HYPERTROPHY OF THE PYLORIC MUSCLE IN GASTRIC ULCERATION*

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Hypertrophic pyloric stenosis in the adult may be the end-result of conservative management of the infantile variety (in which cases there is an appropriate history) or it may commence in adult life. The latter variety may be the only lesion[1] or it may be associated with other abdominal disorders, of which gastric ulceration is one of the commonest. In such cases it is often difficult to decide whether the ulcer is the cause or the result of the muscle hypertrophy. There is no doubt that the stasis of pyloric hypertrophy can cause gastric ulceration,[1,2] but in many instances there is no history of preceding obstruction, which suggests that the ulcer was the primary event. This paper records some observations in this type of case.

Pyloric Muscle Thickness in Gastric Ulceration

A study was made of an unselected series of 31 stomachs resected for chronic gastric ulceration without any history of obstruction. At operation special care was taken to avoid injury to the pyloric muscle, and immediately after resection the stomach was opened along the greater curvature, pinned out on a board and fixed in 10% formalin in saline. After fixation, sections were cut of the whole thickness of the pyloric region of the stomach wall on the lesser and greater curvatures and anterior and posterior walls midway between the curvatures. The sections were stained with haematoxylin and eosin.

The thickness of the muscle coats was measured at the thickest part of the muscle, using a measuring device on the stage of a microscope.

Similar measurements were made on 10 normal stomachs removed at autopsy on patients without any evidence of abdominal disease and these were used as controls.

Measurements revealed no significant difference in thickness of the longitudinal layer of muscle of the ulcer cases as compared with the controls.

*Date received: 8 January 1969.

TABLE I. CIRCULAR MUSCLE THICKNESS

Site	*Mean of controls (mm.)*	*Mean of GU series (mm.)*	*Difference of means*	*t*	*N*	*P*
Greater curve	3·87	5·47	1·60	2·28	34	3%
Lesser curve	4·09	6·32	2·23	2·63	32	<2%
Anterior wall	4·10	5·02	0·92	1·78	34	10%
Posterior wall	4·06	5·43	1·37	2·04	35	5%

N = number of degrees of freedom.
P = level of significance.

The results obtained with the circular muscle are shown in Table I. The muscle in the ulcer group was significantly thicker than in the control group on the greater and lesser curvatures and on the posterior wall, but on the anterior wall this difference was not significant.

The Effect of Proximity of the Ulcer to the Pylorus

There was no significant correlation between the circular muscle thickness on the lesser curvature and anterior and posterior walls and the distance of the gastric ulcer from the pylorus, but on the greater curvature the coefficient of correlation is -0·66 (- indicates that distance is greater for thinner muscle). This is illustrated in Fig. 1. By the Standard t test this value is highly significant (probability of chance $< 0{\cdot}1\%$ and 95% confidence limits as determined by Fisher's Z method are -0·38 and -0·84).

Time Relationship between the Onset of Muscle Hypertrophy and Gastric Ulceration

In the cases reported above it has been assumed that the ulcer preceded the pyloric hypertrophy because in none of them was there a history of pyloric obstruction preceding the ulcer symptoms. In one additional case this time relationship could be studied accurately.

Illustrative case report. A male, aged 27 years, developed a radiologically proved gastric ulcer in January 1964, when the pylorus appeared normal (Fig. 2). The ulcer

healed on standard medical treatment and in March 1964 the barium meal showed no ulcer and a normal pylorus (Fig. 3). Ulcer symptoms recurred in June 1965 when the barium meal revealed an elongated and narrowed pyloric canal (Fig. 4). He was again treated medically with relief of symptoms until July 1966, when he presented with ob-

