric function in the late phase***

- The follow-up period ranged between 3 and 11 months. The average interval to follow-up was 6.1 (±2.9) and 5.3 (±2.3) months in B-II and B-I groups in order (no significant difference). Upon presentation for follow-up, the patient was asked about GI symptoms and simple symptom score was done. Sixteen patients (69.5%) had GI symptoms in variable degrees on basis of the simple score adopted in this work, (Table 5).

Table (5)*: Symptomatology at the time of presentation for follow-up*

Group	Patients with symptoms			
	Total	Grade-I (mild)	Grade-II (moderate)	Grade-III (severe)
B-II	9/13 (69.2%)	1	4	4
B-I	7/10 (70%)	1	4	2

In normal volunteers, GRS control values were: 41.25 (±7.89) minutes for average LT and 92.75 (±13.44) minutes for average T1/2. The diagnosis of DGE (Fig.2) was considered possible when the values of either or both of these parameters (LT and T1/2) were above +1 standard deviation of the volunteers' values and certain when they were above +2.

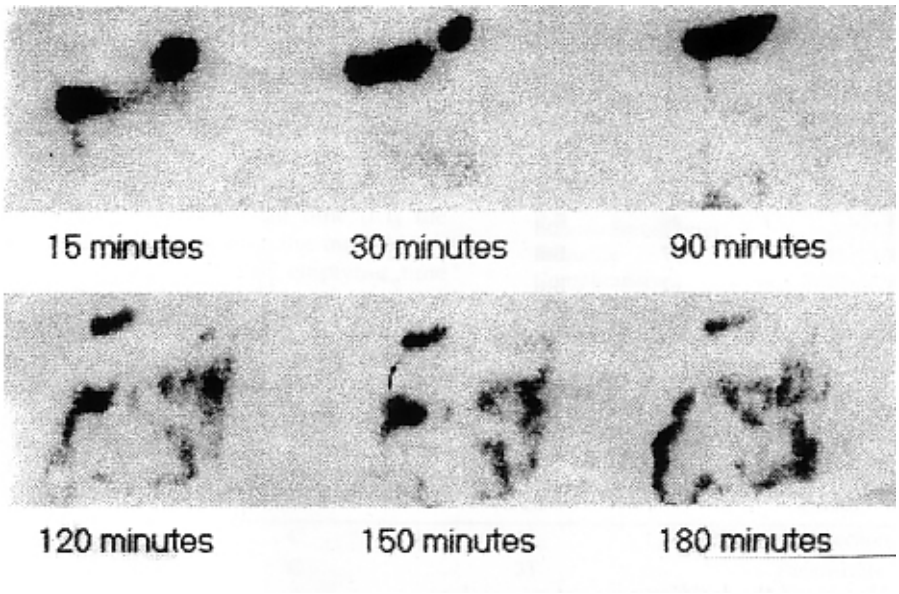

Fig.(2): Gastric radionuclide scan of a patient who has DGE

Severity of delay was assessed according to the degree of shift from the normal range. This was assessed in units of standard deviation from the mean. On these bases, 7 Patients (30.4%) had late DGE, (Table 6). Of these, 4 patients were in group B-II and 3 patients in group B-I.

Table (6): ***Parameters of GRS***

No	*OP*	*TL*	*TLS*	*T1/2*	*T1/2S*	*Diagnosis*
2	I	43.0	0.2	101.0	0.6	NN
3	I	39.0	-0.3	88.0	-0.4	NN
4	I	31.0	-1.3	72.0	-1.5	NN
6	I	45.0	0.5	90.0	-0.2	NN
9	I	51.0	1.2	97.0	0.3	DN
12	I	59.0	2.2	114.0	1.6	DD
16	I	43.0	0.2	90.0	-0.2	NN
17	I	61.0	2.5	97.0	0.3	DN
20	I	44.0	0.3	88.0	-0.4	NN
21	I	36.0	-0.7	76.0	-1.2	NN
1	II	47.0	0.7	103.0	0.8	NN
5	II	55.0	1.7	124.0	2.3	DD
7	II	48.0	0.9	98.0	0.4	NN
8	II	39.0	-0.3	96.0	0.2	NN
11	II	49.0	1.0	105.0	0.9	NN
13	II	44.0	0.3	99.0	0.5	NN
14	II	46.0	0.6	98.0	0.4	NN
19	II	58.0	2.1	138.0	3.4	DD
22	II	43.0	0.2	111.0	1.4	ND
23	II	42.0	0.1	102.0	0.7	NN
24	II	33.0	-1.0	98.0	0.4	NN
25	II	47.0	0.7	118.0	1.9	ND
26	II	48.0	0.9	104.0	0.8	NN

TLS: time lag shift from the median normal value in units of standard deviation.
T1/2S: T1/2 shift from the median normal value in units of standard deviation.
N: within normal and D: delayed.

