

# **Surgical Treatment of Type II Diabetes Mellitus**

*Essay*

**Submitted for partial fulfillment of Master Degree in  
General Surgery**

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## **Aim of The Work:**

To spot light on the potential surgical treatment of type II diabetes mellitus.

## **INTRODUCTION**

Diabetes mellitus is a chronic debilitating disease which account for more than 171 million people in 2000 & suspected to be 366 million people in 2030 (**World Health Organization “WHO”, 2005**).

The prevalence of diabetes for all age-groups worldwide (WHO) was estimated to be 2.8% in 2000 and 4.4% in 2030. The urban population in developing countries is projected to double between 2000 and 2030.

The most important demographic change to diabetes prevalence across the world appears to be the increase in the proportion of people more than 65 years of age. These findings indicate that the “diabetes epidemic” will continue even if levels of obesity remain constant. Given the increasing prevalence of obesity, it is likely that these figures provide an underestimate of future diabetes prevalence (*Wild et al., 2004*).

The overall global prevalence of type 2 diabetes is rising steadily. Previously, type 2 diabetes was predominantly a disease of middle-aged and older people. In recent decades, the age of onset has decreased and type 2 diabetes has been reported in adolescents and children worldwide, particularly in high prevalence populations. Japan has seen an approximate fourfold rise in the incidence of type 2 diabetes in 6- to 15-year-olds, and between 8 and 45% of newly presenting children and

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adolescents in the U.S. have type 2 diabetes (*Alberti et al., 2004*).

Diabetes prevalence, costs, and complications are growing at alarming rates in the United States & worldwide. The prevalence of diabetes is increasing at similar rates for men and women. Some complications, such as lower-extremity amputation and end-stage renal disease, are more prevalent among men, particularly among ethnic minority groups. Diabetes is also a significant contributor to erectile dysfunction (*Hardy & Bell, 2004*).

Type II diabetes which is adult onset diabetes mellitus account for 90% -95% from all cases of diabetes. Factors shown to increase the risk of type 2 diabetes are ethnicity (African Americans, Hispanics, and American Indians), physical inactivity, age, obesity, and family history (*Mokdad et al., 2001*).

In the past decade, we have witnessed an epidemic of both type 2 diabetes and obesity. The prevalence of type 2 diabetes has increased by 33% in the United States, and 62% of Americans are classified as obese (BMI more than 30kg/m<sup>2</sup>) or overweight (BMI 25–29.9kg/m<sup>2</sup>). The recent increase in the prevalence of obesity is closely paralleled by the increase in the prevalence of diabetes. Indeed, this new unprecedented phenomenon has been referred to as “diabesity.” There is a clear strong relationship between obesity and the risk for diabetes (*Caprio, 2003*).

Diabetes has a major impact on both affected individuals and society in general, with a total cost in excess of \$132 billion annually not to mention its

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associated increased morbidity and lost quality-of-life years (*Gambert & Pinkstaff, 2006*).

Complications of diabetes including various types of diabetic comas which necessitate urgent hospital admission & long term morbidities. In one study, people with diabetes were reported to be 3.3 times more likely to be diagnosed with ischemic heart disease, 3.1 times more likely with peripheral vascular disease, 2.8 times more likely with hypertension, and 2.3 times more likely with cerebrovascular disease (*Macleod and Tooke, 2001*).

Diabetic glomerulopathy accounts for 25–35% of people entering end-stage renal disease (ESRD) programs, at a cost of > \$1 billion annually (*Gambert & Pinkstaff, 2006*). Diabetic retinopathy & Diabetes is the leading cause of adult blindness in the United States. Visual impairments affect ~ 25% of all adults with diabetes, or nearly 1.6 million Americans. Diabetic retinopathy affects nearly 70% of people with type 1 diabetes and > 60 % of those with type 2 diabetes (*American Diabetes Association, 2004*).

Diabetes is the sixth leading cause of death in the United States, accounting for 3.0% of deaths each year. An estimated 400,000 adults with diabetes die each year (*Gambert & Pinkstaff, 2006*).

Other complications include erectile dysfunction, peripheral neuropathy, urinary tract infections, respiratory infections & pneumonia, skin infections & impaired wound healing & psychological problems. In a study by Eppens and associates indicates that youth with type 2 diabetes have significantly higher rates of microalbuminuria and

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hypertension than their peers with type 1 diabetes, despite shorter diabetes duration and lower HbA1c. The results of this study support recommendations for early complications screening and aggressive targeting of glycemic control in patients with type 2 diabetes (*Eppens et al., 2006*).

