Prognostic Factors Following Curative Resection for Pancreatic Adenocarcinoma

To the Editor:

The conclusion of this study is drawn from a population-based analysis of 396 patients. The inclusion was based upon the code for operation radical pancreaticoduodenectomy or Whipple procedure, which also included total pancreatectomy, proximal pancreatectomy, partial pancreatectomy and distal pancreatectomy. This means that it includes a wide range of pancreatic cancers. In addition, there is no information about the specific kind of the tumor in case of a process in the pancreatic head region, which in every series of Whipple resection will include a substantial number of ampullary carcinomas and distal bile duct carcinomas. Furthermore, a central pathologist should specifically look for neuroendocrine tumors. Lacking this information and considering the variation in the types of tumor, it is impossible to come to a valid conclusion. This is supported by the fact that the survival rate is extremely high. The 5-year survival rate is depicted in the figures above 30%, median survival time is nearly 18 months, which has to mean that tumors like distal bile duct cancer and ampullary cancer are included in the series and may be also endocrine tumors.

As I have said above, this also means that conclusions about the prognostic factors cannot be made. The title of the article is misleading, for it suggests that prognostic factors be given for pancreatic adenocarcinoma, which usually means carcinoma of the head of the pancreas instead of a variation of cancers. We know that the prognosis of cancer in corpus or tail of the pancreas is worse. On the other hand the prognosis of cyst adenocarcinoma is better. We also know that the prognosis of distal bile duct and peri-ampullary cancer is much better. Thus, conclusions about prognostic factors cannot be made from this study.

One of the most significant findings was the adjuvant combined chemo-radiotherapy as a prognostic factor. This is in contradiction with the evidence that exists in the literature. We live in the era of evidence-based medicine. Evidence should nowadays be based upon level I or level II studies and there are only 2 large prospective randomized studies that can be taken into account, which are the studies of Klinkenbijl et al (Ann Surg 1999; 230:776-782) and Neoptolomos et al (Lancet, November 2001). These 2 large randomized studies clearly indicate that there is no significant effect of chemo-radiotherapy in pancreatic cancer. The study in the Lancet is not even mentioned in the article!

To my surprise these 2 studies are misinterpreted by the authors. They write that: "two prospective randomized clinical trials are in progress," "although preliminary data from the EORTC showed no significant benefit of adjuvant-radiotherapy or chemotherapy on 2-year survival, additional data from this ongoing as well as those from the ESPAC-1 trial, should go a long way toward elucidating the role of adjuvant chemo-radiotherapy in the treatment if pancreatic cancer patients." This is a serious misinterpretation, these trials are not ongoing trials and provide a firm statement with good statistical evidence. Both trials are closed and finished.

The studies that are mentioned by the authors that are consistent with their findings are all retrospective analysis, except for the historic GITSG-trial, which had a insufficient, very small number of patients.

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Are Actual Standard Fluid Regimens in Major Surgery Safe?

To the Editor:

The paper by Brandstrup et al[1] is the first clinical trial to convincingly show that standard fluid replacement methods in colorectal surgery are associated with increased morbidity and mortality. Long ago, fluid and saline solutions were carefully administered during the antidiuretic perioperative phase of surgery, to maintain balance and prevent weight gain.[2,3] However, these cornerstone ideas were somehow replaced in the last decades by the paradigm that surgical patients need exceptional high volume of fluids, irrespectively of measured requirements. The recommended standard fluid replacement[4] seems to drive from principles of goal-directed therapy[5] aimed at increasing cardiac output in high-risk surgical patients. However, the consequences of extrapolating these high requirements to major surgery were not, until now, assessed by clinical trials. Moreover, there is a surprising refusal in surgical and intensive care trainees to admit that excess perioperative fluid could be one of the underlying mechanisms of cardiopulmonary postoperative complications. Paradoxically, despite the lack of the studies supporting the benefit of the recommended high fluid replacement, actual researchers have to demonstrate that the old physiologic approach targeted toward maintenance of body weight is associated with a better outcome.

This paper also shows that the standard group received a greater fluid volume only during surgery and the first postoperative day, however, body weight was maintained higher in this group for 6 days. It must be pointed out that even the restricted group underwent an important positive fluid balance (not described in the paper) revealed by the weight gain (also maintained 5 days) respect the weight recorded the morning

before surgery. Moreover, the author says nothing about the weight of neither the resected colon nor the expected 200-300g daily loss related to postoperative catabolism. These issues probably conceal the real weight gain in both groups and stress the inability of the kidneys to get rid of postoperative fluid excess. The latter findings are in close agreement with our own experience in medium complexity surgery[6] and gives strong emphasizes to the growing risk of fluid overload by the end of the first postoperative week. Brandstrup's outstanding study might be the starting point to answer the basic question: what are fluid requirements during and after major surgery.