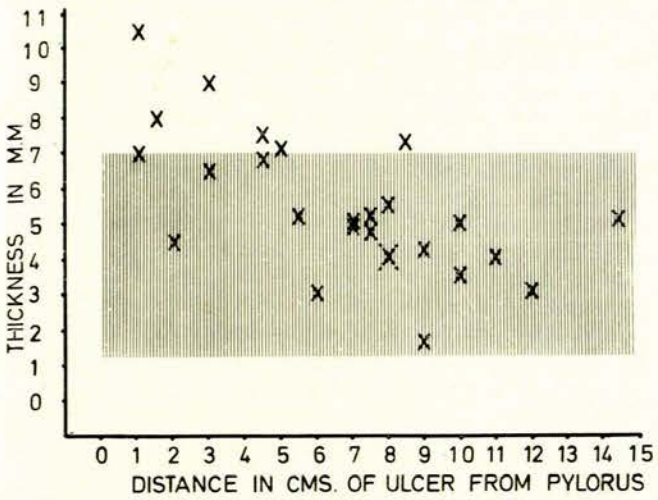

Fig. 1. Relationship between thickness of the pyloric muscle on the greater curvature and the distance of the ulcer from the pylorus. (Shaded area represents the limits of normal controls.)

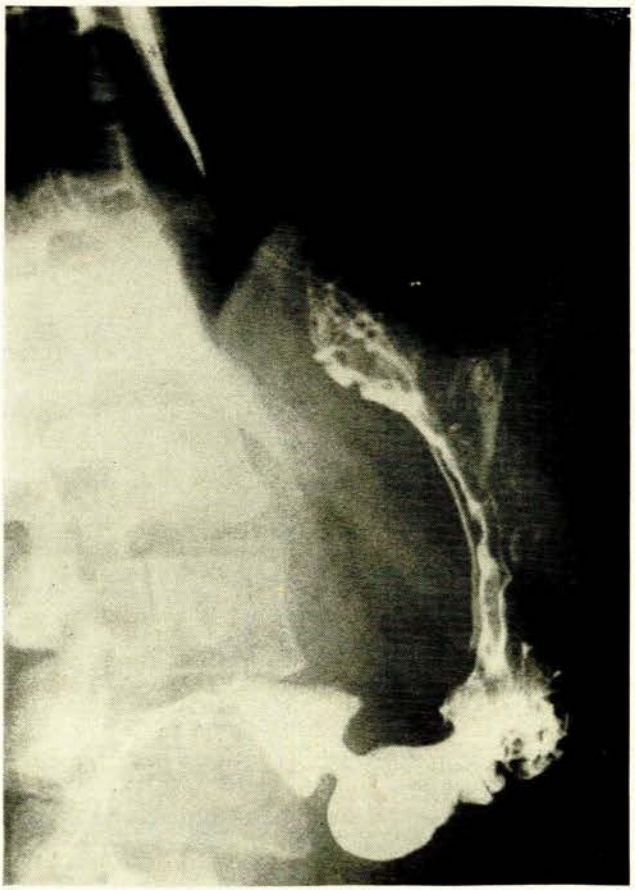

Fig. 2. Gastric ulcer with normal pyloric canal (January 1964).

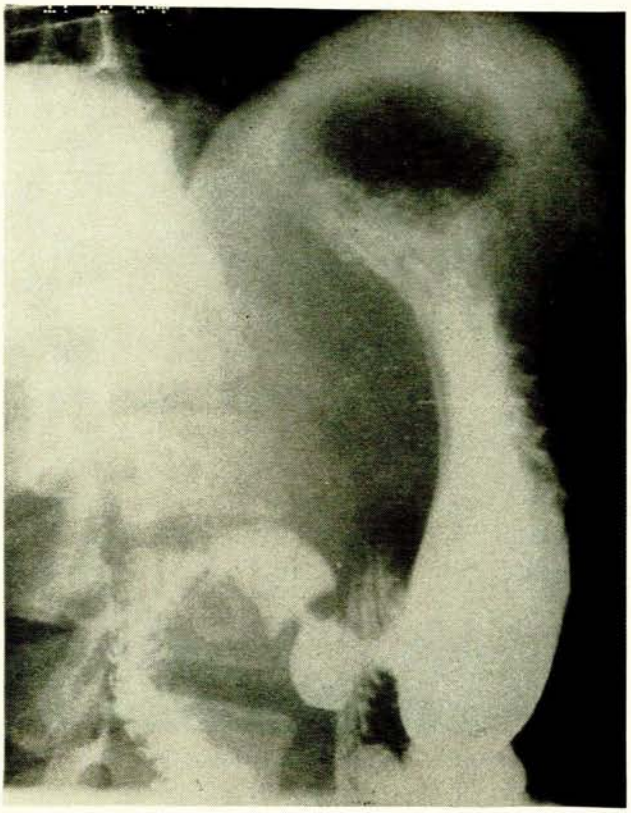

Fig. 3. Gastric ulcer healed and pyloric canal normal (March 1964).