Several randomized, controlled clinical trials (RCTs), completed during the past several years or currently underway, are defining the scientific basis of contemporary diabetes care. In terms of glycemic control landmark RCTs have demonstrated that meticulous glycemic control reduces risk of microvascular and neurological complications of diabetes (*Skyler et al., 2004*).

Treatment includes traditional daily physical activity, restriction of carbohydrate intake, weight loss & pharmacological agent including insulin and oral hypoglycemic drugs.

Recently, bariatric surgery was found to be the cause of magnificent resolution of type 2 diabetes in more than 83% of patients (*Schauer et al., 2003*).

Even in absence of obesity, bariatric surgery may be the potential cure of type 2 diabetes (*Rubino and Marescaux, 2004*).

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# Anatomy

## 1-The stomach:

The stomach is the most dilated part of the digestive tube, and is situated between the end of the esophagus and the beginning of the small intestine (*Mc Minn, 2003*).

The shape and position of the stomach are so greatly modified by changes within itself and in the surrounding viscera that no one form can be described as typical (Fig 1-2) (*Susan et al., 2008*).

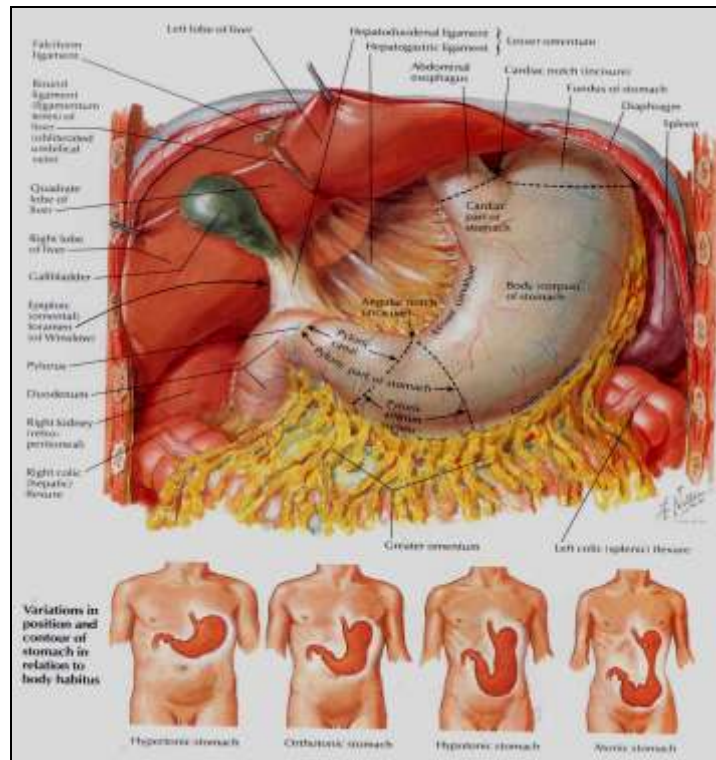


Fig (1-1): Stomach in Situ (*Netter, 2002*).

The lesser curvature gives attachment to the two layers of the hepatogastric ligament, and between these

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two layers are the left gastric artery and the right gastric branch of the hepatic artery (*Snell et al., 2004*).

The cardiac orifice and the lowest portion of the abdominal oesophagus are typically closed at rest by tonic contraction of the lower oesophageal musculature. The gastric mucosa lining the orifice is puckered into ridges. The mucosa is slightly thickened at the cardiac orifice with a raised profile, forming part of the 'mucosal rosette' that lines the orifice. The 'rosette' aids closure of the cardiac orifice and prevent reflux of stomach contents into the oesophagus (*Susan et al., 2008*).

Beside mucosal rosette, **Skandalakis et al.** demonstrated other structures have been held responsible for closing the cardia and preventing reflux from the stomach into the esophagus including the angle of His at which the esophagus enters the stomach, the pinchcock action of the diaphragm, the phrenoesophageal membrane & the sling of oblique fibers of the gastric musculature (*Skandalakis et al., 2006*).

The circular pyloric constriction on the surface of the stomach usually indicates the location of the pyloric sphincter and is often marked by a prepyloric vein of Mayo crossing the anterior surface vertically downwards. Proximally, it merges into the gastric antrum without a definite external boundary; distally, it ends abruptly at the thin-walled duodenum. At its narrowest point, the luminal diameter never exceeds 19 mm. The size is important in estimating the optimal size of artificial openings, such as in gastrojejunostomies or pyloroplasties (*Skandalakis et al., 2006*).