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In Reply:

On behalf of "The Danish Study Group on Perioperative Fluid Therapy" I thank Dr. Jorge for the encouraging comments on our work and for giving me this opportunity to discuss the literature and report further details on the trial.

Dr. Jorge points out the fact that liberal fluid therapy in major elective surgery has not been proven beneficial in clinical randomized trials and that data obtained from trials including patients with traumatic injury, shock, sepsis or other acute conditions should not be extrapolated to patients undergoing elective surgery or vice versa. Dr. Jorge suggests that standard fluid replacements may be influenced by the principles of goal directed therapy, investigating the effects of standard fluid therapy (not fluid restriction) versus standard fluid therapy plus additional fluid given to obtain a maximal cardiac output. To discuss these trials in a letter reply would not do them justice, but the very idea of given fluid replacement to maximal cardiac output, intriguing as it may seem, is also to demand maximal work on the heart throughout surgery. Personally, I fail to see the potential benefit of this, especially in elderly patients. The majority of the trials have tested the influence of fluid therapy in combination with other therapy (ie, Dopexamine) and the results of these trials have not been unanimous. Trials of goal-directed therapy investigating effects of fluid therapy alone[1–7] has ended up with a very small volume difference between the groups on the day of operation (200–658 mL). With no registration of the fluid given on the surgical ward, the interpretation of the results of these trials is most difficult. The most exhaustive and recent trial of goal directed therapy in major surgery has failed to show any superiority of the treatment, but on the contrary serious adverse effects.[8]

In relation to our trial, Dr. Jorge points out the fact that in both groups a weight gain was maintained postoperatively and suggest that this weight gain would have been even greater if the weight of the removed colon and the katabolic weight loss were considered. I agree to this notion and to the observed inability of the kidneys to excrete postoperative fluid excess. This is important because tissue edema consequently is maintained for a long period of time. I do not think, however, that the postoperative weight gain in the restricted group reflects a fluid overload. The patients in both groups were allowed to eat and drink freely, but in addition they were fed by tube to obtain a sufficient daily caloric intake. Feeding was commenced 4 hours postoperatively and 500 mL of Nutriconcentrated® was given on the day of surgery. In my opinion, the postoperative weight increase observed in the restricted group was most likely a result of feeding combined with postoperative intestinal paralysis rather than a genuine fluid overload. Currently, however, no simple and clinical feasible method exists to differentiate between fluid in the intestines and fluid in the tissues, and I cannot prove my point of view. Like Dr. Jorge, we find this data interesting and a paper including further data on fluid balance and body weight is in preparation.

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Effects of Intravenous Fluid Restriction on Postoperative Complications: Comparison of Two Perioperative Fluid Regimens: A Randomized Assessor-Blinded Multicenter Trial

To the Editor:

We read with great interest the article by Dr. Birgitte Brandstrup and colleagues[1] in the November 2003 issue of the Annals of Surgery. Their objective was to investigate the effects of restricted versus standard intravenous fluid regimens on complications after colorectal surgery. Having carefully studied the paper we have several comments regarding the design and conclusions of the study.

Although this was a multicenter study they do not mention any protocol or guidelines for fluid management applied during the postoperative period in the standard regimen group. Furthermore, they state that postoperative fluid administration was given according to the "standard" treatment of each hospital and each ward. We find this a significant flaw in the design of the study as major differences in fluid resuscitation can occur between physicians or centers. When assessing the complication rate following an intervention, it would be expected to find some criteria for the prophylaxis measures and the treatment of these complications. In the present study, each 1 of the 8 hospitals used its own routine for antibiotic and antithrombosis prophylaxis.

In addition, the researchers intended to include patients with ASA score group I-III. However, due to their exclusion criteria, 98% of patients included had an ASA score of I-II. Hence, most of their patients were relatively healthy. Our clinical experience taught us that the major problems with fluid resuscitation occur in patients with ASA III-IV.

Regarding the regimens: maintenance fluid support in the restricted group during the surgical procedure and the postoperative period consisted of 5% glucose. The recent trauma and critical care literature are not in favor of this kind of therapy.[2] In addition, the standard group was treated with normal saline and HAES 6%, however, when the recommended dose of HAES was reached albumin 5% was administered. The use of 5% albumin for fluid resuscitation is controversial, especially as it may aggravate edema formation in areas of leaky capillaries.[3,4] Hence, we have to conclude that the regimen and types of fluid used are not the currently practiced nor advised.

