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Confusion between type 1 (T1D) and type 2 (T2D) diabetes among bariatric surgeons

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Introduction: Type 1 diabetes (T1D) is an auto-immune disorder with permanent destruction of the insulin-secreting beta cells.

Methods: This is a literature review.

Results & Description: In juvenile T1D, no insulin is present, and thus glucose cannot enter cells; thus, metabolism switches to oxidation of fatty acids, which can lead to diabetic keto-acidosis. Adult-onset T1D, called LADA (Latent Autoimmune Diabetes in the Adult), has onset between ages 30–55 and makes up 9-25% of diabetes in adults. LADA may be detected by anti-GAD, anti-insulin and/or anti-islet cell antibodies. Endogenous insulin (from pancreatic beta cells) disappears when there is an extremely low C-peptide (connecting peptide, normally split off from pro-insulin). LADA progresses to no surviving beta cells. T1D has vascular complications (retinal, renal, leg) if insulin replacement is inadequate and permits glucose to remain elevated, but generally does not develop metabolic syndrome. Obesity can occur in T1D if the patient takes excessive insulin, gets hypoglycemia, which leads to more intake of food (a vicious cycle). This can be controlled by dietary surveillance; if not, bariatric surgery for obese T1D can decrease weight, HbA1c and amount of exogenous insulin needed (and can lead to episodes of hypoglycemia), but the T1D patient will always require insulin.

Conclusions: In normal-weight T1D, bariatric surgery has no indication (GLP-1 from the lower bowel has no beta cells to act upon). T1D requires caloric control, which may be difficult after a bariatric operation. T2D is a different disease. Initially, the obese T2D patient has both elevated plasma insulin and insulin resistance. However, in longstanding uncontrolled T2D, muscle and fat cells (starved for glucose) continually signal a compensatory increase in insulin production; this leads to beta-cell apoptosis, ultimately requiring exogenous insulin. Bariatric surgery, and especially the MGB-OAGB, results in cure of T2D in a very high percentage of cases.

Speaker Biography

Mervyn Deitel has completed his graduation in Medicine from the University of Toronto in 1961 and trained in Surgery at Beth Israel, Bellevue and NY University Hospitals in New York, Roswell Park Cancer Institute in Buffalo and Trauma at Parkland Memorial in Dallas. He has started IV Hyperalimentation (TPN) in 1967 in Canada and started Bariatric Surgery in Canada in 1970 with JI-bypass, later gastroplasties and RYGB. He was a Past Professor of Surgery and of Nutritional Sciences at University of Toronto and was Founding Member of the ASBS in Iowa 1983. He was the President of ASBS 1994-1995 and was awarded the Outstanding Achievement Award of the ASMBS Foundation in 2004. He has founded the Obesity Surgery journal in 1991 and was the Editor-in-Chief from 1991-2008. He was awarded the IFSO Golden Pin in 1997 and IFSO Honorary Life Membership in 2003. He has 202 papers in PubMed and wrote 5 textbooks on Nutrition and on Bariatric Surgery, 21 invited book chapters and has made >500 invited presentations. He served on the Editorial Board of *Journal of American College of Nutrition*, and is an Advisor in Nutrition to the *American Journal of Family Practice*. He is Chief Advisor of the International Bariatric Club.

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