THE GASTRO-CESOPHAGEAL JUNCTION IN PATIENTS WITH DUODENAL ULCERATION

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In seventy patients with radiologically proven duodenal ulcer, the gastro-cesophageal junction was studied manometrically, using open-tip mercury-filled water-filled tubes (internal diameter 1.12 mm) and spherical latex rubber balloons (diameter 0.5 cm). The pressure between the mean intragastric and maximum in the sphincter, measured 9.2 ± 5.6 cm water by open-tip and 57.7 ± 10.0 cm water by balloon recording. The length of the zone of elevated pressure was 4.7 ± 1.4 cm by open-tip and 5.8 ± 1.7 cm by balloon. Twenty-eight (40%) had a zone longer than 5.5 cm by open-tip and 5.5 cm by balloon. Patients with epigastric pain (71%) had a longer zone of elevated pressure by both recording methods than those without pain and lower pressures by balloon only. This is compatible with the hypothesis that epigastric pain of duodenal ulceration may arise from the lower oesophagus, since those with epigastric pain are more likely to have a displaced sphincter which would permit gastro-cesophageal reflux.


METHODS AND CLINICAL MATERIAL

Gastro-oesophageal manometry was performed on 70 patients with radiologically proven duodenal ulceration. The patients were usually studied three to four hours after a meal. A clinical history was taken from each patient with special attention to the exact localisation of symptoms. Subjective descriptions of pain, warmth, burning and heartburn were not separated but the exact anatomical distribution was noted by asking the patient to locate the area in question with one finger and marking this on a diagram. Patients were also asked whether they had regurgitation of gastric contents to the mouth. In considering retrosternal pain and regurgitation an “all or none” assessment was made as to whether they ever occurred, with no attention being paid to intermediate grades of severity or frequency. 10 out of 70 (14.2%) had a hiatal hernia but no special radiological procedures had been performed to demonstrate gastro-cesophageal reflux. 52 (74%) eventually had surgery but some had less severe symptoms that did not warrant an operation.

Pressure measurements were made using three water-filled polythene tubes (int. diameter 1.12 mm). The distal opening was covered with a spherical latex rubber balloon (0.5 cm diameter), and the two remaining tubes had lateral openings 5.0 and 10.0 cm proximal to the balloon. Each tube was connected to an SE 4-18 Mk 2 pressure transducer. The electrical changes produced by the pressure differences were recorded on an SE 2006 6-channel ultraviolet recorder. The units were placed in the mouth, swallowed and passed into the stomach. Pressures were recorded at 0.5 cm intervals as the units were withdrawn into the oesophagus to obtain a resting pressure profile. This was repeated...
and then swallow complexes at different levels in the sphincter and oesophagus were recorded. At each interval the mean of the end-inspiratory and end-expiratory values was measured for both open-tip and balloon. The length of the zone of elevated pressure was defined as the distance between the first rise in pressure above intragastric and the last fall before the intragastric plateau. The mean maximum pressure above the mean intragastric was termed the "barrier" pressure, since it may possibly represent the barrier to reflux of gastric contents into the oesophagus. The resting gastro-oesophageal pressure profile was obtained from the mean of four recordings by open-tip and two by balloon.

**RESULTS**

**Characteristics of the whole group.** The mean length of the zone of elevated pressure at the gastro-oesophageal junction was 4.7 ± 1.4 cm by open-tip and 5.8 ± 1.7 by balloon. The mode by open-tip was 4.0 to 4.2 cm. 42 (70%) had a zone longer than 4.0 cm by open-tip and 43 longer than 5.0 by balloon. The distribution curve for the length was skewed to the left (figure). The "barrier" was 9.5 ± 5.5 cm H$_2$O by open-tip and 27.7 ± 18.0 cm H$_2$O by balloon recording (Table I).