The total amount of fluid administered in the operative day ranged between 1100 to 8050 mL in the restricted group and between 2700 to 11,083 mL in the standard group. We noted the large range of fluids administered to different patients in each group. Based on our experience, normally ASA I-II patients undergoing elective colectomies do not require such large amount of fluids.

Although the randomization was computer generated, in the restricted group most of the patients had an ileocolic anastomosis contrary to a minority in the standard group. We find it of importance to note that ileocolic anastomoses are considered less prone to complications. Interestingly, this fact by itself may have caused a bias in the results in favor of the restricted group. Seven percent of patients in the standard group required repeated surgical interventions due to bleeding and the mortality rate of this group was 4.7%. We find these figures alarming in the setting of elective colorectal surgery, moreover in ASA I-II patients. The authors do not state whether each complication occurred in a different patient or could it be that the same patient that had an anastomotic leakage also suffered from sepsis and intestinal obstruction. Although this is quite possible, it would affect the calculated results for the number of complications per patients.

To conclude, the work presented here demonstrated some flaws, which will limit the interpretation and application of these results. Hence, a properly designed study is necessary to identify an adequate fluid resuscitation protocol in postoperative surgical patients.

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In Reply:

On behalf of the "Danish study group on perioperative fluid therapy" I thank Dr. Mintz and colleagues for their comments on our work, and for giving me this opportunity to report further details on the trial.

As pointed out by Dr. Mintz and colleagues, and as reported in the paper, there was no firm protocol for postoperative fluid therapy in the standard group. The standard postoperative protocols of the centers were to give 1–2 L of intravenous fluid the rest of the day of surgery, and thereafter supplement oral fluid for a total amount of 2–3 L daily. I agree that this may have caused some degree of inhomogeneous treatment of the control group and that inhomogeneous treatment could have caused a negative result of the trial due to a small postoperative fluid difference between the groups. Despite of this, however, a marked difference in outcome was found.

Likewise were antibiotic- and antithrombosis prophylaxis given to all patients in accordance with the routine of the departments. These factors were mentioned in the paper because we, like Dr. Mintz and colleagues, find them of importance for outcome following surgery. Other factors may be of equal importance: differences in postoperative mobilization regimens, the use of irrigation, the use of drains and urinary catheters, suture material, anastomosis formation, the skills of the surgeon, etc. As reported in the paper, many confounders were controlled by exclusion criteria, standardization of treatment and stratification of randomization. We realized, however, that controlling all known (and especially unknown) factors of importance for outcome in a multicenter trial was not possible. Instead we relied on the benefit of block randomization to ensure that each center contributed with an equal number of patients in the 2 groups compared. In our opinion, it is of less importance if differences in various treatments and routines exist between centers, as long as the centers contributes with an equal number of patients to the 2 groups compared, where the fluid treatment is the only difference. The block randomization worked very well.

I agree with Dr. Mintz and colleagues that the major problems with fluid resuscitation occur in ASA-group III-IV patients. However, the aim of the trial was to test the hypothesis that standard fluid therapy actually caused cardiopulmonary and tissue-healing complications also in patients with no history of cardiopulmonary diseases. As seen from Table 2 in the paper the number of patients with cardiopulmonary diseases was similar between groups, while the number of patients with postoperative complications was not.

As pointed out by Dr. Mintz and colleagues the restricted regimen and type of fluid is not currently practiced (with the exception of the majority of Danish hospitals and an increasing number of hospitals in the rest of Scandinavia) nor advised. The trial is the first to examine the paradigm that fluid loss should be replaced qualitatively and quantitatively but fluid overload (recognized as a weight gain) should be avoided. Consequently water lost as perspiration was replaced with a "water preparation" ie, glucose 5%. I disagree in the point of view; the literature discourages glucose infusions to normal elective surgical patients. Preoperative glucose load given intravenously or orally has been shown to reduce the postoperative insulin resistance[1,2] and improve the muscle strength.[3] In the only randomized trial found testing the effect of intraoperative glucose administration, the glucose group had the best outcome.[4] New randomized trials are, however, needed to determine the role of glucose infusions in major surgery. As pointed out by Dr. Mintz and colleagues the role of albumin is controversial. I am looking forward to the results of ongoing trials. One major point must, however, be made regarding albumin. Compared with our restricted regimen, all trials of crystalloids versus colloids until this date have tested the effect of fluid overload with crystalloids versus fluid overload with colloids. I am not surprised if overload with colloids may be more hazardous for the patients.

