GASTRO-OESOPHAGEAL REFLUX AND HIATUS HERNIA: GENESIS, RELATIONSHIP AND TREATMENT MECHANISMS

J.M. BEELEY

M.R.C.P.

Surg. Commander Royal Navy

The relationship between hiatus hernia and gastro-oesophageal reflux

The longstanding concept that a sliding hiatus hernia itself leads to gastro-oesophageal reflux was disproved ten years ago by Wankling and his co-workers. (Wankling, Warrian & Wankling, 1966). They were able to demonstrate that in some patients with hiatus hernia there were normal gastro-oesophageal sphincter pressures, few symptoms and no evidence of reflux on endoscopy. Patients with reflux were shown to be those with a low basal sphincter pressure irrespective of whether or not a hiatus hernia was present. When low basal sphincter pressure was found, there was an inadequate rise in sphincter tone when intragastric pressure rose; with normal basal sphincter pressure, rising intragastric pressure produced forcible sphincter contraction even in patients with hiatus hernia in whom the sphincter is situated above the diaphragm.

This work has been confirmed by Cohen and Harris (1971) and it must now be accepted that the position of the sphincter below the diaphragm is not important in determining its competence. Surgical procedures for hiatus hernia which sometimes improve oesophageal reflux are not, therefore, successful because of the resiting of the lower oesophagus in the abdomen or restoration of its angle of entry into the stomach; the rationale for surgical repair of hiatus hernia is therefore under question.

The assumption that hiatus hernia necessarily leads to oesophageal reflux has to be abandoned, though in clinical practice a hiatus hernia is frequently demonstrable in patients with oesophageal reflux. Beeley and Warner (1972) studied twenty eight patients with hiatus hernia who suffered from effortless regurgitation of stomach contents into the mouth. In unpublished data, they analysed all those symptoms present which have in the past been ascribed either to oesophageal regurgitation or hiatus hernia (table 1). Regurgitation and hearthburn are clearly manifestations of an incompetent oesophageal sphincter and excessive salivation has been shown to result from the presence of acid in the lower oesophagus (Aylwin 1953). Three other symptoms commonly present were dysphagia, "a lump in the throat" and choking bouts and it may be that these reflect on incoordination of the oesophagus above the gastro-oesophageal sphincter. There is already some evidence that a malfuncting gastro-oesophageal sphincter may not be an isolated finding and that it is associated with a high resting pressure in the cricopharyngeal sphincter (Hunt, Connell & Smiley, 1970) and sometimes with pyloric incompetence (Gillison, Capper & Airth, 1969). These three symptoms are also features of pharyngeal pouch and patients with that disorder very commonly prove to have an associated hiatus hernia (Smiley, Caves and Porter, 1970). It is tempting, therefore, to postulate that gastro-oesophageal reflux is a feature of a more generalized incoordination of the upper gastrointestinal tract which includes the presence of a low basal pressure in the gastro-oesophageal sphincter.

Increased intra-abdominal pressure provides a challenge both to the oesophageal sphincter in its ability to prevent reflux of stomach contents and also of the

TABLE 1

"INCIDENCE OF SYMPTOMS IN TWENTY-EIGHT PATIENTS WITH GASTRO-OESOPHAGEAL REFLUX AND ASSOCIATED HAITUS HERNIA"

Symptom	*No. Patients Accepted*	*Definition*
REGURGITATION	28	Effortless appearance of gastric contents in the mouth.
HEARTBURN	27	Burning sensation retrosternally below the manubrium in the midline.
BELCHING	23	Increased frequency of belching compared with pre-symptomatic period.
EPIGASTRIC FULLNESS	21	———
EXCESSIVE SALIVATION	18	Increased salivation compared with pre-symptomatic period.
DYSPHAGIA	17	Sensation of food sticking retrosternally on swallowing.
NAUSEA	17	———
VOMITING	16	Vomiting in association with the presenting symptom complex.
USE OF EXTRA PILLOWS	16	Use of more than two pillows to ameliorate reflux symptoms.
LUMP IN THROAT	15	Something sticking above suprasternal notch whilst eating.
CHOKING	11	Choking or coughing bouts whilst eating, stooping or sleeping.

