下降，起到了终止摄食信号的作用。GLP-1通过降低食欲，有助于减轻体重[17]；⑥能延迟胃排空，进而减缓食物消化，降低餐后血糖。目前已有实验证明在GBP术后胰岛素的分泌增加和GLP-1水平升高相关[18]。有大量研究显示，GBP术后的餐后GLP-1分泌水平明显上升[19]。

构成“肠-胰岛轴”的成员，除了胰岛素、GIP、GLP-1外，还包括胃饥饿素(Ghrelin)、人多肽YY(Peptide YY，PYY)、缩胆囊素(Cholecystokinin，CCK)、瘦素(Leptin)、脂联素(Adiponectin/ADPN)以及胰岛素样生长因子-1 (Insulin-like growth factor 1，IGF-1)等。此外，多种炎症介质，包括肿瘤坏死因-α (TNF-α)、白细胞介素-1β (IL-1β)、白细胞介素-6 (IL-6) 和C-反应蛋白(CRP)等，亦可能参与了血糖调控、胰岛素抵抗[20]，在糖尿病的发病过程中起到了重要作用。近年来已有学者提出糖尿病可能属于炎症性疾病。研究表明GBP术后患者IL-6、CRP、TNF-α水平均有不同程度的降低，脂肪组织内的巨噬细胞浸润减少，胰岛素抵抗得以改善[21]。总之，其调控过程是极其错综复杂的。

本实验的缺陷是：(1)选择的大鼠不是糖尿病大鼠；(2)是动物实验；(3)没有对大鼠血液中胃肠道激素的变化及胰岛素的变化进行测定。所以目前还不能直接将本实验结果推断成人体的实际情况。这些都是我们今后需要进一步研究的内容，以期为外科手术治疗T2DM提供更多的基础理论支持。

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(收稿日期：2015-10-09)