Dr. Mintz and colleagues claim that patients undergoing colorectal surgery normally do not require the fluid volume administered in the trial. Sometimes, however, especially in rectal surgery large blood losses do occur. The patient in the restricted group who received 8050 mL fluid on the day of operation was a 76-year-old woman undergoing rectal extirpation who lost approximately 1.5 times her calculated blood volume. Her weight on the first postoperative day was increased by only 100g. In my opinion, the investigating anesthetist did a splendid job in difficult circumstance.

The patient in the standard group receiving 11083 mL was a 75-year-old male, undergoing low anterior resection who had an initial intraoperative blood loss of 1400, but continued to bleed postoperatively (1100 mL through drains in a few hours) and underwent reoperation pro hemostasis with additional blood loss (and third space replacements).

Dr. Mintz and colleagues are right to emphasize that the level of colonic (but not rectal) anastomosis was different between the groups and may have had a beneficial influence towards the restricted regimen. As already discussed in the paper, we have no reason to believe that the level of colonic anastomosis influences other complications than anastomotic leakage. Only one anastomotic leak registered in this trial affected a colonic anastomosis and this was a leak of the coecum, all other leaks affected anastomosis including the rectum.

Dr. Mintz and colleagues points out that multiple complications in one patient may affect the number of complications per patient shown in the paper's Table 4. Table 4 served several purposes. First, to define the criteria for acceptance of a complication. Second, to show what complications were registered at all. Third, to illustrate the distribution of complications into subgroups. These analyses and the complications included were planned by protocol before anyone knew the results of the trial. Fourth, to illustrate that not only the number of patients with a complication but also the severity and number of complications were marked in-

creased in the standard group. In the R-group no patient had multiple complications, as indeed some patients in the S-group. As reported in the paper, the routine of the department was followed for patients needing reoperation or intensive care (ie, they were given standard fluid replacements). While analyzing these results, it became clear to us that if 1 operation with standard fluid replacement was harmful, then 2 operations with an additional standard fluid load in some cases ended in disaster. Patients treated by the restricted regimen during the primary operation tolerated a secondary operation with standard fluid replacement much better.

I agree with Dr. Mintz and colleagues in the point of view that the results of all trials should be reproduced in additional trials. I hope, however, that these responses to the raised problems will convince the readers of the Annals of Surgery as well as Dr. Mintz and colleagues that despite a few weaknesses, this trial is indeed both properly designed and accomplished.

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Effect of Duodenal-Jejunal Exclusion in a Non-Obese Animal Model of Type 2 Diabetes: A New Perspective for an Old Disease

To the Editor:

I found the article by Rubino and Marescaux[1] very interesting. The author investigated the effect of surgery on type 2 diabetes observed in cases of obese patients with type 2 diabetes who underwent Roux-en-Y gastric bypass (GBP) and biliopancreatic diversion (BPD).[2–6]

To determine if long-term control of blood glucose following surgery was due to the treatment of obesity or to alterations in the enteroinsular axis induced by duodenal-jejunal exclusion, a gastrojejunal bypass (GJB) was performed on non-obese rats with type 2 diabetes. After treatment, reduced fasting glycemia was observed at an even higher level with respect to a control group treated with Rosiglitazone, with increased glucose tolerance, better insulin sensitivity, and lower levels of FFA and cholesterol. More importantly, blood glucose control was achieved in non-obese rats without any postoperative weight loss. This experimental model definitively demonstrates that reduced fasting glycemia and insulin resistance, as well as improved glucose tolerance are attributable to surgery rather than solely to weight loss.[1]

We would like to point out that this concept has already been demonstrated in a series published by Noya et al in 1998, presenting a case study of 10 moderately obese patients (mean BMI of 33.20 kg/m^2) who underwent biliopancreatic diversion preserving the stomach and pylorus, a duodenal-jejunal switch without the restrictive gastric surgery as that proposed by Rubino in his paper, to treat hypercholesterolemia, hypertriglyceridemia and type 2 diabetes mellitus. In all treated patients, cholesterol and triglyceride levels normalized and blood glucose stabilized within normal range in 9 patients during the first few weeks postoperatively, despite the fact that no dietary restrictions were applied and before a significant weight loss was gained.[7]

Rubino attributed the result of diabetes control to duodenal-jejunal exclusion, suggesting a potential role of the proximal gut in the pathogenesis of the disease and putting forward the possibility of alternative therapeutic approaches for the management of type 2 diabetes.[1] In fact, he has focused attention on glucose-dependent-insulinotropic peptide (GIP) a secretin produced by the duodenal K-cells that presents a marked decrease in its insulinotropic effect in type 2 diabetic patients.[1,8]

Though GJB has not produced significant changes on its secretion, the author proposes that the deficit of the enteroinsular system corrected by surgery lies in the bypassed duodenal-jejunal tract. Instead, we believe that the physiopathology of Glucagon-like Peptide 1 (GLP- 1), a secretin produced by the L-cells of the terminal ileum in patients with type 2 diabetes mellitus, and its alterations following surgery can provide a more likely explanation for the resolution of diabetes observed by Rubino.