When each of the two defining parameters for DGE was considered separately, it was observed that DGE in group B-II patients was mainly due to a delay in T1/2 (present in all 4 cases) rather than TL (occurred in 2 patients). The reverse was observed in group B-I. Moreover, as a group, the B-II patients had a significant delay in T1/2 compared with B-I patients. This was evidenced by observing the average SD shifts (+1.08 vs -0.11) and average T1/2 (107.2±12.5 vs 91.3±12.0 minutes) and a higher incidence of delay (4/13 patients or 31% vs 1/10 patients or 10%). This was not observed for TL, (Table 7).

Table (7): *The extent of DGE in both procedures*

	Group	*B-II*		*B-I*	
		TL	T1/2	TL	T1/2
Number of patients	Normal	11 (85%)	9 (69%)	7 (70%)	9 (90%)
	Abnormal	2 (15%)	4 (31%)	3 (30%)	1 (10%)
	-1SD	1 (8%)	2 (15%)	1 (10%)	1 (10%)
	-2SD	1 (8%)	1 (8%)	2 (20%)	----
	-3SD	----	1 (8%)	----	----
Overall	Average	46.1±6.4	107.2±12.5	45.2±9.5	91.3±12.0

- HIDA scan (Fig. 3) demonstrated bile stasis in 4 patients (one in group B-I and 3 in group B-II), which was associated with minimal BGR in patients of group B-II only.

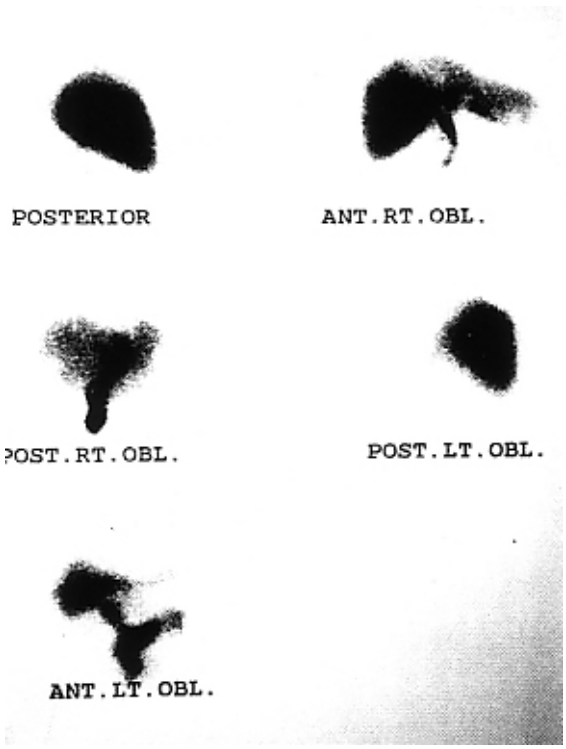

Fig. (3): HIDA scan of a patient showing bile stasis but no reflux

- Endoscopy revealed antral gastritis in 3 patients (all of group B-II) and reflux esophagitis in 1 patient (in group B-II). No other lesions (e.g. ulcers) were observed. The pyloric ring proved to be patent in all patients even those ho had marked delayed emptying. Detected abnormalities were compared with symptomatology in each patient, (Table 8).

Table (8)*: Correlating symptoms and test results in all patients.*

No	*Operation*	*Symptom score*	*Endoscopy*		*HIDA scan*		*DGE*	
			Antral gastritis	*Reflux esophagitis*	*Stasis*	*BGR*	*LT*	*T1/2*
2	B-I							
3	B-I	+++						
4	B-I	++						
6	B-I	+						
9	B-I	+++			+		+	
12	B-I	++					++	+
16	B-I	++						
17	B-I						++	
20	B-I							
21	B-I	++						
1	B-II							
5	B-II	++		+			+	++
7	B-II	+++			+	+		
8	B-II	+++	+		+	+		
11	B-II	++						
13	B-II							
14	B-II	+++			+	+		
19	B-II	++	+				++	+++
22	B-II							+
23	B-II	+						
24	B-II	+++	+					
25	B-II	++						+
26	B-II							

DISCUSSION

Since 1978, when Traverso and Longmire reintroduced PPPD, many advantages of this procedure over CPD have been reported. These advantages are both technical and functional.

