as often, the horse and cart are interchangeable. When I say hiatal hernia, I do not of course include the paraœsophageal form, where a slip of diaphragmatic muscle anchors the cardia in the right place.

The following points are of practical bearing from the general pathology of this complex: (1) Hyperacid gastric juice is more dangerous than normal juice – one reason for the association of duodenal ulcers and œsophagitis. (2) The damage in œsophagitis is a good deal greater microscopically than to the naked eye. Gross perforation is not necessary for the complication of mediastinitis; if perforation does occur, it may be into pleura or pericardium, as well as mediastinum and, if below or partly below the insertion of the elastic ligament, there will also be upper peritonitis. (3) Inhalation pneumonia is a risk at any stage, which stresses the importance of posture. The presence of a tube passing into the stomach may be absolutely indicated by other surgical considerations but could not a tube, dangling through the cardia, sometimes promote reflux around it and so increase the risk of inhalation? (4) No age is exempt, infantile œsophagitis being well recognized.

Mr Robert Brain
(Guy's Hospital, London)

Peptic Strictures of the Œsophagus associated with Duodenal Ulcer and Operations for its Relief

This rather rare type of œsophageal stricture is used to illuminate some of the fundamental problems and arguments in the management of peptic strictures in general.

Incidence: This brings one straightaway to an extraordinary discrepancy in the figures given for the association of duodenal ulcer and hiatal hernia. The literature of general surgeons or those primarily involved in seeing and treating patients with duodenal ulcer indicates that the association is common, e.g. both Burge *et al.* (1966) and Casten *et al.* (1963) give an incidence of 50%; others have even higher percentages. But our own figures for the past five years show that we have operated on 234 patients with hiatal hernia, only 9 of whom had associated duodenal ulcers and one an adult type pyloric muscle hypertrophy, giving a total incidence of only 4·2%. This difference is believed to reflect the effects of specialization, patients having been pre-grouped according to whether there was a predominance of the symptoms of reflux œsophagitis over those of duodenal ulcer or vice versa.

However, there is general agreement on the low incidence of post-gastrectomy and post-vagotomy strictures of the œsophagus; consequently they appear in the literature in small series, for example 4 cases by Bingham (1958), 2 by McKeown (1958) and 2 by Hurst (1961). This paper gives our experience of 5 patients following gastrectomy, 2 of them with 'irreversible' type strictures that needed resection, and 3 after vagotomy with strictures of the 'reversible' type (Tables 1 and 2). This distinction separates strictures that are purely 'inflammatory', with little or no fibrous replacement of the œsophageal wall, from those with complete fibrous replacement whose hernias are in addition invariably irreducible.

Case 1 in the post-vagotomy list (Table 2) is especially interesting because, although the patient had had an earlier gastrectomy, it is quite clear from the history that it was a vagotomy six years later that provoked a serious stricture.

Ætiology: Accepting the reflux of acid/pepsin into the œsophagus as the all-important factor, one would expect a high incidence of peptic œsophagitis with the high tone type of stomach liable to cardiac incompetence, hypersecretion and duodenal ulcer. For the same reasons a reduction in acid/pepsin secretion with facilitation for rapid gastric emptying should improve or cure this disease. Why then is there this small but very definite worsening or, as is most usual, a first appearance of severe reflux œsophagitis with rapid stricture formation after an operation designed both to reduce acid/pepsin and to provide easy gastric drainage?

Since the reduction of acid/pepsin after either gastrectomy or vagotomy is a fact, an explanation has been sought on the following lines: (1) Is reflux worse? (2) Is gastric drainage ineffective? (3) Are there other important factors such as trauma from intubation after operation and/or the substitution of alkaline/trypsin enzymes for the acid/pepsin?