GLP-1 is a peptide secreted by the L-cells of the terminal ileum in response to nutrients and neural stimuli. It exerts a powerful insulinotropic action, the so-called incretin effect, delays gastric emptying, increases satiety and fullness, and has anabolic, glycogenic and lipogenic actions on liver, fatty and muscle tissues.[9]

In type 2 diabetic patients, the incretin effect of GLP-1 is diminished or disappears entirely, as occurs with GIP, therefore the deficit of entero-insular axis theorized by Rubino has been already demonstrated.[10]

Radioimmunoassay of GLP-1 has shown that this event can be attributed to its reduced secretion in both basal conditions and postprandially. Oral glucose

load during a euglycemic hyperinsulinemic clamp has demonstrated that there is a lower rate of GLP-$1_{7\text{-}36amide}$ in obese patients with type 2 diabetes both before and after load.[11]

A reduced GLP-1 response was also observed following a mixed meal in obese patients with type 2 diabetes, as compared with a control group of normoglycemic obese patients.[12,13] The secretion of GIP, on the other hand, is normal in type 2 diabetic patients, but its effect is lost.[10] The reduced incretin effect of GLP-1 can in fact be attributable to impaired secretion, whereas with GIP the reduced effect can be attributed to a defect of its receptors and this makes GIP useless as a hormone for treating type 2 diabetes.[8]

Moreover, each surgical procedure (jejunoileal bypass, GBP, BPD) that produces the early arrival of food at the terminal ileum triggers the hypersecretion of GLP-1 and accompanies the resolution of type 2 diabetes mellitus, as noted in animal models and in humans.[14–18] Therefore, it is our opinion that GJB has had such an effect on diabetes, as reported by Rubino, because of early arrival of indigested food in the terminal ileum and the consequent stimulation of GLP-1 secretion.

In accordance with Mason, new research prospects are open for surgical methods that can increase GLP-1 secretion even in normal-weight or moderately obese patients:[17] GJB, as proposed by Rubino in animal models and by Noya in humans, may represent one of these methods. This becomes particularly important due to the fact that pharmacological research is trying to develop GLP-1 synthetic analogs with a clinical application for the treatment of type 2 diabetes mellitus. However, their therapeutic utility is still limited by their short half-life (1–2 minutes).[19]

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In Reply:

We thank Dr. Patriti and colleagues for their interest in our paper. One of their arguments is that the concept of a direct antidiabetic effect of bariatric surgery had already been demonstrated by Noya et al with an uncontrolled case-series of 10 moderately obese patients undergoing a stomach-preserving biliopancreatic diversion.[1] We are afraid that this claim is not supported scientifically.

First, a small, uncontrolled case-series type of study is not the proper instrument to demonstrate a direct effect of surgery on type 2 diabetes (T2D) as there are several possible reasons that could justify improved glycemia after a bariatric operation. For instance, since patients undergoing Roux-en-Y gastric bypass (RYGB) or biliopancreatic diversion (BPD) eat small, rather fluid and low-caloric meals in the early postoperative period, it is admittedly impracticable to rule out that the rapid normalization of plasma glucose and improved insulin resistance after these surgeries be simply the effect of decreased caloric intake. To rule out this possibility one would need to do a comparative study with matched subjects undergoing pair-feeding or a period of strict restriction of food intake, ideally with a random allocation. This is a quite difficult study to perform clinically, and has not been done as of yet. We decided to assess the issue with an animal investigation, which, of course, lend itself better to the set up of these experimental conditions.

Although several independent observations documented rapid remission of T2D after RYGB and BPD,[2–4] all these studies had not been designed to specifically test the efficacy of surgery as a treatment of T2D. The indication for surgery was indeed morbid obesity. Hence, the recruitment of patients with diabetes was unintentional and one

could rightfully argue that some of the patients included in these series might not necessarily resemble the typical type 2 diabetic population. Furthermore, due to the strict epidemiologic association many experts believe that a causative role of obesity in type 2 diabetes mellitus is beyond doubt.[5] On the other hand, nonsurgical interventions aimed at fighting obesity and hyperlipidemia have a well known beneficial effect on T2D.[6] Consequently, even if control of T2D precedes significant weight loss we could not totally exclude that the effect is secondary to the treatment of obesity. Surgical weight loss indeed might just be a surrogate marker of the improvement of other metabolic abnormalities related to obesity. The report of Noya et al[1] did not evidently solve these concerns as their observations were made on an obese population with severe hyperlipidemia. However, an interesting findings was that control of T2D occurred in 9 out of 10 patients with mean BMI at 33.2 kg/m^2, supporting the already plausible hypothesis that an arbitrary cut-off at BMI 35 is very unlikely to represent a natural limit for the effectiveness of RYGB and BPD on T2D.