Technically, the procedure was frequently reported to be easier in performance[13] and to take shorter operating time [14,15].

Functional sequels of PPPD were reported to be superior to CPD. Gastric function is maintained by sparing the antrum with normal secretion of gastric gastrin and normal gastric digestion[16]. This results in a better weight gain after PPPD than CPD, a finding which has been consistently reported by many authors[17,18,19,20]. Gastrointestinal symptoms after PPPD are also milder in comparison with CPD[20,21].

However, 2 factors in PPPD raised concerns: its radicality in malignancy and the observed disturbed function of the preserved pylorus.

Concerns about radicality of PPPD in comparison with CPD were based on the theoretical possibility of leaving tumor tissue at the pancreato-duodenal boundary in the surgeon's attempt to save the first duodenal part. These concerns were recently relieved by 2 findings. First, the most critical boundary in resection (as revealed by histological examination) was reported to be the pancreato-retroperitoneal one where most of positive margins were observed in CPD and PPPD[22,23,24]. Second, the long-term survival rates in CPD and PPPD were reported to be comparable[22,25] or even better in PPPD[24].

The second problem with PPPD remains mostly unsolved. Despite the fact that PPPD is physiologically appealing, the basic two functions of the preserved

pylorus, emptying and competence, proved to be disturbed after PPPD as evidenced by DGE and BGR, in order.

This work was planned to address 2 groups of issues: comparing the outcome in 2 reconstructive methods after PPPD (B-I and B-II), and focusing on pyloric function after these 2 methods.

Our patient groups were comparable for age and sex. Surgically, both procedures resulted in comparable morbidity and mortality. The incidence of complications in the studied patients is high (38.5%). Close figures are observed in recent reports, e.g. 54%[14], 44%[26] and 35-40%[27].

The mortality rate (11.5%) is high compared with other reports, e.g. 2.4%[12], 1%[26] and 9%[28], The majority of published results on PPPD come from specialized centers with cumulative experience. This is one of the factors which reduced mortality markedly over the last years.

Early DGE is a frequent complication of PPPD. The incidence of this disorder was reported by several investigators to be quite higher in PPPD than in CPD[29] but others reported the incidence to be comparable after both procedures[26].

The incidence of early DGE in our patients was 70%. This incidence is higher than the figures reported by other investigators which ranged from 6 to 50% of cases[6,26,28,29,30,31,32,33,34]. The use of prokinetic agents in our patients was restricted to avoid multiplicity of intervening factors. The incidence of DGE in patients who had postoperative complications was significantly higher than others (100% and 56.3%). This is in agreement with previous reports[26].

Assessing gastric emptying in the early phase is generally approached by observing nasogastric output and marking the date when the patient tolerates oral meals for the first time[26]. In this work, each of the two parameters was further analyzed separately and correlated to the performed procedure. The higher incidence of food intolerance as the sole defining parameter for DGE in B-II patients (89%) compared with B-I patients (14%) may be contributed to by factors other than the actual delay in gastric emptying, e.g. BGR, transient gastritis or others.

The incidence of GI symptoms in our patients (69.5%) was higher than that reported by others, e.g. 20% by Lupo et al[31]. One possible explanation is that we considered all patients' symptoms even the mild discomfort. Our suggested simple score encouraged this attitude rather than considering symptoms of certain gravity only. This score helped as well in sorting the patients and predicting underlying disorders. All the patients who had BGR, 71% of those with late DGE and 67% of those who had antral gastritis had symptoms of grade-II or III.

Most investigators approve that early DGE, which follows PPPD, gradually improves with time[6,16,35,36,37]. The period required to recover from DGE was estimated by Kobayashi et al[30] to be 2-5 weeks for liquids and 3 months for solids. On the other hand, gastric emptying was reported to remain delayed after a period extending between 2 months and 5 years after PPPD[38].

In this study, we employed gastric radionuclide scan to study gastric emptying. This method was previously utilized by the majority of investigators[39,40]. Still others used other methods as barium meal[16] or radio-opaque markers[41].

