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007. The effects of RYGB on tissue insulin sensitivity, beta cell function and post-meal glucose flux are maintained 7 years after surgery in both diabetic and non diabetic patients.

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Purpose

The improvement in Type 2 Diabetes (T2D) after gastric bypass (RYGB) is accompanied by change in insulin sensitivity (IS), β -cell function, post-meal glucose ux. Long-term studies indicate that the remission rate of T2D was higher at 2 that 10 years post-surgery. Most of the studies analysed the effects of surgery on glucose metabolism in the early years after surgery Our aim was to determinate if the effects of surgery on tissue IS, post meal glucose flux and β -cell function (β -GS) are maintained long term after surgery.

Methods

We recalled 24 patients (14 T2D and 10 nondiabetic (ND)) the underwent RYGB 7 years earlier (7ys). In each patient we performed the same protocol that was performed Before RYGB (B) and 1 year later (1y). The protocol consisted in a mixed meal test (MTT) and euglycaemic-insulin-clamp combined with glucose and glycerol tracer to measure β -cell function (β -GS), glucose fluxes, adipose tissue insulin resistance (AT-IR), hepatic Insulin Resistance (H-IR), muscle IS (M/I).

Results

Both ND and T2D patients at 7 ys regained 15 ± 6 % of weight lost 1y after RYGB. T2D was resolved 1y post-surgery and this outcome was maintained at 7ys (HbA1c 56 ± 6 vs 36 ± 1 vs 41 ± 2 mmol/mol; B, 1y and 7ys). M/I improved at 1y (from 7.1 ± 1.5 to 13.5 ± 1.0 in ND and from 5.4 ± 0.8 to 13.8 ± 1.4 in T2D), $p < 0.001$, and maintained at 7ys in both ND and T2D (16.5 ± 2.5 in ND to 13.5 ± 1.5 nmol. kgffm⁻¹. min⁻¹.PM⁻¹ in T2D; $p = ns$ vs 1y). Hepatic-IR improved at 1y (from 0.9 ± 0.2 to 0.49 ± 0.08 in ND and 1.05 ± 0.2 to 0.72 ± 0.10 in T2D, $p < 0.02$) and maintained at 7ys (0.52 ± 0.10 in ND to 0.59 ± 0.12 nmol. kgffm⁻¹. min⁻¹.PM⁻¹ in T2D; $p = ns$ for both). The same results for AT-IR that improved at 1y ($p = 0.03$) and maintained at 7ys in both ND and T2D ($p = ns$). Plasma glucose prole and the dynamic of the oral glucose Ra were similar in ND and T2D at 1y and 7ys . Post meal suppression of Endogenous glucose production during the rst 90 min was improved in both groups at 7ys compared to 1y. (AUC of EGP 0-90 $p < 0.05$, 1 yr vs 7 yrs, for both group). In T2D, the improvement in β -GS seen at 1y (33 ± 5 to 64 ± 8 pmol.min⁻¹.m⁻².mM⁻¹, $p = 0.001$) was maintained at 7ys (79 ± 15) at a similar level as in ND (136 ± 16 vs 88 ± 8 vs 83 ± 9 , B, 1- and 7ys).

Conclusions

In both ND and T2D, RYGB induces marked improvements in glucose tolerance, insulin sensitivity (muscle, liver, adipose tissue) and β -cell function that are maintained 7 years after surgery.