### **Blood supply:**

The stomach is not only well vascularised, but also contains a rich submucosal vascular plexus. The stomach is supplied by left gastric, right gastric, right gastero-

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epiploic and left gastero-epiploic arteries and those directly or indirectly from the celiac axis (Fig 1-2).

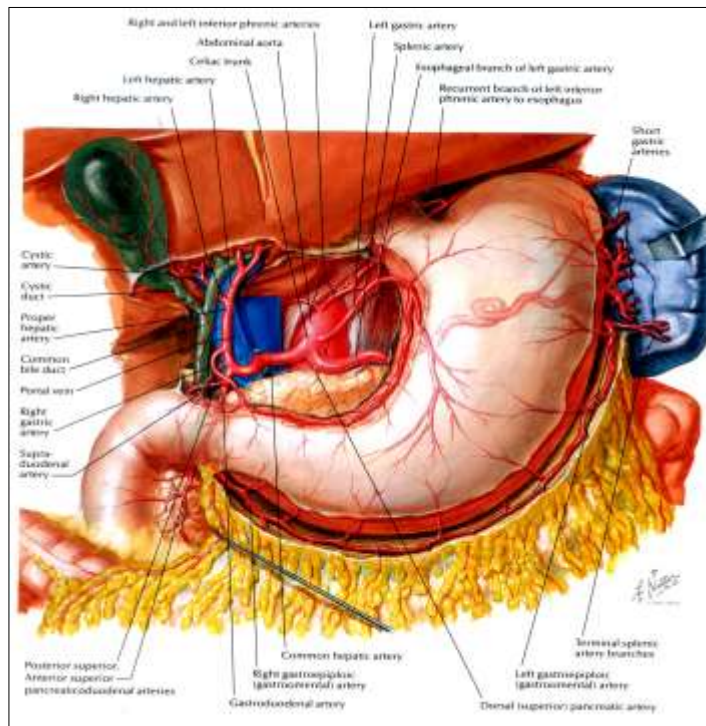


Fig (1-2): The stomach and its arterial supply (*Netter, 2002*).

### **Left Gastric Artery:**

In approximately 90% of individuals, the left gastric artery is a branch of the celiac axis. However, it may arise as an independent unit from the common hepatic artery, splenic artery, aorta, or superior mesenteric artery. In 4% of individuals, it arises from a gastrosplenic trunk; in 3%, it has a direct aortic origin; in 2%, the left gastric is a branch of a hepatogastric trunk. The left gastric artery travels up and to the left retroperitoneally to the proximal one-third of the lesser curvature of the stomach. Here, in approximately 95% of cases, it provides esophageal branches. It may give origin (in 24% of cases) to an aberrant left hepatic branch from which the esophageal and cardioesophageal arteries often arise. This latter vessel

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should be sought when the left gastric artery appears uncommonly large. Identification of an aberrant origin may be of importance during surgical mobilization of the upper stomach (*Skandalakis et al., 2006*).

Along the lesser curvature, it anastomoses with the right gastric artery forming arcade. This arcade gives off delicate branches to the lesser curve but most of its bigger branches pierce the seromuscular coat anteriorly and posteriorly to join the submucous vascular plexus of the stomach (*Cuschieri, 2002*).

The most common variant is an origin from the left hepatic artery, when the left gastric artery passes between the peritoneal layers of the superior lesser omentum to reach the lesser curvature of the stomach (*Susan et al., 2008*).

The left gastric artery commonly divides into an anterior and a posterior branch before attaining the lesser curvature and, in such cases, the esophageal and cardioesophageal arteries may arise from either of those vessels; more commonly, however, the cardioesophageal artery arises from the anterior gastric branch. The anterior branch angles rather obliquely across the body of the stomach toward the greater curvature. It ends in numerous small ramifications and forms a vascular "crow's foot" (of Payne) (*Skandalakis et al., 2006*).

It should be noted that the blood supply of the proximal gastric pouch depends on three sources: (a) the ascending branch of the left gastric artery, (b) the short gastric arteries, and (c) the posterior gastric artery (if present). If the spleen is removed, the short gastric arteries must be sacrificed. Every effort should be made to avoid injuring the ascending branches of the left gastric arteries. Adhesions of the posterior gastric wall must not be cut; as such adhesions may contain the posterior gastric artery (*Skandalakis et al., 2007*).