We preferred to have control values for gastric emptying by studying volunteers.

Despite accuracy of the GRS in estimating gastric emptying yet wide variations in the standard values of its parameters (LT and T1/2) were consistently observed. The reasons are mainly variations in the nature of the meal[42], food structure, technique, equipment and methods of interpretation[40,43].

The three standard methods for interpreting GRS are: 1) visual, by observing time when activity first appeared in the duodenum), 2) curve-based, by resorting to time-activity curves to determine time of 2% decrease from peak stomach activity, and 3) mathematical, employing standard equations[39,40,42,43]. The problem of interpreting GRS results can be solved, at least in one study, by calibrating results using normal controls for the same methodology. Accordingly, we adopted this policy in this study.

The exact mechanism for the pathogenesis of disturbed pyloric function after PPPD is not understood. Theories included: torsion or angulation of the reconstructed alimentary tract[44], tachygastria which is observed in electrogastrography[45], deficient motilin after duodenectomy[46], damage to the duodenal pacemaker[47], duodenal ischemia[6] or disturbed function of the first jejunal loop[48].

In the attempt to further assess the severity of DGE and its possible mechanism we suggested 2 new interpretative approaches: 1) Studying each of the GRS parameters separately, and 2) Assessing DGE severity on basis of the degree of shift from the mean in units of standard deviation. According to this methodology, a specific delay in T1/2 but not TL was observed in group B-II patients compared with those of group B-I. In other words, DGE in B-II patients was more likely to involve the

plateau phase of gastric emptying rather than the initiation phase.

One possible explanation for this observation is that the delay may be due to a postgastric cause rather than a true gastroparesis or pyloric obstruction. This is supported by previous reports that demonstrated functional disturbance in the proximal jejunal loop[48]. It is also supported by the reported finding that preserving vagal pyloric fibers did not improve gastric emptying after PPPD[49]. A third evidence is that stasis in the proximal jejunal loop was also reported in association with postcibal asynchronism in B-II patients[50]. Again, disturbed intestinal myoelectric activity after B-II was also reported with suppression of intestinal response to a meal[51].

The results of this work suggest that the B-II reconstruction is associated with a specific form of late DGE which basically involves the actual emptying process rather than its initiation.

Several methods have been attempted to reduce or manage DGE in PPPD. They can be grouped into: pharmacological, nursing and surgical methods. Pharmacological agents that were tried for DGE included proton pump blockers[52], erythromycin[53], motilin[54] and Cisapride[45]. Manipulating administered food according to the postoperative phase was also attempted, priming with fluids which were observed to be associated with better emptying than solids[30].

Suggested surgical methods to avoid DGE included: care about vascularity of the duodenal stump[6], preservation of pyloric vagal fibers[32], resorting to antecolic duodenojejunostomy[55], or employing the probably more physiologic B-I reconstruction[48].

The second functional problem in the pyloric sphincter after PPPD is incompetence which is evidenced by BGR. This problem has been well documented in the late postoperative period after B-II or Roux-en-Y reconstruction[6,31,37,38]. In these series, BGR was observed in approximately half of the patients after B-II reconstruction of PPPD. In B-I reconstruction, BGR was reported to be absent 2 months after operation[48]. In this work we found bile stasis to be the more frequent disorder after PPPD. Reflux was minimal and limited to patients with B-II reconstruction.

CONCLUSION

In conclusion, despite the continuous progress in surgery for this disease, which resulted in a steady decrease in operative mortality, the results of surgery for periampullary carcinoma are not, at the present time, gratifying. This is mainly due to 3 factors: the high incidence of early postoperative morbidity, the poor long-term survival and the poor quality of life in survivors particularly as regards nutritional state. This should encourage the search for technical alternatives in order to improve the results particularly for functional aspects.

The described advantages of B-I reconstruction over B-II (milder DGE and lower incidence of BGR, antral gastritis and reflux esophagitis) can be added to those previously reported by other authors. One example is the physiological mixture of food and fresh bile in the jejunum due to leaving no blind intestinal segment and simulating the normal anatomical arrangement[48]. This results in a better weight gain in the long term follow-up[56].

On the other hand, both procedures have comparable technical feasibility and acceptable morbidity and mortality rates as reported here and previously[28]. These factors suggest that B-I reconstruction is probably the better alternative functionally and that its application in periampullary carcinoma is to be considered.

