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THE GASTRO-ŒSOPHAGEAL JUNCTION IN PATIENTS WITH DUODENAL ULCERATION

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In seventy patients with radiologically proven duodenal ulcer, the gastro-œsophageal junction was studied manometrically, using open-tip non-perfused 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 9.5 ± 5.5 cm water by open-tip and 27.7 ± 18.0 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 4.5 cm by open-tip and 5.5 cm by balloon. Patients with epigastric pain (74%) 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 œsophagus, since those with epigastric pain are more likely to have a displaced sphincter which would permit gastro-œsophageal reflux.

INDEX TERMS: Duodenal ulcer. Gastro-œsophageal junction. Hiatal hernia. Manometry.

If patients with a duodenal ulcer have epigastric pain, situated just below the xiphisternum between the rib margins, it can usually be reproduced by instilling dilute hydrochloric acid into the lower œsophagus (Earlam, 1970). It has recently been shown that if this pain has been present at night in the four weeks before the test, it is 100% reliable and can be reproduced in all such patients (Earlam, 1972). The amount needed to cause pain is about 30 ml 0.1N HCl and a similar quantity of sodium bicarbonate solution can cause its disappearance. Duodenal ulcer pain other than in this site can not be reproduced similarly. It was deduced that epigastric pain could arise from the lower œsophagus. If this were true, patients with such pain would be more likely to have gastro-œsophageal reflux than those without. The purpose of the present study was to determine (a) the characteristics of the gastro-œsophageal junction in duodenal ulceration, (b) the association between hiatal hernia and duodenal ulcer manometrically and (c) whether duodenal ulcer patients with epigastric pain were more likely to have an incompetent gastro-œsophageal sphincter than those without.

METHODS AND CLINICAL MATERIAL

Œsophageal 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 on X-ray but no special radiological procedures had been performed to demonstrate gastro-œsophageal 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 2005 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 œsophagus to obtain a resting pressure profile. This was repeated

and then swallow complexes at different levels in the sphincter and œsophagus 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 intra-œsophageal 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 œsophagus. The resting gastro-œsophageal 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-œsophageal 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_2O by open-tip and 27.7 ± 18.0 cm H_2O by balloon recording (Table I).

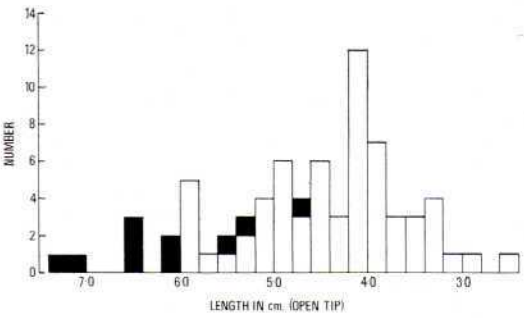

FIG. 1. *Frequency distribution curve for the length of the zone of elevated pressure at the gastro-œsophageal junction. The black squares represent those with a radiologically demonstrable hiatal hernia.*

TABLE I
ZONE OF ELEVATED PRESSURE AT THE GASTRO-ŒSOPHAGEAL JUNCTION

	Open-Tip		*Balloon*	
	Barrier (cm H_2O)	*Length* (cm)	*Barrier* (cm H_2O)	*Length* (cm)
Total (70)	9.5 ± 5.5	4.7 ± 1.4	27.7 ± 18.0	5.8 ± 1.7
With epigastric pain (52) 74%	9.1 ± 4.5	4.8 ± 1.6	25.4 ± 11.6	6.0 ± 1.9
Without (18) 26%	10.5 ± 7.8	4.4 ± 0.8	34.6 ± 29.5	5.3 ± 0.9
	p = 0.16	p = 0.02	p = 0.04	p = 0.06
With regurgitation (43) 60%	9.6 ± 5.5	4.9 ± 1.6	28.5 ± 19.6	6.1 ± 1.9
Without (27) 40%	9.2 ± 5.7	4.3 ± 0.9	26.6 ± 15.3	5.3 ± 1.2
	p = 0.37	p = 0.05	p = 0.32	p = 0.05
With retrosternal pain (34) 50%	9.6 ± 5.4	4.7 ± 1.0	25.6 ± 10.1	6.2 ± 2.1
Without (36) 50%	9.3 ± 4.5	4.4 ± 1.1	29.8 ± 23.2	5.5 ± 1.3
	p = 0.44	p = 0.11	p = 0.17	p = 0.05

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 4.5 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%).

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-œsophageal 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-œsophageal junction (Mann and Hardcastle, 1968). Œsophageal manometry is a more accurate method than radiology for determining whether the gastro-œsophageal 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 œsophagus, 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-œsophageal 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 15% of patients with duodenal ulceration had a hiatal hernia by œsophageal 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-œsophageal reflux causes the epigastric pain of duodenal ulceration then the presence of a hiatal hernia would permit more acid to reflux into the lower œsophagus and cause pain. Patients without epigastric pain, having a more competent gastro-œsophageal 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 œsophagus 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 œsophagus does not cause pressure changes whether there is pain or not arising from the lower œsophagus (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 œsophagus 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-œsophageal junction which cannot protect the lower œsophagus from acid regurgitation, than those without.

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La giunzione gastro-esofagea in pazienti con ulcera duodenale

Riassunto

L'articolo è dedicato ai rapporti fra ulcera duodenale ed ernia iatale ed è frutto dell'analisi delle pressioni intra-esofagee in 70 pazienti con ulcera duodenale radiologicamente dimostrata. Ricerche radiologiche precedenti avevano mostrato che l'ulcera duodenale si associa con l'ernia iatale 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 esofageo inferiore e pressione intragastrica: 9,5 ± 5,5 cm H_2O con cateteri ad estremità aperta e 27,7 ± 18,0 cm H_2O con i palloncini.

La diagnosi di ernia iatale 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 esofageo inferiore è sicuramente dislocato al di sopra del diaframma. Seguendo questo criterio l'incidenza di ernie iatali 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 esofago-gastrica 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 indicherebbero che gli ulcerosi con sintomatologia dolorosa epigastrica hanno lo sfintere esofageo inferiore dislocato sopra il diaframma e con ogni probabilità anche insufficiente.

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