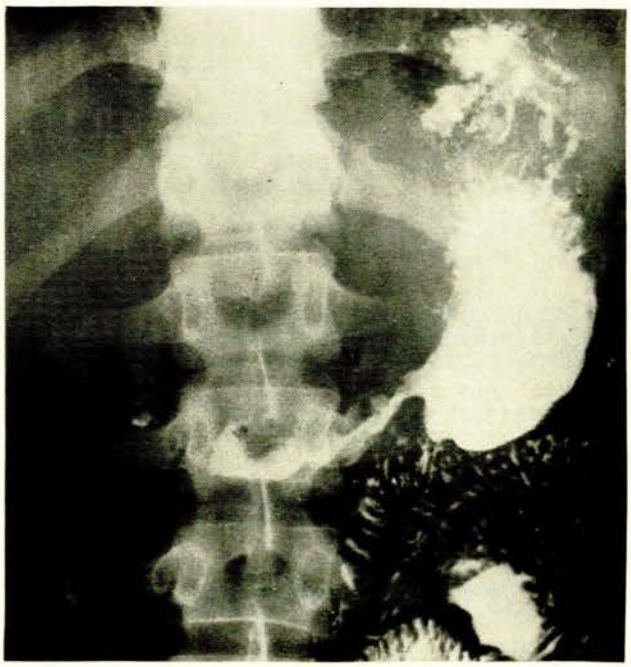

Fig. 4. Elongated pyloric canal (June 1965).

structive symptoms; on barium meal there was no ulcer but an elongated narrowed pyloric canal (Fig. 5), with delay in gastric emptying.

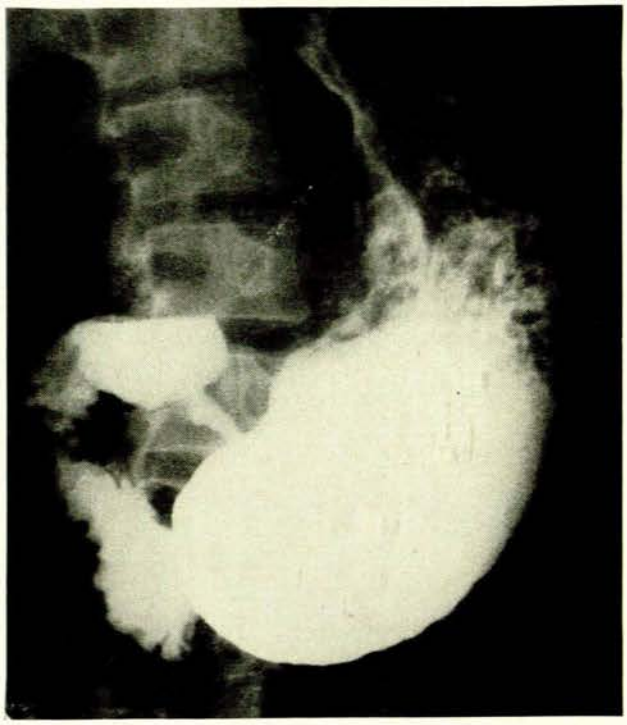

Fig. 5. Elongated and narrowed pyloric canal (July 1966).

A Bilroth I partial gastrectomy was performed with complete relief of symptoms. The stomach was examined as described above.

TABLE II. MUSCLE THICKNESS

	Longitudinal muscle		*Circular muscle*	
Site	*Case (mm.)*	*Mean of controls (mm.)*	*Case (mm.)*	*Mean of controls (mm.)*
Greater curve	0·6	0·9 (0·2 - 2·2)	6·0	3·8 (1·2 - 4·8)
Lesser curve	0·6	0·8 (0·4 - 1·0)	9·4	4·0 (3·2 - 6·0)
Anterior wall	1·1	0·8 (0·2 - 1·4)	4·0	4·1 (2·8 - 6·0)
Posterior wall	0·8	0·6 (0·2 - 1·0)	8·2	4·0 (2·8 - 5·4)

There was no active ulcer but the scar of a healed ulcer was present on the lesser curvature 8 cm. from the pylorus. The pyloric muscle was hypertrophied and measurements of muscle thickness at the pylorus are shown in Table II. The longitudinal layer was within normal limits but there was hypertrophy of the circular layer except on the anterior wall.