REFERENCES

1. Howard JM: Development and progress in resective surgery for pancreatic cancer. World J Surg 1999 Sep;23(9):901-6.

2. Whipple AO, Parsons WB, Mullins CR: Treatment of carcinoma of the ampulla of Vater. Ann Surg1935; 102:763–79.

3. Whipple AO: The rationale of radical surgery for cancer of the pancreas and ampullary region. Ann Surg1941; 114:612–15.

4. Watson K: Carcinoma of the ampulla of Vater. Successful radical resection. Br J Surg1944; 31:368–73.

5. Traverso LW, Longmire WP Jr: Preservation of the pylorus in Pancreaticoduodenectomy. Surg Gynecol Obstet1978;146:959–62.

6. Braasch JW, Deziel DJ, Rossi RL, Watkins E Jr, Winter PF: Pyloric and gastric preserving pancreatic resection. Experience with 87 patients. Ann Surg 1986; 204:411–18.

7. Grace PA, Pitt HA, Longmire WP: Pylorus preserving pancreatoduodenectomy: an overview. Br J Surg 1990;77:968–74.

8. Yeo CJ, Cameron JL, Sohn TA, Lillemoe KD, Pitt HA, Talamini MA: Six hundred fifty consecutive pancreaticoduodenectomies in the 1990s: pathology, complications and outcomes. Ann Surg1997; 226:248–60.

9. Watanapa P, Williamson RCN. Resection of the pancreatic head with or without gastrectomy. World J Surg1995; 19:403–9.

10. Imanaga, H. A new method of pancreaticoduodenectomy designed to preserve liver and pancreatic function. Surgery 1960; 47, 577–86.

11. van Berge Henegouwen MI; Moojen TM; van Gulik TM; Rauws EA; Obertop H; Gouma DJ: Postoperative weight gain after standard Whipple's procedure versus pylorus-preserving pancreatoduodenectomy: the influence of tumour status. Br J Surg 1998 Jul;85(7):922-6.

12. Yamaguchi K; Tanaka M; Chijiiwa K; Nagakawa T; Imamura M; Takada T: Early and late complications of pylorus-preserving pancreatoduodenectomy in Japan 1998. J Hepatobiliary Pancreat Surg 1999;6(3):303-11

13. Braasch J: Pancreaticoduodenal resection. Curr. Probl. Surg. 1988; 25:327-368.

14. Wang TY; Shyr YM; Su CH; Wu CW; Lui WY: Comparison of pylorus-preserving and classic pancreaticoduodenectomy. Chung Hua I Hsueh Tsa Chih (Taipei) (China 1999 Mar;62(3):152-8.

15. Klinkenbijl JHG, van der Shelling GP, Hop WCT, van Pel R, Bruining HA, Jeekel J. The advantage of pylorus-preserving pancreatoduodenectomy in malignant disease of the pancreas and periampullary region. Ann Surg1992; 216:142–5.

16. Traverso, LW & Longmire, Wp Jr. Preservation of the pylorus in pancreaticoduodenectomy: a follow-up evaluation. Ann Surg 1980; 192, 306–10.

17. Takada, T, Yasuda, H, Shikata, J, Watanabe, S, Shiratori, K & Takeuchi, T. Postprandial plasma gastrin and secretin concentrations after a pancreatoduodenectomy. A comparison between a pylorus-preserving pancreatoduodenectomy and the Whipple procedure. Ann Surg 1989; 210, 47–51.

18. Kingsnorth, AN, Formela, LJ, Chen, D & Rehfeld, JF: Plasma gastrin and cholecystokinin responses after pylorus-preserving pancreatoduodenectomy and defunctioned Roux loop pancreaticojejunostomy. Br J Surg 1994; 81, 1356–9.

19. Zerbi, A, Balzano, G, Patuzzo, R, Calori, G, Braga, M & Di Carlo, V: Comparison between pylorus-preserving and Whipple pancreatoduodenectomy. Br J Surg 1995; 82, 975–9.

20. Wenger FA; Jacobi CA; Haubold K; Zieren HU; Muller JM: Gastrointestinal quality of life after duodenopancreatectomy in pancreatic carcinoma. Preliminary results of a prospective randomized study: Pancreatoduodenectomy or pylorus-preserving pancreatoduodenectomy. Chirurg 1999 Dec;70(12):1454-9.

