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THE GASTRO-OESOPHAGEAL JUNCTION BEFORE AND AFTER OPERATIONS FOR DUODENAL ULCER

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SUMMARY

Thirty-seven patients with duodenal ulceration were studied with oesophageal manometry preoperatively and then again 6–12 months after operation. In 28 patients who had been treated by truncal vagotomy and either a pyloroplasty or a gastroenterostomy the length of the zone of elevated pressure at the gastro-oesophageal junction was reduced, although the maximum pressure was unchanged. In 9 patients who had had a Polya partial gastrectomy the zone was also shortened, but there was a significant fall in the maximum pressure as well. It is suggested that this reduction of the resting pressure in the sphincter may account for the gastro-oesophageal reflux which is frequently seen after a Polya partial gastrectomy, and may be caused by low circulating gastrin levels since the antrum has been removed.

GASTRO-OESOPHAGEAL reflux is known to occur after a partial gastrectomy and there have been several reports of oesophagitis and oesophageal stricture after such operations (Bingham, 1958; McKeown, 1958; Benedict, 1960; Cox, 1961). It has been suggested that reflux occurs because the gastro-oesophageal angle is altered by these procedures. There is said to be more reflux after a Billroth-I operation than after a Polya partial gastrectomy (Windsor, 1964), and this is cited as evidence for the loss of this angle since the alleged longitudinal tension after a Billroth-I operation could be expected to eliminate the angle of His. There is, however, no real proof that the gastro-oesophageal angle is different after these two types of partial gastrectomy. Reflux is also present after vagotomy and drainage for a duodenal ulcer (Clarke, Penry, and Ward, 1965) and the loss of the gastro-oesophageal angle cannot be invoked as the cause, so factors other than the loss of a mechanical flap valve may be responsible for reflux. Recent work on the hormonal influences on the sphincter has demonstrated that gastrin increases resting pressures (Giles, Mason, Humphries, and Clark, 1969; Cohen and Lipshutz, 1971; Rosenberg and Harris, 1971), and these hormonal levels are reduced when the antrum is removed (Trudeau and McGuigan, 1971). The present study was undertaken to investigate the gastro-oesophageal sphincter using manometry before and after operations for duodenal ulceration in order to find out whether reflux occurred because the resting pressures were reduced.

PATIENTS AND METHODS

Thirty-seven patients who required surgical treatment for a duodenal ulcer were divided into two groups depending on the type of operation that they underwent. Twenty-eight patients were treated by a truncal vagotomy and either pyloroplasty or gastroenterostomy. Nine patients were treated by a Polya partial gastrectomy with or without a vagotomy. The choice of operation was usually determined by the maximal acid output as determined by the standard pentagastrin test. If more than 40 mEq. H^+ were secreted in an hour after pentagastrin a partial gastrectomy was performed, but this was often done with levels lower than this.

Pressure measurements at the gastro-oesophageal junction were made with techniques which have previously been described (Earlam, 1970). The recording units were passed through the mouth into the patient's stomach. Pressure measurements were made with three water-filled polythene tubes (external diameter 1·65 mm., internal diameter 1·12 mm.). The distal tube was covered with a 0·5-cm.-diameter balloon and the remaining two tubes had lateral openings 3 and 6 cm. from the balloon. SE 4–8 MK 2 transducers converted pressures to electrical activity which was recorded by an SE 2005 6-channel ultra-violet recorder on 15-cm. paper. The units were drawn slowly through the gastro-oesophageal junction at 0·5-cm. intervals to obtain values for the sphincteric pressure in the resting state. Two resting pressure profiles were obtained in each test.

End-expiratory and end-inspiratory pressures were measured throughout the zone of elevated pressure at the gastro-oesophageal junction. From these, mean pressures were calculated for each patient from open-tip and two balloon recordings. The three parameters used were the length of the zone of elevated pressure, the intragastric pressure, and the difference between the mean maximal sphincteric pressure and the mean intragastric pressure which for the purposes of this study has been called the 'barrier pressure'.

