Effect of highly selective vagotomy upon the lower oesophageal sphincter

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SUMMARY Recently highly selective vagotomy has been suggested as both a cause of gastro-oesophageal reflux and a potential cure. This study was designed to investigate whether this operation produced any change in the resting pressure or the length of the lower oesophageal sphincter in patients undergoing highly selective vagotomy. A group of patients undergoing truncal vagotomy and drainage were also studied for comparison. No alteration in the resting pressure or length of the lower oesophageal sphincter was noted after either operation. It is therefore unlikely that interference with the sphincter is responsible for post-vagotomy reflux.

Symptomatic gastro-oesophageal reflux is a well recognised complication of partial gastrectomy and truncal vagotomy and drainage. Manometric studies have shown that the length of the lower oesophageal sphincter is decreased by truncal vagotomy and drainage, although the pressure remains unchanged, whereas after partial gastrectomy both of these measurements are decreased. Recently it has been reported that the incidence of symptomatic gastro-oesophageal reflux after highly selective vagotomy without drainage may be 25%. Conversely, it has been suggested that highly selective vagotomy is a logical adjunct to an anti-reflux procedure in the management of gastro-oesophageal reflux. In the face of such contradictions this study aimed to define more clearly the effect that vagotomy, particularly highly selective vagotomy, produced upon the lower oesophageal sphincter mechanism.

Methods

Patients

Twenty patients with endoscopically and radiologically proven duodenal ulceration underwent surgical treatment, this being a highly selective vagotomy (HSV) in 14 and a truncal vagotomy and drainage (TV+D) in six.

Lower oesophageal manometry was performed on each subject on two occasions, three days before and at least three months after operation. A station pull-through was carried out in each case by a standard technique which has been shown to give reproducible results in individual subjects on separate occasions. End expiratory pressures were measured in the gastric lumen and in the zone of increased pressure in the lower oesophagus, the difference between these two being the lower oesophageal sphincter pressure (LOSP). The total length of the lower oesophageal sphincter was measured, and the length of the sphincter below the pressure inversion point (PIP), where the respiratory fluctuation changes from positive to negative with inspiration, was also noted.

At the time of manometry all patients were assessed clinically with regard to gastro-oesophageal reflux symptoms.

Results

No patient had symptomatic gastro-oesophageal reflux either pre- or postoperatively.

The manometry results have been expressed as medians, with the range of observations in parentheses. The data have been analysed by non-parametric methods, using Wilcoxon's signed rank test for paired data and rank sum test for non-paired data.

The LOSP in patients with duodenal ulcer before undergoing HSV was 8·7 KPa (2·9-15·6). After operation the pressure was 9·1 KPa (2·9-16·3). In the TV+D group the preoperative value for LOSP was 10·6 KPa (7·3-13·6) and postoperatively it was 10·6 KPa (5·1-17·0). There is no significant

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difference between these values. The normal LOSP by our techniques is 11.39 KPa ± sd. 0.47.

The length of the pressure zone in the HSV group was 3.0 cm (1.0–5.5) before operation, and 3.0 cm (2.0–4.5) afterwards. Similarly, in the TV+D group the pre- and postoperative lengths were 3.5 cm (2.0–4.5) and 3.0 cm (2.5–4.0). Again these values did not differ significantly in the groups or between the groups.

The length of the pressure zone below PIP was unchanged after operation, being 1.5 cm (0.5–5.0) before and 1.5 cm (0.5–3.5) after HSV and 2.0 cm (0.5–2.0) before and 1.5 cm (1.0–2.5) after TV+D.

Discussion

The results of this study indicate that neither highly selective nor truncal vagotomy produces any significant effect upon the resting lower oesophageal sphincter mechanism, as judged by either the length of the pressure zone or the muscular tone generated in it. This is particularly important after highly selective vagotomy because the technique of this operation involves considerable mobilisation of the lesser curvature and fundus of the stomach and the gastro-oesophageal junction. This mobilisation is much more extensive than that necessary in a truncal vagotomy. More recently it has been shown that an adequate denervation in a highly selective vagotomy can be achieved only if at least the lower 4 cm of the oesophagus is also fully mobilised and laid bare. This has consistently been part of the technique of the surgeon who performed the operations in this study. It is therefore reassuring to know that an adequate vagotomy is probably not achieved at the expense of damage to the lower oesophageal sphincter. However, this study did not attempt to measure changes in pressure when the sphincter was challenged by external abdominal compression. In normal subjects this has been shown to lead to an increase in sphincter pressure. This effect is reduced but not abolished by truncal vagotomy and drainage but no similar study has been reported after highly selective vagotomy.

Our finding, that truncal vagotomy also does not affect the lower oesophageal sphincter length, is at variance with the results of Thomas and Earlam. However, they used a non-perfused catheter system which had poor recording fidelity and thus was not suitable for accurate delineation of the sphincter. Although the number of patients in this study was small, the manometric technique used has been shown to give reproducible results.

The absence of symptoms of gastro-oesophageal reflux in the subjects studies may also be a reflection of the small numbers in the study, because reflux is often an intermittent and short-lived problem. Objective evidence of reflux is difficult to measure in patients after surgery for duodenal ulceration. Manometry of the lower oesophagus by itself does not differentiate between those subjects with reflux and those without. One of the best tests for abnormal gastro-oesophageal reflux is prolonged monitoring of the distal oesophageal pH. However, after vagotomy, acid secretion is greatly reduced and this form of investigation cannot show accurately the presence of refluxed fluid. The Standard Acid Reflux Test, in which the patient performs various manoeuvres to provoke the reflux of instilled acid, would overcome this particular problem but does not accurately reflect the reflux status of the individual. Because of these difficulties, this study was not designed to look for reflux, instead it aimed to record the resting pressure and length of the lower oesophageal sphincter accurately, both before and after operation. Both of these indices of sphincter function were unchanged by either surgical procedure.

The addition of a truncal vagotomy and drainage to an anti-reflux operation, in the belief that this would further lessen the chance of continuing reflux by reducing acid secretion, has not found favour. The results of this double procedure have in fact been worse than those of an anti-reflux procedure alone, mainly because of a high incidence of post-vagotomy diarrhoea and dumping. These complications are most unusual after highly selective vagotomy. On purely technical grounds our findings do suggest that no direct harm will accrue to the lower oesophageal sphincter after highly selective vagotomy. However, Kennedy, et al. found that the combination of a highly selective vagotomy and a Nissen fundoplication led to an increased risk of necrosis of the lesser curvature of the stomach. On balance it would seem that the case for adding a highly selective vagotomy to an anti-reflux procedure is not proven at the present time.

Conclusion

Neither highly selective vagotomy nor truncal vagotomy produces any change in the resting lower oesophageal sphincter as judged by its pressure or length. Thus, it seems unlikely that either of these surgical procedures significantly interferes with the resting function of the lower oesophageal sphincter mechanism.

The response of the sphincter to challenge by abdominal compression after highly selective vagotomy is yet to be determined.
References

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