21. Zhao Y; Cai L; Zhong S; Zhu Y: 65 cases of preserving pylorus pancreatoduodenectomy: experience and problems. Chin Med Sci J 1994 Sep;9(3):171-5.

22. Tsao JI; Rossi RL; Lowell JA: Pylorus-preserving pancreatoduodenectomy. Is it an adequate cancer operation. Arch Surg 1994 Apr;129(4):405-12

23. Nagakawa T; Ueno K; Ohta T; Kayahara M; Yagi M; Sanada H; Tawara T; Miyazaki I: Evaluation of long-term survivors after pancreatoduodenectomy for pancreatoduodenal carcinoma. Hepatogastroenterology 1995 Apr; 42(2):117-22.

24. Reith HB; Kozuschek W; Traverso LW: Current indications for pylorus saving duodenopancreatic head resection in malignancy. Langenbecks Arch Chir 1996;381(4):207-11.

25. Takao, S, Shimazu, H, Maenohara, S, Shinchi, H & Aikou, T: Modified pancreaticogastrostomy following pancreaticoduodenectomy. Am J Surg 1993; 165, 317–21.

26. van Berge Henegouwen MI; van Gulik TM; De Wit LT; Allema JH; Rauws EA; Obertop H; Gouma DJ: Delayed gastric emptying after standard pancreatico-duodenectomy versus pylorus-preserving pancreaticoduodenectomy: an analysis of 200 consecutive patients. J Am Coll Surg 1997 Oct;185(4):373-9.

27. Yeo CJ: Pylorus-preserving pancreaticoduodenectomy. Surg Oncol Clin N Am 1998 Jan;7(1):143-56.

28. Hishinuma, S, Ogata, Y, Matsui, J & Ozawa, I: Complications after pylorus-preserving pancreatoduodenectomy with gastrointestinal reconstruction by the Imanaga method. J Am Coll Surg 1998; 186, 10–16.

29. Lin PW; Lin YJ: Prospective randomized comparison between pylorus-preserving and standard pancreaticoduodenectomy. Br J Surg 1999 May;86(5):603-7.

30. Kobayashi, I, Miyachi, M, Kanai, M, Nagino, M, Kondo, S, Kamiya, J: Different gastric emptying of solid and liquid meals after pylorus-preserving pancreatoduodenectomy. Br J Surg 1998; 85, 927–30.

31. Lupo, LG, Pannarale, OC, Altomare, DF, Caputi, L, Dell'Erba, L, Ricci, P et al. Is pyloric function preserved in pylorus-preserving pancreatico-duodenectomy? Eur J Surg 1998; 164, 127–32.

32. Morel P, Mathey P, Corboud H, Huber O, Egeli RA, Rohner A: Pylorus-preserving duodenopancreatectomy: long-term complications and comparison with the Whipple procedure. World J Surg1990; 14:642–7.

33. McAfee MK, van Harden JA, Adson MA: Is proximal pancreatoduodenectomy with pylorus preservation superior to toal pancreatectomy? Surgery 1989;105:347-351.

34. Cameron JL, Pitt HA, Yeo CJ: One hundred and forty five consecutive pancreatoduodenectomy without mortality. Ann. Surg. 1993;217:430-438.

35. Fink, AS, DeSouza, LR, Mayer, EA, Hawkins, R & Longmire, Wp Jr: Long-term evaluation of pylorus preservation during pancreaticoduodenectomy. World J Surg 1988; 12, 663–70.

36. Hunt, DR & McLean, R: Pylorus-preserving pancreatectomy: functional results. Br J Surg 1989; 76,173–6.

37. Mosca, F, Giulianotti, PC, Arganini, M, Brandi, LS & Selli, M: Pancreaticoduodenectomy with pylorus preservation. Italian Journal of Surgical Sciences 1984; 14, 313–20.

38. Kozuschek, W, Reith, HB, Waleczek, H, Haarmann, W, Edelmann, M & Sonntag, D. A comparison of long term results of the standard Whipple procedure and the pylorus preserving pancreatoduodenectomy. J Am Coll Surg 1994; 178, 443–53.

39. Williamson, RCN, Bliouras, N, Cooper, MJ & Davies, ER: Gastric emptying and enterogastric reflux after conservative and conventional pancreato-duodenectomy. Surgery 1993;114, 82–6.

40. Christian PE; Datz FL; Moore JG: Confirmation of short solid-food lag phase by continuous monitoring of gastric emptying. J Nucl Med 1991 July;32(7):1349-52.