DISCUSSION

The association of gastric ulceration and pyloric hypertrophy has been recognized for a long time,[3,4] but most authors have been uncertain about the sequence of events,[5] although Burge[2] considered that the pyloric hypertrophy was the cause of the ulcer. The stasis caused by pyloric stenosis certainly can result in gastric ulceration,[1] but the reverse can also occur,[6] as is actually illustrated by some of the case histories recorded by Burge.[2] In the cases reported here there was no history of obstruction preceding the gastric ulceration, suggesting that the gastric ulcer was the primary event in each case.

The normal thickness of the longitudinal muscle layer in these cases is in contrast to the deficiency of the longitudinal muscle in primary hypertrophic pyloric stenosis in the adult.[1] This suggests that there are two distinct entities which can be differentiated clinically by the sequence of ulcer pain and the symptoms of obstruction, and histologically by the thickness of the longitudinal muscle of the pyloric canal.

This phenomenon may be of significance in the management of gastric ulcers because in the presence of gastric stasis the ulcer is unlikely to respond to medical treatment or if it does it will probably relapse. Pyloric hypertrophy should therefore be specifically looked for on barium meals and if present it probably constitutes an indication for surgical treatment without a preliminary trial of medical treatment, although Albot and Magnier[7] point out that prolonged medical treatment will retard the onset of stenosis.

This secondary type of pyloric hypertrophy should also be kept in mind when considering the pathogenesis of gastric ulceration, because delayed gastric emptying in cases with gastric ulcers will not necessarily indicate that gastric stasis was the cause of the ulcer.

It has previously been reported that pyloric muscle hypertrophy is particularly likely if the gastric ulcer is close to the pylorus[8] but, according to the results reported here, this was only the case along the greater curvature.

It is uncertain how this secondary type of pyloric hypertrophy is produced. It is generally assumed to result from reflex spasm which eventually causes hypertrophy, but although pyloric spasm, reversible with drugs, can be seen in many cases with abdominal disease[9] and can be reproduced experimentally in animals by irritating upper abdominal viscera,[10] the progression from (reversible) spasm to hypertrophy has not yet been proved.

SUMMARY

A study of 31 stomachs, resected for chronic gastric ulceration, has revealed that there is a significant increase in thickness of the circular muscle at the pylorus. On the greater curvature this is more prominent the closer the ulcer is to the pylorus. This muscle hypertrophy may be primary (and so the probable cause of the ulcer) or secondary to the ulcer. Because in these cases there was no history of pyloric obstruction preceding the onset of ulcer symptoms, it is assumed that the hypertrophied pylorus occurred secondary to the gastric ulceration. A case is reported in detail where the ulcer clearly preceded the development of the muscle hypertrophy. This phenomenon must be kept in mind when considering the pathogenesis and management of gastric ulceration.

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REFERENCES

1. Du Plessis, D. J. (1966): Brit. J. Surg., **53**, 485.
2. Burge, H. (1964): *Vagotomy*. London: Edward Arnold.
3. Horwitz, A., Alvarez, W. C. and Ascanio, H. (1929): Ann. Surg., **89**, 521.
4. Truesdale, P. E. (1915): Surg. Gynec. Obstet., **21**, 298.
5. Craver, W. L. (1957): Gastroenterology, **33**, 914.
6. Raffensperger, E. C. (1955): *Ibid.*, **28**, 458.
7. Albot, G. and Magnier, F. (1953): Arch. Mal. Appar. dig., **42**, 347.
8. Foulk, W. T., Comfort, M. W., Butt, H. R., Dockerty, M. B. and Weber, H. M. (1957): Gastroenterology, **32**, 395.
9. Cohn, A. I. and Gold, R. L. (1948): *Ibid.*, **10**, 782.
10. Kirklin, B. R. and Harris, M. T. (1933): Amer. J. Roentgenol., **29**, 437.