First, the mechanisms of islet cell hyperplasia after foregut bypass are not clear. While GLP-1, a hormone produced in the hindgut, is probably a major contributor to improved glucose metabolism after gastric bypass, there are potentially many other factors involved.[1]

Alteration of other gut hormones such as ghrelin can affect counterregulatory hormones after bypass. Other foregut hormones likely play a role in improving glucose homeostasis after bypass. Rubino and colleagues[2] conducted a series of experiments in rodents supporting the theory that exclusion of the duodenum and proximal jejunum directly ameliorates type 2 diabetes independently of food intake, body weight, malabsorption, or nutrient delivery to the hindgut. They suggested that "anti-incretin" signals normally produced in the foregut may be suppressed with a surgical bypass.

Further investigation is warranted in this area to better define the biochemical links between gastric bypass surgery and improvement in—and potentially excess secretion of—insulin postoperatively.[3]

Second, gastric bypass was introduced in the late 1960s, and since then several hundred thousand gastric bypass operations have been performed. After nearly 40 years of experience with this operation, nesidioblastosis has not emerged as a clearly defined or common complication of the procedure.

Finally, the rarity of this problem is offset by the enormous benefit to patients with type 2 diabetes and insulin resistance who undergo gastric bypass surgery. The small number of case reports of nesiodoblastosis after gastric bypass does suggest a relationship between hyperinsulinemic hypoglycemia and surgery that bypasses the foregut. A true causal relationship, though, has not been established, and the benefit of the surgery with regards to diabetes resolution far exceeds the risk of developing nesidioblastosis.

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2. **Rubino F, Forgione A, Cummings DE, et al.** The mechanism of diabetes control after gastrointestinal bypass surgery reveals a role of the proximal small intestine in the pathophysiology of type 2 diabetes. Ann Surg 2006; 244:741–749.
3. **Cummings DE.** Gastric bypass and nesidioblastosis—too much of a good thing for islets? N Engl J Med 2005; 353:300–302.

CORRECTIONS

Drug-eluting stents

(FEBRUARY 2007)

In the review article entitled "Understand-ing and minimizing late thrombosis in drug-eluting stents" published in the February 2007 issue of the *Cleveland Clinic Journal of Medicine* (2007; 74:129–136), a callout in the margin of page 132 was incorrectly included during copy editing, implying worse clinical outcomes with stent thrombosis after drug-eluting stenting compared with bare metal stenting. Although, as described, formation of collateral blood vessels may be inhibited, there is no clinical evidence to date documenting any excess risk in this regard.

Glycemic control (dosage error)

(FEBRUARY 2007)

The article by Dr. Stephen Clement, "Better glycemic control in the hospital: beneficial and feasible" (*Cleve Clin J Med* 2007; 74:111–120) contained a dosage error. The second sentence on page 118, first column, first paragraph, stated "For prolonged NPO status, insulin drip is preferred, with a starting dose of 0.2 units/kg/hour perioperatively." The correct dose should be 0.02 units/kg/hour.