Is reflux worse? The overriding importance of reflux in this disease can be appreciated in many ways: one of the most striking is the simple clinical observation that regurgitation and heartburn decrease as, coincidentally, dysphagia increases, in patients with strictures; œsophagoscopy may show healthy squamous epithelium extending down to a tight stricture. For this

Table 1

Peptic strictures associated with duodenal ulcer operations

Case	*Sex*	*Age*	*Operation for duodenal ulcer*	*Duration of intubation*	*Bed position*	*Onset of dysphagia*	*Reflux and hiatal hernia after operation*	*Treatment for stricture*	*Result*
Bingham (1958)									
1	M	41	Gastroenterostomy	2 days	Flat	10 days	Both	Gastrectomy and bouginage	Self-bouginage
2	M	68	Gastroenterostomy		Flat	5 weeks	Both	Bouginage	Self-bouginage
3	M	61	Gastroenterostomy and vagotomy	2 days		1 month	Both	Bouginage	Self-bouginage
4	M	39	1942: Gastroenterostomy 1955: Polya gastrectomy 1957: Billroth I gastrectomy	5 days	Flat	3 weeks	Both	Bouginage Bouginage Gastroplasty (Collis type)	
McKeown (1958)									
1	F	72	Polya/Hofmeister gastrectomy	3 days		3 weeks	Neither	Billroth I gastrectomy	Cured
2	M	64	Polya/Hofmeister gastrectomy			3 weeks	Both	Billroth I gastrectomy	Cured
Hurst (1961)									
1●	F	40	Polya/Hofmeister gastrectomy			2 weeks	Both■	Bouginage	Self-bouginage
2	M	50	Polya/Hofmeister gastrectomy			2 weeks	Neither	Bouginage	Self-bouginage
Brain (1966)									
1	M	58	Polya gastrectomy	2 days	Sitting	2 weeks	Both	Failed bouginage. Colonic transplantation	
2●	M	46	Polya gastrectomy	Nil	Sitting	2 weeks (worse)	Both▲	Failed bouginage. Hiatal hernia repaired at four months	
3	M	64	Polya gastrectomy	2 days	Sitting	3 days	Both	Bouginage	
4	M	64	Polya gastrectomy	2 days	Sitting	1 month	Both■	Failed bouginage. Jejunal transplantation	
5	F	64	Radical Polya gastrectomy			1 year	Both	Failed bouginage. Jejunal transplantation	
Windsor (1964)									
			Billroth I gastrectomy (32 cases)				Reflux 50%		
			Polya gastrectomy (29 cases)				Reflux 29%		

●Had reflux œsophagitis before operation
■Pre-operative X-rays showed no reflux or hiatal hernia
▲Pre-operative X-rays showed reflux and hiatal hernia

reason dilatation as a definitive measure cannot be recommended, since it provokes further reflux, followed later by increasing obstruction and a lengthening of the stricture.

Unfortunately pre-operative information on cardiac incompetence is almost entirely lacking in our old gastrectomy patients' notes but reflux can be expected in about 29% of patients with duodenal ulcers (Clarke *et al.* 1965). Following gastrectomy, Windsor (1964) found cardiac incompetence in 50% for the Billroth type, while Clarke *et al.* (1965), in a series of 32 cases, found a pre-operative reflux incidence of 29% converted to 90% by transabdominal vagotomy. The reason for this increase is damage to the 'cardiac sphincteric mechanism', probably the extrinsic part concerned with the phreno-œsophageal fascial closure of the hiatus; such damage, inevitable in vagotomy, is highly probable in gastric resections when the left gastric vessels, around which the fascia passes, are taken high on the posterior abdominal wall.

Pursuing the point of differing techniques, little information can be gained from a study of the types of gastrectomy and the possible variations in their reflux incidence. McKeown (1958) blamed the Polya type for his 2 post-gastrectomy strictures and treated them successfully by conversion to Billroth, whereas Windsor (1964) did the reverse, finding that 16 out of 32 patients with Billroth had reflux, as against only 8 out of 29 after a Polya gastrectomy: Windsor added that forcible pulling downwards of the lesser curve to meet the duodenum straightens the œsophago-