Tests were performed in the week prior to surgery and then between 6 months and 1 year after operation. At the time of the postoperative test all the patients were interviewed and assessed clinically with regard to symptoms of gastro-oesophageal reflux. It was not thought justifiable to perform an oesophagoscopy, a Hollander insulin test, or a further pentagastrin test on any of the postoperative patients in the absence of severe symptoms.

RESULTS

The zone of elevated pressure at the gastro-oesophageal junction (*Table I*) measured 4·4 ± 1·1 cm. (mean ± S.D.) in length in the 37 patients taken as a whole group. Following a vagotomy and drainage operation the length measured by open tip was reduced to 3·5 ± 1·0 cm. and after a Polya gastrectomy to 2·9 ± 0·8 cm. A reduction in length was also demonstrated by balloon measurement. The levels

of significance according to Student's *t*-test are shown in *Table I*.

The barrier was not significantly reduced after a vagotomy and drainage operation (*Table I*). Patients who had had a pyloroplasty did not differ from those who had had a gastro-enterostomy. After a Polya partial gastrectomy the barrier pressure was reduced to $3{\cdot}2 \pm 2{\cdot}5$ cm. H_2O by open-tip measurement and to $16{\cdot}3 \pm 8{\cdot}0$ cm. H_2O by balloon recording.

The intragastric pressure was increased after both types of operation (*Table II*).

Of the 37 patients, only 3 had postoperative heartburn, and all of these had had a vagotomy and pyloroplasty. There was neither a hiatus hernia nor a low pressure demonstrated by manometry at the gastro-oesophageal junction in these 3 patients.

Table I.—PRESSURES MEASURED AT THE OESOPHAGEAL JUNCTION BEFORE AND AFTER OPERATION FOR DUODENAL ULCER

Type of Operation and Pressure measured	Open-tip Recording		Balloon Recording	
	Pre	Post	Pre	Post
Total ($n=37$)				
Zone of elevated pressure (cm.)	$4{\cdot}4 \pm 1{\cdot}1$	$3{\cdot}4 \pm 0{\cdot}9$ $P<0{\cdot}001$	$5{\cdot}4 \pm 1{\cdot}5$	$4{\cdot}3 \pm 1{\cdot}3$ $P<0{\cdot}001$
Barrier (cm. H_2O)	$9{\cdot}8 \pm 6{\cdot}4$	$7{\cdot}5 \pm 5{\cdot}1$ $P=0{\cdot}1$	$27{\cdot}1 \pm 13{\cdot}9$	$20{\cdot}8 \pm 12{\cdot}0$ $P=0{\cdot}04$
Truncal vagotomy and drainage ($n=28$)				
Zone of elevated pressure (cm.)	$4{\cdot}1 \pm 0{\cdot}8$	$3{\cdot}5 \pm 1{\cdot}0$ $P=0{\cdot}015$	$5{\cdot}2 \pm 1{\cdot}3$	$4{\cdot}4 \pm 1{\cdot}4$ $P=0{\cdot}03$
Barrier (cm. H_2O)	$10{\cdot}3 \pm 7{\cdot}1$	$8{\cdot}8 \pm 4{\cdot}9$ $P=0{\cdot}35$	$28{\cdot}8 \pm 14{\cdot}9$	$22{\cdot}4 \pm 12{\cdot}8$ $P=0{\cdot}07$
Polya partial gastrectomy ($n=9$)				
Zone of elevated pressure (cm.)	$5{\cdot}2 \pm 1{\cdot}5$	$2{\cdot}9 \pm 0{\cdot}8$ $P<0{\cdot}001$	$6{\cdot}6 \pm 1{\cdot}4$	$3{\cdot}9 \pm 1{\cdot}1$ $P<0{\cdot}001$
Barrier (cm. H_2O)	$8{\cdot}0 \pm 3{\cdot}3$	$3{\cdot}2 \pm 2{\cdot}5$ $P<0{\cdot}005$	$24{\cdot}8 \pm 10{\cdot}3$	$16{\cdot}3 \pm 8{\cdot}0$ $P<0{\cdot}01$