**Incidence of hiatal hernia.** In this study a length of 4.0 cm by open-tip and 5.0 cm by balloon has been accepted as the upper limit of normal for the healthy adult, as shown previously with similar techniques (Atkinson et al., 1957; Code et al., 1962). From the frequency distribution curve (Fig. 1) it may be seen that all patients with a radiologically demonstrable hiatal hernia had an elongated zone. If other upper limits are accepted as normal then 28 (40%) were abnormal by open-tip (> 4.5 cm) and 29 by balloon (> 5.5 cm). The diagnosis of hiatal hernia in this study depended not only on the length of the zone, but on the presence of two respiratory reversals, plateaus and the different swallow complexes in this sphincter which enable it to be located accurately (Code, et al., 1962). 9 out of the group of 18 without epigastric pain had a zone longer than 5.5 cm by open-tip, but with no other evidence of a hiatal hernia. No such patients were noted in the group with epigastric pain. If this sub-group is excluded the incidence of patients with a zone of elevated pressure longer than 4.5 cm by open-tip with definite manometric signs of a hiatal hernia is 19 out of 70 (15%).

**Table I**

<table>
<thead>
<tr>
<th>Zone of Elevated Pressure at the Gastro-Oesophageal Junction</th>
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<tbody>
<tr>
<td><strong>Open-Tip</strong></td>
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<tr>
<td>Total (70)</td>
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<tr>
<td>With epigastric pain (52) 74%</td>
</tr>
<tr>
<td>Without (18) 26%</td>
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<tr>
<td><strong>Balloon</strong></td>
</tr>
<tr>
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</tbody>
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No such patients were noted in the group with epigastric pain. If this sub-group is excluded the incidence of patients with a zone of elevated pressure longer than 5.5 cm by balloon is 19 out of 70 (15%).
Comparison of the groups with and without epigastric pain. 52 patients (74%) with epigastric pain had a shorter zone of elevated pressure than 18 without, both by open-tip and balloon (Table I). The "barrier" was similar in both groups by open-tip measurements, but lower by balloon in those with epigastric pain. Those without regurgitation or retrosternal pain tended to have a shorter zone of elevated pressure, but the figures were not significantly different ($p > 0.05$). The barrier in these groups was similar by open-tip and balloon recordings.

**DISCUSSION**

The gastro-cesophageal junction in duodenal ulcer patients is longer than expected but otherwise has no definite pressure characteristics that can separate it from the normal, healthy population. The "barrier" (mean maximum pressure minus mean intragastric pressure) is within expected limits. Neither the increased gastric secretions nor altered hormonal levels in duodenal ulceration have appeared to change the sphincteric resting pressures.

The association of hiatal hernia with duodenal ulcer has differed greatly in previous studies. Radiologically the percentage varies from 11% (Paulson, Shaw and Kee, 1962; Pridie, 1966) to 25% (Cruze, Byron and Hill, 1959) and on one occasion even to 77% (Burge et al., 1966). In the present study the radiological association was 14%.

In a previous manometric study 15 patients with duodenal ulceration had a normal gastro-oesophageal junction (Mann and Hardcastle, 1968). Oesophageal manometry is a more accurate method than radiology for determining whether the gastro-oesophageal sphincter is displaced. But it now seems likely that not every elongated zone represents a hiatal hernia, because some may have a long intra-abdominal segment of oesophagus, which also gives a lengthened zone. This sub-group is only found in those without epigastric pain and it is presumed that it represents an unusually competent gastro-oesophageal junction. Although zones longer than 4.5 cm by open-tip are considered to represent hiatal hernias, this is an arbitrary division. Any length greater than 4.0 cm may possibly represent a degree of sphincter displacement which cannot be called a definite hiatal hernia. 42 (70%) had a zone longer than 4.0 cm by open-tip of which 9 out of 18 without epigastric pain had no hiatal hernia. The incidence by this standard would be 33 out of 70 (49%). However, for this study a more conservative upper limit of normal is accepted as 4.5 cm, and 13% of patients with duodenal ulceration had a hiatal hernia by oesophageal manometry using this standard.

There is an association between hiatal hernia and duodenal ulcer in this study and others, which is greater than would be expected in a normal population. It is considered that the factors causing ulceration are unlikely to produce a hiatal hernia, so there must be another reason for this association.

