Serum gastrin in duodenal ulcer

Part IV Effect of selective gastric vagotomy

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SUMMARY Serum gastrin has been measured in 30 patients following selective gastric vagotomy. Basal serum gastrin was 52±5-7 pg/ml which was significantly lower than the corresponding level in 50 patients following truncal vagotomy (84±7-9 pg/ml). After a standard protein meal serum gastrin rose to 136±8-3 pg/ml at 60 minutes after the meal. The peak rise above basal levels was significantly lower than that achieved in patients who had undergone truncal vagotomy.

These results complement our previous hypothesis that section of extragastric vagal fibres permits the release of additional gastrin above that expected with the diminution of acid secretion, and hence the decrease in inhibition of gastrin release from the antrum.

Korman, Hansky, and Scott (1972) suggested that truncal vagotomy permits the release of gastrin from the antrum and other sites. They postulated that the mechanism for this release is a combination of diminution in gastric acid secretion and removal of an inhibitor to extragastric gastrin release by section of extragastric vagal fibres.

The validity of this hypothesis has been examined in patients with duodenal ulcer by comparing gastrin levels following selective gastric vagotomy with those following truncal vagotomy. If the hypothesis is correct then selective gastric vagotomy by preservation of extragastric vagal fibres should permit the inhibitor to remain active and prevent the release of extragastric gastrin.

Material and Methods

After an overnight fast, serum gastrin was estimated in 30 patients who had undergone bilateral selective gastric vagotomy and drainage for active duodenal ulcer. The surgical procedure was as reported by Coupland and Cumberland (1971), the operation having been performed from one month to four years previously.

Six females and 24 males comprised the group and their mean age was 50 years with a range of 23 to 76 years. The drainage procedure was pyloroplasty in 23 patients and gastroenterostomy in the other seven. Twenty-eight patients had been tested for completeness of vagotomy either at the time of surgery by the electrical stimulator test (Burge, Roberts, Stedeford, and Lancaster, 1969) or postoperatively by the acid secretory response to insulin hypoglycaemia (Hollander, 1946). Only one patient of those tested had evidence of incomplete gastric vagotomy.

Four patients with a complete gastric vagotomy were then further investigated. Each fasting patient was given a standard protein meal (Korman, Soveny, and Hansky, 1971) and peripheral venous blood collected at -30, 0, 15, 30, 45, 60, 75, 90, 105, and 120 minutes after protein. The protocol was identical to that reported for truncal vagotomy (Korman et al, 1972). The serum gastrin responses could thus be compared. Serum gastrin was estimated by radioimmunoassay (Hansky and Cain, 1969; Hansky, Soveny, and Korman, 1971b).

Statistical analysis of group means was performed by Student’s t test using standard formulae (Snedecor and Cochran, 1968).

Results

Mean ± SEM basal serum gastrin in the 30 patients with selective gastric vagotomy and drainage was 52±5-7 pg/ml (range 16-116 pg/ml). This level was
The figure compares the serum gastrin response to a protein meal in four patients with selective gastric vagotomy and five patients with truncal vagotomy. In the selective vagotomy group serum gastrin rose significantly from a basal level of 52 ± 8·5 pg/ml to a peak of 136 ± 8·3 pg/ml at 60 minutes after protein (p < 0·0005). The peak rise above basal levels of the truncal vagotomy group was significantly greater than in patients with selective gastric vagotomy (p < 0·025).

**Discussion**

The basal gastrin level after truncal vagotomy is significantly higher than in unoperated duodenal ulcer patients (84 and 16 pg/ml respectively). Because induced intragastric neutralization with bicarbonate in the unoperated patients only increased serum gastrin to 45 pg/ml, it was considered that reduction in acid inhibition of gastrin release could not wholly account for the rise in basal gastrin after truncal vagotomy (Korman et al, 1972; Hansky, Korman, Cowley, and Baron, 1971a). It was therefore suggested that vagal section allows the release of additional gastrin from the antrum and other sites, and indeed studies in animals had indicated that this extra release was dependent on section of extragastric vagal fibres (Landor, 1964; Middleton, Kelly, Nyhus, and Harkins, 1965).

The similarity between the basal serum gastrin after selective gastric vagotomy (52 pg/ml) and that found with induced intragastric neutralization in unoperated patients indicates that the rise in basal gastrin after gastric vagotomy is due to reduction in acid inhibition of gastrin release. The significantly higher basal gastrin level after truncal vagotomy than after selective gastric vagotomy confirms that section of extragastric vagal fibres permits the release of additional gastrin.

Comparison of gastrin responses to a protein stimulus are of interest. The absolute rise in serum gastrin was significantly greater after truncal vagotomy (185 pg/ml) than after selective gastric vagotomy (84 pg/ml). These responses complement the difference in basal levels and confirms that substantially more gastrin is released with protein stimulation following section of extragastric vagal fibres.

The role of this additional gastrin released after section of extragastric fibres remains speculative. Certainly acid secretion is similar after both truncal and selective gastric vagotomy (Bank, Marks, and Louw, 1967) so that any effect on acid secretion is insignificant.

Detailed studies of bowel function after selective vagotomy are unavailable but the incidence of

![Diagram of serum gastrin response to protein meal following truncal and selective vagotomy](image-url)
diarrhoea seems to be less than in truncally vagotomized patients (Frohn, Desai, and Burge, 1968; Williams and Irvine, 1966; Coupland and Cumberland, 1971) although Kennedy and Connell (1969) reported no differences. Perhaps the additional gastrin has a role in the genesis of the diarrhoea as there is some evidence that gastrin affects intestinal motility (Smith and Hogg, 1966). A correlative study of gastrin response to food and the incidence of diarrhoea may provide the answer.

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References