By documenting control of T2D in a non-obese animal model of T2D our study rejects the argument that surgical control of diabetes by duodenal-jejunal bypass is a secondary outcome of the treatment of obesity.

The second argument of Patriti and colleagues is that the stomach-preserving modified BPD performed by Noya et al is the same as our model of duodenal-jejunal exclusion (DJE). This is also inaccurate as the difference is quite substantial.

In fact, the hypothesis that DJE can achieve, "per se," control of T2D,[7] could not be reliably verified by using standard RYGB or BPD techniques, as restriction of the stomach (as in RYGB) and diversion of bile and pancreatic juices down to the terminal ileum (as in BPD) would exert confounding interference and metabolic effects that influence diabetic outcomes.

We therefore developed a model that minimizes influence from other factors. That's why we not only avoided mechanical restriction of food intake by preserving the stomach but we also carefully set the length of the biliary and alimentary limb of our Roux-en-Y reconstruction to minimize the risk of nutrient malabsorption. In fact, unlike the modified-BPD of Noya et al, our model leaves most of the intestinal mucosa exposed to the mix of bile and nutrients and this allows to more specifically address the role of the proximal bowel bypass in the treatment of T2D.

Although we believe our study demonstrated a direct effect of DJE on T2D, it does not explain yet what exactly makes this effect possible. Our study indeed strengthens the hypothesis that an endocrine effect be involved in the surgical control of T2D, but, it remains unclear which hormone response induced by DJE is determinant in the control of T2D. It may be either the production of a "protective" factor enhancing insulin sensitivity and/or insulin secretion or the suppression of a gastrointestinal signal produced in the duodenum-jejunum and causing insulin resistance or strictly involved in its pathogenesis.

As we said in our paper, a possible candidate for the first of these 2 hypothesis is GLP-1, an incretin hormone that enhances insulin secretion. However, whereas increased GLP-1 levels have been reported after jejuno-ileal bypass,[8] more recent studies consistently failed to demonstrate significant GLP-1 changes after RYGB.[9–10] Hence, we would be more cautious than Patriti and colleagues in considering the changes in GLP-1 as the hormonal mechanism by which DJE controls T2D; at least until more evidence becomes available. In contrast, several studies consistently showed that glucose-dependent insulinotropic polypeptide (GIP) levels fall shortly after RYGB[9–10] and we think this has potential implications in surgical control of T2D. Reduced levels of GIP may be a downstream effect of one or more other coordinate hormonal changes that improve insulin sensitivity/secretion. Indeed, the acute insulin response to glucose, which is influenced by GIP,[11] is characteristically attenuated in T2D patients and seems to be normalized by RYGB,[12] in spite of reduced circulating levels of the hormone. This suggests that surgery may reverse a sort of GIP-resistance in T2D.

The demonstration that surgery can directly influence T2D as opposed to being a secondary effect of the treatment of obesity is not a mere intellectual exercise; it has, instead, important implications. One is that it implies the new concept of "diabetes surgery" as an independent new surgical discipline for which surgeons need to develop specific knowledge and competence. Indeed, clinical studies with diabetes-specific endpoints are now justified to define whether or not surgical treatment of type 2 diabetes should be extended also to moderately obese or overweight patients as well as which surgical technique has the best risk/benefit ratio and whether there are specific indications and contraindications for surgical treatment of type 2 diabetes.

We are also confident that our technique of DJE may become a valuable model for diabetes research at large. In fact, future investigations aimed to define its mechanism of action might help finding new molecular targets for medical treatment of type 2 diabetes and possibly even shed light on the causes of the disease.

In a time in which the worldwide increasing incidence of type 2 diabetes poses a very serious threat to health care systems surgeons can and should make their part in the fight against this disease.

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Laparoscopic Adjustable Silicone Gastric Banding Versus Vertical Banded Gastroplasty in Morbidly Obese Patients

To the Editor:

We read with interest the article by Morino et al.[1] We congratulate the authors on their nicely performed first randomized trial to compare different laparoscopic bariatric procedures. The study demonstrates that the laparoscopic restrictive procedure, either laparoscopic vertical banded gastroplasty (LVBG) or laparoscopic adjustable silicone gastric banding (LASGB) are safe, minimally invasive procedures. However, we have reservations about their conclusion that LVBG is more effective than LASGB in terms of late complications, reoperations, and weight loss. We want to provide 2 comments, which might result in this quire conclusion.