41. Yung BC; Sostre S: Lag phase in solid gastric emptying: comparison of quantification by physiological and mathematical definitions. J Nucl Med 1993 Oct;34(10):1701-5.

42. Horikawa Y: Gastric emptying of three different size of indigestible radiopaque markers in healthy subjects. J Smooth Muscle Res 1998 Apr;34(2):83-8.

43. Siegel JA; Urbain JL; Adler LP; Charkes ND; Maurer AH; Krevsky B; Knight LC; Fisher RS; Malmud LS: Biphasic nature of gastric emptying. Gut 1988 Jan; 29(1):85-9.

44. Ziessman HA; Atkins FB; Vemulakonda US; Tall J; Harkness B; Fahey FH: Lag phase quantification for solid gastric emptying studies. J Nucl Med 1996 Oct;37(10):1639-43.

45. Ueno T; Tanaka A; Hamanaka Y; Tsurumi M; Suzuki T: A proposal mechanism of early delayed gastric emptying after pylorus preserving pancreatoduodenectomy. Hepatogastroenterology 1995 Jul;42(3):269-74.

46. Takeda T; Yoshida J; Tanaka M; Matsunaga H; Yamaguchi K; Chijiiwa K: Delayed gastric emptying after Billroth I pylorus-preserving pancreatoduodenectomy: effect of postoperative time and cisapride. Ann Surg 1999 Feb;229(2):223-9.

47. Tanaka M, Sarr MG: Role of duodenum in the control of canine gastrointestinal motility. Gastroeneterology 1988;94:622-629.

48. Hermon-Taylor JH, Cade CF: Localization of the duodenal pace-maker and its role in the organization of duodenal myoelectric activity. Gut 1971;12:40-47.

49. Hishinuma S; Ogata Y; Matsui J; Ozawa I: Evaluation of pylorus-preserving pancreatoduodenectomy with the Imanaga reconstruction by hepatobiliary and gastrointestinal dual scintigraphy. Br J Surg 1999 Oct;86(10):1306-11.

50. Nishikawa M; Tangoku A; Hamanaka Y; Suzuki T; Rayford PL: Gastric pH monitoring after pylorus preserving pancreaticoduodenectomy with Billroth I type of reconstruction. J Am Coll Surg 1994 Aug;179(2):129-34.

51. Kotoura Y; Takahashi T; Ishikawa Y; Ashida H; Hashimoto N; Nishioka A; Fukuda M; Utsunomiya J: Hepatobiliary and gastrointestinal imaging after pancreaticoduodenectomy--a comparative study on Billroth I and Billroth II reconstructions. Jpn J Surg 1990 May;20(3):294-9.

52. Shiota M: Clinical and experimental study on reconstruction after pancreatectomy--Part II. Myoelectric activity of small intestine after pancreatoduodenectomy. Nippon Geka Gakkai Zasshi 1988 Aug;89(8):1241-51.

53. Toyota N; Takada T; Yasuda H; Amano H; Yoshida M; Isaka T; Hijikata H; Takada K: The effects of omeprazole, a proton pump inhibitor, on early gastric stagnation after a pylorus-preserving pancreaticoduodenectomy: results of a randomized study. Hepatogastroenterology 1998 Jul-Aug;45(22):1005-10.

54. Yeo CJ, Barry MK, Sauter PK, Sostre S, Lillemoe KD, Pitt HA: Erythromycin accelerates gastric emptying after pancreaticoduodenectomy. A prospective, randomized placebo-controlled trial. Ann Surg1993; 218:229–38.

55. Matsunaga H; Tanaka M; Naritomi G; Yokohata K; Yamaguchi K; Chijiwa K: Effect of leucine 13-motilin (KW5139) on early gastric stasis after pylorus-preserving pancreatoduodenectomy. Ann Surg 1998 Apr;227(4):507-12.

56. Horstmann O; Becker H; Post S; Nustede R: Is delayed gastric emptying following pancreaticoduodenectomy related to pylorus preservation?. Langenbecks Arch Surg 1999 Aug;384(4):354-9.

57. Ogata, Y, Hishinuma, S, Matsui, J, Ozawa, I & Takahashi, S. Pylorus-preserving pancreatoduodenectomy in malignant disease of the pancreatic head and periampullary region. Gastrointestinal Surgery 1995; 18, 49–57.