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### **Right Gastric Artery:**

The right gastric artery is a small branch that arises most commonly from the proper hepatic artery (50% to 68%), left hepatic artery (28.8% to 40.5%), common hepatic (3.2%), or other, less frequent, sources. It gives origin to one or more suprapyloric branches. Anterior and posterior branches from these anastomose with infrapyloric vessels, and, with the supraduodenal artery, supply the distal gastric unit (e.g., the antrum, pyloric canal, and the first inch of the first part of the duodenum). The right gastric artery passes along the lesser curvature for approximately 4 to 6 cm before anastomosing with the left gastric artery (*Skandalakis et al., 2007*).

### **Right Gastroepiploic (Gastro-omental) Artery:**

The right gastroepiploic artery originates from the gastroduodenal artery behind the first part of the duodenum, anterior to the head of the pancreas. It passes inferiorly towards the midline between the layers of the gastrocolic omentum. It lies inferior to the pylorus and then runs laterally along the greater curvature. It ends by anastomosing with the left gastroepiploic artery. It is adjacent to the pylorus but, more distally, lies 2 cm from the greater curvature of the stomach. Gastric branches ascend onto the anterior and posterior surfaces of the antrum and lower body of the stomach while epiploic branches descend into the greater omentum. It also contributes to the supply of the inferior aspect of the first part of the duodenum (*Susan et al., 2008*).

All the branches from the gastero-epiploic arcade pierce the seromuscular to join the submucous vascular plexus of the stomach. The nutritional supply to the stomach (with the exception of the lesser curvature) is derived from this plexus and this account for the viability of the stomach when only one artery (right gastro-epiploic)

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is retained during gastric reconstruction after oesophagectomy (*Cuschieri, 2002*).

It occasionally arises from the superior mesenteric artery or from the anterior superior pancreaticoduodenal artery. Before entering the gastrocolic ligament it gives off infrapyloric branch. For omental viability during distal gastrectomy, the right gastro-omental artery should be preserved by one of the following methods: ligating its gastric branches close to the stomach wall, ligating the anterior omental, or ligating the right gastro-omental distal and close to the origin of the right omental (*Skandalakis et al., 2006*).

### **Left Gastroepiploic (Gastro-omental) Artery:**

The left gastroepiploic artery arises from the splenic artery as its largest branch near the splenic hilum. It runs anteroinferiorly between the layers of the gastrosplenic ligament and into the upper gastrocolic omentum. It lies between the layers of peritoneum close to the greater curvature, running inferiorly to anastomose with the right gastro epiploic artery. It gives off gastric branches to the fundus of the stomach through the gastrosplenic ligament and to the body of the stomach through the gastrocolic omentum. These are necessarily longer than the gastric branches of the right gastroepiploic artery and may be 8-10 cm long. Epiploic (omental) branches arise along the course of the vessel and descend between the layers of the gastrocolic omentum into the greater omentum. A particularly large epiploic branch commonly originates close to the origin of the left gastroepiploic artery, descends in the lateral portion of the greater omentum and provides a large arterial supply to the lateral half of the omentum (*Susan et al., 2008*).

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### **Short Gastric Arteries:**

Approximately five to seven short gastric arteries arise from the terminal branches of the splenic artery or from the left gastro-omental artery. They may appear to emerge from the substance of the spleen at its hilum, having arisen from terminal splenic branches within the organ. The short gastric vessels provide arterial blood supply of the fundus and upper part of the body of the stomach (*Skandalakis et al., 2007*).

### **Posterior Gastric Artery:**

A distinct posterior gastric artery may occur. When present, it arises from the splenic artery in its middle section posterior to the body of the stomach. It ascends behind the peritoneum of the lesser sac towards the fundus. It reaches the posterior surface of the stomach in the gastrophrenic fold (*Susan et al., 2008*).

This artery has received great attention, particularly because of its potential clinical significance. Inadvertent ligation or division of the artery can cause focal necroses or postoperative hemorrhage (*Skandalakis et al., 2007*).

### **Venous drainage:**

The gastric veins parallel the arteries in position and course. The right and left gastric veins drain into the portal vein; the short gastric veins and left gastro-omental veins drain into the splenic vein, which joins the superior mesenteric vein (SMV) to form the portal vein. The right gastro-omental vein empties in the SMV. A prepyloric vein ascends over the pylorus to the right gastric vein. Because this vein is obvious in living persons, surgeons use it for identifying the pylorus (*Moore & Dalley, 2006*).

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