Table II.—INTRAGASTRIC PRESSURE BEFORE AND AFTER OPERATION FOR DUODENAL ULCER

Type of Operation and Pressure measured	Open-tip Recording	
	Pre	Post
Total ($n=37$)	$17{\cdot}9 \pm 5{\cdot}0$	$22{\cdot}6 \pm 4{\cdot}7$ $P<0{\cdot}001$
Truncal vagotomy ($n=28$)	$18{\cdot}6 \pm 5{\cdot}1$	$22{\cdot}8 \pm 4{\cdot}8$ $P<0{\cdot}001$
Polya partial gastrectomy ($n=9$)	$15{\cdot}4 \pm 4{\cdot}3$	$21{\cdot}6 \pm 4{\cdot}7$ $P<0{\cdot}01$

DISCUSSION

The most significant finding in this study is that the zone of elevated pressure at the gastro-oesophageal junction is shortened and reduced in height after a Polya gastrectomy. This finding is compatible with the clinical impression that reflux does occur after such an operation, although in this small series there were no such symptoms. It is suggested that the fall in sphincteric pressure is due to the decrease in circulating gastrin which occurs when the antrum is removed (Trudeau and McGuigan, 1971). Gastrin has been shown to increase the sphincteric tone (Giles and others, 1969; Cohen and Lipshutz, 1971; Rosenberg and Harris, 1971). In patients with the Zollinger-Ellison syndrome, where there is a high circulating gastrin level, sphincter pressures are higher than normal (Isenberg, Csendes, and Walsh, 1971). The reduction in the length of the zone of elevated pressure demonstrates that the sphincter remains at the hiatus and has not been displaced. There is no way of demonstrating by manometry whether a gastro-oesophageal angle exists or not, so that the results of this study cannot settle this particular point.

The pressure characteristics of the gastro-oesophageal junction in duodenal ulceration have already been described in a larger series of patients (Earlam, 1972). The height of the barrier pressure is within normal limits but the length is increased. It was suggested in that study that this increased length in the whole group was due to an increased number of cases with proximally displaced sphincters which allowed reflux. Since none of these patients had a radiologically demonstrable hiatus hernia the displaced sphincter should be considered as a small hiatus hernia which can only be demonstrated by manometry.

Both before and after a vagotomy and drainage operation the resting pressure at the gastro-oesophageal junction is normal. This pressure is produced by a combination of gastro-oesophageal sphincteric squeeze and the superimposed effect of the crura, and it is known that displacement of the sphincter alone may reduce the pressure by eliminating the diaphragmatic component. In the present study the effect of reducing a hiatus hernia would be expected to increase the pressure, just by combining the effect of the sphincter and the diaphragm, but there are obviously other factors which are involved which would counterbalance this effect. Gastrin is well known to have an effect on the gastro-oesophageal sphincter and the resting level of gastrin may be different after a vagotomy (Korman, Hansky, and Scott, 1972), but other gastro-intestinal hormones, such as secretin, cholecystokinin, and pancreozymin (Thomas and Earlam, 1973), also have an effect on the sphincter and the level of these hormones may be altered. There are also other factors, such as acid in the fundus which increases sphincteric pressure (Giles, Humphries, Mason, and Clark, 1969) whereas acid in the antrum reduces the pressure, presumably by the gastrin mechanism. In the presence of so many factors which could be altered by a vagotomy and drainage procedure no conclusion can be drawn from this study as to which reflexes or what hormonal levels have been altered by the operation. More dynamic tests of sphincteric function are obviously needed to test the altered reflexes. This study demonstrates only that the resting sphincteric pressure is unaltered by a truncal vagotomy.

This study has demonstrated that the zone of elevated pressure at the gastro-oesophageal junction is reduced in length after a truncal vagotomy and drainage operation. Any decrease in length can either be an artefact due to the overall reduction in pressure or be caused by the superimposition of sphincteric and diaphragmatic pressures by reduction of a hiatus hernia. Since the barrier pressure was not significantly reduced after a truncal vagotomy the reduction in the

length of the zone of elevated pressure is most likely due to the correction of a hiatus hernia. In a truncal vagotomy the oesophagus is usually reduced well below the hiatus so that the main trunks of the vagus can be divided as they lie near the oesophagus and the oesophageal wall cleared of small fibres. The resulting fibrosis probably prevents the gastro-oesophageal junction from slipping back too far into the chest and it remains reduced. In none of the present series was any stitches inserted into the crura to tighten the hiatus.