ability of the diaphragm to prevent the upper stomach herniating into the thorax. It is therefore possible that hiatus hernia and oesophageal reflux have a common aetiology rather than a causal relationship. It is well known that oesophageal reflux may remit if constricting corsets are removed or if obesity is treated and hiatus hernia may be unmasked radiologically even in some asymptomatic subjects by various procedures which increase intra-abdominal pressure. Thus a rise in abdominal pressure may precipitate both oesophageal reflux and hiatus hernia. Until more is known about the genesis of these two abnormalities, it is best to attach little importance to the finding of a hiatus hernia during radiological procedures which may unmask one even in asymptomatic subjects, (Dyer, and Pridie, 1968). Similarly, whilst easy reflux of heavy barium compounds may suggest a weak gastro-oesophageal sphincter, it is not proof that normal gastric contents also reflux. Measurements of oesophageal pH in subjects with reflux symptoms are at present the best method of

confirming acid reflux (Pattrick, 1970) whilst barium examinations give supportive evidence and exclude other upper gastro-intestinal pathology.

Treatment mechanisms gastro-oesophageal reflux

When stomach contents appear effortlessly in the mouth, the gastro-oesophageal sphincter is weak. Whether retrosternal burning or oesophagitis occur probably depends on the corrosiveness of gastric contents and also upon the duration of their presence in the oesophagus. The logical approach to management of reflux symptoms would be one which achieves the following:—

1. Strengthening of the gastro-oesophageal sphincter.
2. Reduction of intra-abdominal/intragastric pressure.
3. Rapid drainage of refluxed gastric contents back into the stomach.
4. Reduction of corrosiveness of gastric contents.
5. Re-inforcement of the oesophageal mucosal barrier.

1. The Gastro-Oesophageal Sphincter

The pharmacological mainstay of treatment for oesophageal reflux has long been the use of antacids in various physical and chemical formulations. Whilst no controlled blind trials have been reported confirming or refuting the traditional efficacy of simple antacids compared with placebo in controlling reflux, more is now known about their possible mode of action. Present evidence suggests that, by raising gastric antrum pH, a release of gastrin results and itself raises sphincter pressure (Giles *et al* 1969, Castell & Harris, 1970, Castell and Levine, 1971). It is now thought likely that this gastrin-mediated effect on the sphincter is more important than the short-lived neutralization of gastric contents.

The fact that atropine reduces sphincter pressure (Lind, Crispin & McIver, 1968, Rosenberg & Harris, 1971) and blocks the effect of gastrin on the sphincter (Cohen & Guelrud 1971) suggests that there is no logical place for the use of anticholinergic drugs for oesophageal reflux. Intravenous metoclopramide raises sphincter pressure in normal subjects (Heitman & Muller, 1970) but does not reduce reflux in patients with or without hiatus hernia and is therefore unlikely to convey benefit (Glanville & Walls, 1972).

The instillation of acid into the stomach causes a fall in sphincter pressure which may be a result of inhibited gastrin release (Castell & Harris, 1969, Castell & Harris, 1970); this observation possibly explains the heartburn sometimes produced by acid fruit juices which should be avoided in patients with reflux symptoms. Smoking should also be curtailed as it lowers cardiac sphincter pressure (Lind, Crispin & McIver, 1968, Rosenberg & Harris, 1971) and, by causing pyloric incompetance (Read & Grech, 1973), may encourage movement of corrosive bile salts from duodenum to oesophagus.

Intravenous secretin decreases sphincter pressure by inhibiting the constrictor effect of gastrin (Cohen & Lpishutz, 1971) but another enterogastrone may be more potent in this respect as corn oil entering the duodenum or duodenal acidification cause a greater relaxation of the sphincter (Castell & Harris 1970, Lipshutz, Hughes & Cohen, 1972). When fatty meals precipitate symptomatic reflux, another enterogastrone is therefore probably responsible.

2. Intra-Abdominal Intragastric Pressure

In contrast to normal subjects, those with oesophageal reflux show an increment of sphincter pressure smaller than a precipitating increment of gastric pressure (Cohen & Harris 1968, Cohen & Harris 1971). In such patients the sphincter zone becomes incompetent during abdominal compression (Wankling, Warrian & Lind, 1965, Lind, Warrian and Wankling 1966).

The implications of these observations are that abdominal corsets and tight garments should be discarded and obesity corrected. Little is known about the effects of body position on intragastric or intra-abdominal pressure but certain straining movements are observed to precipitate ra-

diological reflux and therefore manouvres resulting in heartburn or regurgitation should be avoided.

Belching is a common feature of oesophageal reflux (see table 1) and the expulsion of air from the stomach probably beneficially reduces intragastric pressure. Polymethylsiloxane may, by lowering surface tension of stomach contents facilitate the formation of a single gas bubble in the stomach which would be more likely to be expelled than gastric contents.