The author had a 0% late reoperation rate in LVBG which is very unusual. We started to perform LVBG since 1998 and had accumulated experiences over 600 cases. We had similar results with the authors that LVBG is a safe and effective mini-invasive bariatric operation with only 1% major complication rate reported previously.[2–4] Our procedure had been observed by Mason and regarded as a easy but standard procedure.[5]The excessive weight loss following surgery in our patients is 53.1% at 2 years following surgery, which is similar to the author's results and other studies from European patients.[1,6,7] However, LVBG has a major disadvantage of causing gastrointestinal symptoms, because patients are unable to eat regular food and the incidence of vomiting is very high. In our previous study, we found that the gastrointestinal quality of life improved significantly at 6 months after LVBG but returned to preoperative values at 1 year after surgery, despite an 81% patient satisfaction rate.[8] In addition, some patients may regain body weight gradually after 2 years. Therefore, a reoperation surgery is unavoidable in patients with VBG either due to intolerance or inadequate weight loss.[9] We had a 7.7% accumulated reoperation rate at 5 years after LVBG. Half of the revision surgeries were changing to gastric bypass and the other half were gastrogastric bypass with regain of body weight, all by laparoscopic surgery. The 0% late reoperation rate in the authors' series is unusual. The reasons might be that the follow-up period is not long enough or the surgeons tend to neglect the requirement from the patients.

On the other hand, the authors had a 24.5% reoperation rate in LASGB group and 20% of the bands were removed. This result is also unusual for an experience hand on LASGB. Following its introduction, the technique of LASGB underwent several modifications.[9,10] After the development of techniques of pars flaccida approach for band placement above the bursa omentalis and gastrogastric suturing knots, the reoperation rates of band are reported to decrease to less than 5%. Specifically, O'Brien et al from Australia reported a decreasing reoperation rate for band slippage from 12.5% to only 1% after a learning curve of 350 cases.[11] We started to perform the LASGB since 2001 after learning the technique from O'Brien. We had accumulated 81 cases of LASGB until now with a 0% major complication rate, 0% band slippage and only 2 (1.5%) reoperations are required until now. One patient received port refixation for dislocation of the port and the other required band removal because of intolerance. Therefore, the 18% of band slippage and 20% band removal rate in the authors' study implicated that their techniques are not complete, correct, or are still in the learning curve.

Because the technique performance is not equal in the authors' study, they would have a conclusion that LVBG was significantly superior to LASGB in terms of weight loss under the intension-to-treat principle. This result can not reflect the disparity of LVBG and LASGB but only the maturity of separate technique of the authors. In addition, the comparison of weight reduction between LVBG and LASGB should not be concluded earlier than 3 years following surgery because the different nature of the devices and disparity of the rate of weight loss. The rate of weight loss after LVBG is usually rapid in the first 6 months and then slows down until a plateau is reached 1 to 2 years after surgery. Patients with LVBG are expected to regain some weight after 2 to 5 years

after surgery.[8] On the contrary, the rate of weight reduction after gastric banding is slow and steady. The expected excess weight loss for banding is 30% to 40% at 1 year after surgery. The plateau can be reached 3 years after surgery with a 50% to 60% excess weight loss comparable to the best of LVBG and further weight loss can be expected even after 5 years.[9–12] Our experience with the LSAGB has been more similar to the above experience. Therefore, in contrast to the authors' experience, we have suspend the routine clinical application of the LVBG, and use LSAGB in selected patients in which the advantages of a less complex and totally reversible procedure are the principle requirements determining the surgical technique.

In conclusion, randomized trials to compare different laparoscopic bariatric surgeries are essential for the continuing progress of bariatric surgery. However, because of the technique difficulties and prolonged learning curve of laparoscopic bariatric surgery, a good quality control of surgical procedures are mandatory before a conclusion is made.

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Laparoscopic Adjustable Silicone Gastric Banding Versus Vertical Banded Gastroplasty in Morbidly Obese Patients

To the Editor,

I read with interest the randomized trial published by Dr. Morino and colleagues[1] comparing the laparoscopic adjustable silicone banding (LASGB) with vertical banded gastroplasty in obese patients. I know the difficulties to conduct randomized trials in surgery and congratulate Dr. Morino and colleagues for their timely work. The article typically illustrates the problem of evaluating an emerging and evolving surgical technique, and raises the question of when a randomized trial should be conducted in the life of a procedure. The Italian study began in February 1999, just before the publication of another German randomized trial suggesting that the evaluated LASGB procedure to be no longer considered as a standard.[2]