If gastro-oesophageal reflux causes the epigastric pain of duodenal ulceration then the presence of a hiatal hernia would permit more acid to reflux into the lower oesophagus and cause pain. Patients without epigastric pain, having a more competent gastro-oesophageal junction that prevents reflux, would be protected from this type of pain. It is suggested that in a hospital series, such as this, those patients with little or no protective mechanism for the lower oesophagus would seek treatment more than those without. This would cause a disproportionate number of patients with duodenal ulcer to have an associated hiatal hernia. In a true epidemiological study of patients with duodenal ulceration the incidence of hiatal hernia and an incompetent sphincter should be normal, but this investigation has not yet been done.

The hypothesis that patients with epigastric pain have a sphincter more likely to allow reflux than those without such pain was tested further by comparing the two groups. The "barrier" was greater in those without pain by balloon measurement only. Since the variation is so great with the balloon, the lack of difference by open-tip is probably more reliable. Acid in the lower oesophagus does not cause pressure changes whether there is pain or not arising from the lower oesophagus (Earlam, 1970), so the type of pain is unlikely to affect the present study. Division of the patients according to symptoms did not produce a significant difference manometrically. In the present study no
attention was paid to whether this occurred occasionally, each year, or almost every day. It is considered that a grading of the severity of these symptoms might have separated the groups.

It is proposed that data from this study support the hypothesis that the epigastric pain of duodenal ulceration arises from the lower oesophagus and that patients with an ulcer can be divided into two groups (1) with epigastric pain and (2) with other abdominal pain. Patients with epigastric pain are more likely to have a hiatal hernia or an incompetent gastro-oesophageal junction which cannot protect the lower oesophagus from acid regurgitation, than those without.

REFERENCES


EARLAM, R. J. (1972): Further experience with the epigastric pain reproduction test in duodenal ulceration. B. M. J., 1, 663.


La giunzione gastro-esofagea in pazienti con ulcera duodenale

Riassunto

L'articolo è dedicato ai rapporti fra ulcera duodenale ed ernia iatral ed è frutto dell'analisi delle pressioni intra-oesofagee in 70 pazienti con ulcera duodenale radiologicamente dimostrati. Ricerche radiologiche precedenti avevano mostrato che l'ulcera duodenale si associa con l'ernia iatral da un minimo dell'11% ad un massimo del 77% dei casi. Nei 70 ulcerosi, che costituiscono l'oggetto del presente articolo, la zona di alta pressione (ZEP) è apparsa più lunga della norma, pari a 4,7 ± 1,4 cm se misurata con cateteri ad estremità aperta ed a 5,8 ± 1,7 cm se misurata con palloncini. Normale si è dimostrata invece la differenza fra pressione massima dello sfintere oesofageo inferiore e pressione intragasstrica: 9,5 ± 5,5 cm H₂O con cateteri ad estremità aperta e 27,7 ± 18,0 cm H₂O con i palloncini.

La diagnosi di ernia iatral è stata posta quando la ZEP, misurata con i cateteri ad estremità aperta, superava i 4,5 cm di lunghezza, poiché in questi casi lo sfintere oesofageo inferiore è sicuramente dislocato al di sopra del diaframma. Seguendo questo criterio l'incidenza di ernie iatrali è risultata del 15%.

Nei pazienti che presentavano dolori epigastrici, l'installazione di acido cloridrico (100 ml 0, 1N) nell'esofago distale risvegliava la sintomatologia dolorosa. La prova aveva invece esito negativo quando il dolore era avvertito in altre sedi addominali. È possibile pertanto che il dolore epigastrico dell'ulcera origini dall'esofago distale, nel senso che la giunzione oesofago-gasstrica sia insufficiente e permetta il reflusso di materiale acido. Quando il dolore corrisponda ad altre sedi, il meccanismo che si oppone al reflusso sarebbe più efficiente. Su la base di questa tesi, l'autore ha diviso i pazienti in due gruppi, a secondo che presentassero o meno dolore epigastrico, ed ha dimostrato che la ZEP era in effetti più lunga nel primo gruppo.

Questi risultati induceranno che gli ulcerosi con sintomatologia dolorosa epigastrica hano lo sfintere oesofageo inferiore dislocato sopra il diaframma e con ogni probabilità anche insufficiente.

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