When acid enters the oesophagus it reflexly produces an increased volume of gastric acid (Ward 1970) and this may result in a rise of intragastric pressure. The passage of acid drinks down the oesophagus may similarly increase gastric acid secretion and should therefore be avoided. Large meals traditionally promote reflux symptoms and, because they probably raise intragastric pressure, should be exchanged for small frequent ones.

Atropine – like compounds are probably undesirable as they abolish sphincter response to abdominal compression (Bertarello, Tuttle & Grossman 1960).

3. Oesophageal Drainage

Oesophagitis probably only results when refluxed gastric contents remain in the oesophagus for some time. In normal subjects an efficiently coordinated oesophagus and gastro-oesophageal sphincter allow occasionally refluxed contents to return to the stomach rapidly.

Raising the head of the bed and sleeping with the thorax elevated shorten reflux episodes (Habibulla, Ammann & Leigh Collis, 1971) by facilitating drainage of any gastric contents entering the oesophagus and are recommended in management of reflux symptoms.

If any benefit is derived from anticholinergic drugs it is likely to be explained, incongruously, by reduction of sphincter tone which may avoid entrapment of gastric contents in the oesophagus provided an upright posture is maintained. Oesophageal clearance would not, however, be enhanced by the reduction of peristalsis which follows atropine administration (Kantrowitz, Siegal and Hendrix, 1966).

4. Corrosiveness of Gastric Contents

Both acid and pepsin are likely to be involved in the production of oesophagitis. Bile may be present in refluxed gastric contents if the pylorus is incompetent, as after smoking, and is known to break the integrity of mucosal resistance to hydrogen ions. Whatever progress is made in the management of gastric ulcer, in whose aetiology bile is involved, is likely to be relevant also to the management of oesophagitis. In the meantime alkalis are advocated as they produce a transient reduction of damaging hydrogen ions.

A low density compound capable of floating on gastric contents would probably be preferentially refluxed into the oesophagus when the patient is erect and the sphincter weak. A recently marketed compound of alginate and small quantities of antacid ("Gaviscon"), which has been proved by properly controlled trials to control reflux (Beeley & Warner, 1972; Stanciu & Bennet, 1974), may act in some instances as a refluxant. Having entered the oesophagus through a weak sphincter, its non-corrosive character would be preferable to gastric contents and would not produce the reflex gastric secretion which attends the presence of acid in the oesophagus. The trials suggest, but do not prove, that it may also form a physical barrier to reflux at the cardia.

5. Reinforcement of the Mucosal Barrier

The mucus coating the oesophagus is probably important in its protection. The ability of the eel to survive in either salt or fresh water depends upon its exterior coat of mucus and when deprived of this, fresh water immersion causes death. Little is at present known about ways of stimulating its secretion in the human gastrointestinal tract.

Bile is known to disrupt the gastric mucosal barrier allowing hydrogen ions to enter the epithelium in exchange for sodium ions (Ivey *et al* 1970 & 1971, Black *et al* 1971) and similar effects might be anticipated in the oesophagus when bile is present in refluxed gastric contents. This property of bile may be related to its deter-

gent effect on the mucus coating of the upper gastrointestinal tract.

Commercial preparation of antacids are often formulated to improve their coating properties in the hope that they may temporarily enhance mucosal protection. The physical properties of antacid compounds are likely to be important in determining their efficiency in controlling oesophagitis. Alginate compounds may, by virtue of their gelatinous property, adhere to the oesophageal mucosa and afford some protection.

Polymethylsiloxane has been added to some antacid preparations in the hope that by "siliconizing" the oesophageal mucosa, it may form a barrier to hydrogen ions; this concept seems unduly optimistic as it is unlikely, preferentially, to repel hydrogen ions when in the small intestine, it allows normal absorption of food substances.

The inclusion of local anaesthetic agents in compounds used for reflux symptoms is to be deplored as they abolish the warning symptoms of oesophagitis which may, if progressive, result in ulcer or stricture.

Summary

The cause of both hiatus hernia and gastro-oesophageal reflux is not yet known but both are liable to spontaneous remission. The rationale for various treatment regimes is discussed. Mechanical, chemical, humoral, neurogenic and mucosal factors are involved in the protection against oesophagitis. In the present state of knowledge, there are good theoretical grounds for employing both physical measures and antacids and clinical trials have established the efficacy of alginate/antacid compounds in controlling oesophageal reflux. Where vigorous conservative measures have failed to control oesophagitis, various surgical procedures are sometimes successful but their mechanism is probably not related to the effect of obliteration of a hiatus hernia on sphincter competence.

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