These results differ from two previous studies comparing the gastro-oesophageal pressure profiles before and after truncal vagotomy and a drainage operation, which both found that there was a significant fall in pressure (Williams and Woodward, 1967; Mann and Hardcastle, 1968), but these studies were done within a few weeks of the operation. The results of the present study are not incompatible with these earlier findings since the pressures have been measured 6–12 months after truncal vagotomy, when full recovery of the sphincter would be expected.

There is considerable controversy over the technicalities of oesophageal manometry as to whether perfused or non-perfused open-tip tubes should be used. The method used in this study was to fill the small polythene tubes with water without continuous perfusion. Since perfusion always increases the pressures recorded and accentuates differences, the addition of perfusion would have not altered the results. Perfusion techniques differ according to the size and length of the tubing used as well as the rates of perfusion, and therefore have the additional difficulty of standardization. The advantage of the present system is that the technique is exactly the same as that used by many other workers so the results can be compared.

The effect of gastro-oesophageal reflux depends on the nature of the substance being refluxed as well as on the ability of the sphincter to prevent reflux. If the acid and pepsin levels in the stomach have been satisfactorily reduced by removing the antrum and part of the body of the stomach, gastro-oesophageal reflux of the gastrin contents is unlikely to cause symptoms arising from the lower oesophagus, even though some reflux may occur through a weak sphincter. After a vagotomy and drainage operation the level of acid produced by the stomach is not reduced to such a low level and the acidity may be still high owing to an incomplete vagotomy so that there remains enough noxious material capable of being refluxed. Since after such an operation there is no evidence that the sphincter is weakened, the resistance to reflux is usually capable of preventing symptoms even though the gastric contents are more acid than after a partial gastrectomy.

The clinical implications of this study are related to surgical techniques. Since there is evidence that the sphincter is reduced into the abdomen after a truncal vagotomy and drainage operation, there appears to be no rationale for inserting stitches into the crura or maintaining the position of the cardia as a routine, unless the hiatus is known to be wide and flaccid. This study provides no information as to the position of the gastro-oesophageal sphincter after a selective or highly selective vagotomy, in which procedures the lower oesophagus is not so thoroughly mobilized.

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REFERENCES

BENEDICT, E. B. (1960), *Gastroenterology*, **39**, 285.
BINGHAM, J. A. W. (1958), *Br. med. J.*, **2**, 817.
CLARKE, S. D., PENRY, J. B., and WARD, P. (1965), *Lancet*, **2**, 824.
COHEN, S., and LIPSHUTZ, W. (1971), *J. clin. Invest.*, **50**, 449.
COX, K. R. (1961), *Br. J. Surg.*, **49**, 307.
EARLAM, R. J. (1970), *Br. med. J.*, **4**, 714.
— — (1972), *Rendic. Riunioni rom. Gastroent.*, **4**, 69.
GILES, C. R., HUMPHRIES, C., MASON, M. C., and CLARK, C. G. (1969), *Gut*, **10**, 852.
— — MASON, M. C., HUMPHRIES, C., and CLARK, C. G. (1969), *Ibid.*, **10**, 730.
ISENBERG, J., CSENDES, A., and WALSH, J. H. (1971), *Gastroenterology*, **61**, 655.
KORMAN, M. G., HANSKY, J., and SCOTT, P. R. (1972), *Gut*, **13**, 39.
MCKEOWN, K. C. (1958), *Br. med. J.*, **2**, 819.
MANN, C. V., and HARDCASTLE, J. D. (1968), *Gut*, **9**, 688.
ROSENBERG, S. J., and HARRIS, L. D. (1971), *Gastroenterology*, **60**, 711.
THOMAS, P. A., and EARLAM, R. J. (1973), *Br. J. Surg.*, **60**, 306.
TRUDEAU, W. L., and MCGUIGAN, J. E. (1971), *Gastroenterology*, **60**, 725.
WILLIAMS, J. A., and WOODWARD, D. A. K. (1967), *Surg. Clins N. Am.* **47**, 1341.
WINDSOR, C. W. O. (1964), *Br. med. J.*, **2**, 1233.

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