According to the authors, the LapBand (Bioenterics, Carpinteria, CA) is positioned close to the gastric wall. This procedure is now out of date. The current "standard" is to use the "pars flaccida route." In 2001, a randomized trial[2] suggested the superiority of this technique over that used by Dr. Morino and colleagues. Afterward most bariatric surgeons adopted this technique and observed a significant falling of the rate of pouch dilatation and slippage following LASGB.[3] Thus, the high morbidity rate reported in this trial is, in my opinion, related not to the LASGB itself but to a technique currently abandoned. This fact limits the external validity of this trial and should bring us to interpret the results with caution. One can hypothesize that the "pars flaccida route" could not involve such a high morbidity rate. To illustrate this hypothesis, 2 randomized trials from Sweden have shown that banding carries a smaller risk of reoperation than vertical banded gastroplasty[4] or has similar outcomes[5] which is also in accordance with the conclusions of the review published by Clegg and colleagues assessing the bariatric surgery and invalidating any advantage of vertical gastroplasty over gastric banding.[6]

If one assumes that the results were reported as "means" and on intention to treat basis, one can consider that the poor "mean" results after LASGB in terms of weight loss could be due to removal of 8 bands (nearly 1 patient out of 5). It is obvious that the more bands are removed (because of food intolerance or pouch dilatation or slippage) the more "mean" residual weight excess is important. What about the successful LASGB procedures? In the Swedish trial,[4] weight loss was greater after banding than after vertical gastroplasty.

In conclusion, the trial published by Dr. Morino and colleagues did not favor the LapBand, but it involved a procedure currently abandoned. This consti-

tutes a major flaw, which explains the poor results after banding and hampers the external validity of the trial. In the future, a properly designed trial should evaluate recognized standard procedures.

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Reply:

We thank Dr. Lee and Dr. Slim for their comments and welcome the opportunity to respond. In his letter, Dr. Lee states that a 0% late reoperation rate after laparoscopic vertical banded gastroplasty (LVBG) is unusual compared with his personal experience of 7.7% reoperation rate at 5 years due to food intolerance or inadequate weight loss. In our RCT and in our clinical practice we select very carefully patients to be submitted to restrictive bariatric surgery considering binge eaters, sweet eaters and patients with a BMI > 50 as controindications to LVBG. This clinical attitude probably explains the difference in reoperation rates after LVBG between Dr. Lee's series and our series: inadequate weight loss after restrictive bariatric surgery is common in sweet eaters and in superobese patients,[1–3] while food intolerance is frequent in binge eaters.[2,4] By carefully preoperatively selecting patients many series present low reoperation rates after LVBG: we recently published a 2.2% reoperation rate at 5 years,[3] the Italian national Registry for Bariatric Surgery presents a 1.8% reoperation rate at 6 years.[5]

Dr. Slim states that "two randomized trials from Sweden have shown that banding carries a smaller risk of reoperation than vertical banded gastroplasty[6] or has similar outcomes."[7] In our article we already stated that these 2 trials are at present of limited interest as they compare open bariatric procedures; furthermore Nilsell still uses the original Mason's vertical banded gastroplasty and Lundell uses a non-adjustable Gore Tex band: both techniques are not performed in laparoscopic surgery.

Both Drs. Lee and Slim are surprised by the high reoperation rate after laparoscopic adjustable silicone gastric banding (LASGB) in our series. Furthermore, Dr Slim states that the technique we used is at present abandoned because of the high late complication rates, while Dr. Lee states that the technique we used has in his hands much better results. The disagreement of these 2 authors on the results of LASGB using the perigastric route is in itself an answer: slippage rates using the perigastric route in the literature[8–11] varies between 5% and 21% (ours is 18%), the perigastric route is not at all abandoned as it is demonstrated by Dr. Lee's experience and by numberless recent publications;[8,9,12] in fact the perigastric route is still the most common technique of LASGB in Italy[5] and we suspect all over the world.

Furthermore, Dr. Slim evocates the high percentage of band removal as an explication to the poor results in terms of weight loss in the LASGB group. Eight patients had a band removed in our series as a consequence of slippage and/or intolerance with dysphagia and vomiting; no difference in terms of mean BMI and EWL at 3 years were found comparing these patients to the global LASGB group: BMI 36.2 versus 35.9, EWL 37% versus 39%.

Finally, both authors state that a long learning curve is requested for LASGB. All LASGB in our series were placed by the same surgeon (MM) with a previous experience of more than 5000 laparoscopic procedures, 300 laparoscopic bariatric procedures and 40 LASGB; if a longer learning curve is required very few surgeons will be able to complete it.

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