Table 2

Peptic œsophagitis and stricture after vagotomy for duodenal ulcer

Case	*Sex*	*Age*	*Operation for duodenal ulcer*	*Intubation*	*Bed position*	*Onset of dysphagia*	*Reflux and hiatal hernia after operation*	*Treatment*	*Result*
Brain (1966)									
1	M	62	Billroth I gastrectomy. 1959: vagotomy	Nil	Sitting	3 years ●	Both ■	Repair of hiatal hernia	Hæmatemesis and stomal ulceration Cured
2	F	55	Vagotomy	Nil		3 weeks	Both ■	Repair of hiatal hernia	Cured
3	M	58	Vagotomy	Nil		9 months ●		Repair of hiatal hernia	Cured

● Regurgitation and heartburn at once
■ Pre-operative X-rays showed no reflux or hiatal hernia

gastric angle and causes the incompetence, while conversion to a Polya type allows relaxation, restoration of the angle and a return of competence. The situation becomes even more confused by the recommendations of Boerema (1955) and Ziperman *et al.* (1963), who both advised pulling downwards of the whole stomach and its suture to the anterior abdominal wall as a cure for hiatal hernia and incompetence.

From our own small series it would appear that peptic strictures are most likely to follow the very radical gastrectomies, implying excessive damage to the cardia and possibly alkaline/trypsin regurgitation (considered later).

Is gastric drainage ineffectual? Three facts support this: (1) Post-gastrectomy biliary vomiting is to be found in 15 – 20% of patients. (2) Toye & Williams (1965) show that this vomiting or regurgitation is accompanied by true 'stasis' in the gastric remnant. (3) My own small experience of pyloroplasty as an adjunct to bouginage for treating the major inoperable strictures shows that no beneficial effects were achieved in 13 patients.

For these reasons I believe that writers like Burge *et al.* over-emphasize the importance of pyloric obstruction and gastric drainage. Any cause of frank vomiting, which incidentally is not a symptom of reflux œsophagitis, may exaggerate or even cause a hiatal hernia as a secondary phenomenon; yet the relief of such an obstruction can do no more than restore a normal emptying rate. Final control will always be determined by the competence of the cardia. Similarly it can be argued that a simple reduction of stomach capacity by gastrectomy increases its liability to regurgitation.

Are there other factors? Intubation is a known promoter of reflux, particularly in the ill patient in the recumbent posture: an elegant experiment by Nagler & Spero (1963) proved this. In the second patient in my series (Table 1), known to have reflux œsophagitis before his operation and in whom intubation was avoided, no obvious beneficial effects could be seen in his post-operative course. Nevertheless, the postoperative intubation of gastric remnants via the anterior abdominal wall should be recommended.

The alkaline/trypsin enzymes, given access through reflux to the œsophagus, are potent causes of œsophagitis: Cross & Wangensteen (1951) proved this experimentally, while the devastating effect of duodenal juices on the squamous epithelium of the abdominal wall has been seen by us all. The fifth patient in our first series (Table 1) illustrates this factor in its purest form: she had a lower œsophageal stricture, following a total gastrectomy and simple loop anastomosis, cured by excision and a Roux-en-Y conversion, thus keeping the duodenal juices well away from the œsophagus.

REFERENCES

Bingham J A W (1958) *Brit. med. J.* ii, 817
Boerema I (1955) *Arch. Chir. Neerl.* **7**, 351
Burge H W, Gill M G, MacLean C D T & Lewis R H (1966) *Thorax* **21**, 66
Casten D F, Bernhang A, Nach R J & Spinzia J (1963) *Surg. Gynec. Obstet.* **117**, 87
Clarke S D, Penry J B & Ward P (1965) *Lancet* ii, 824
Cross F S & Wangensteen O H (1951) *Proc. Soc. exp. Biol., N.Y.* **77**, 862
Hurst D W (1961) *Brit. med. J.* ii, 1460
McKeown K C (1958) *Brit. med. J.* ii, 819
Nagler R & Spero H M (1963) *New Engl. J. Med.* **269**, 495
Toye D K M & Williams J A (1965) *Lancet* ii, 524
Windsor C W O (1964) *Brit. med. J.* ii, 1233
Ziperman H H, Mathewson C, Stanek R G & Brugger A M (1963) *Surg. Gynec. Obstet.* **116**, 608