Hiatus hernia reflux syndrome

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Summary

Some of the features of the syndrome, the mechanism of symptom production, and the role of radiology in diagnosis are defined. Current theories of the anti-reflux mechanism and recent concepts of the pathological changes involved are described.

Definitions

Hiatal herniation is the result of the capacity to herniate. In most people found to have a hiatal hernia by radiology or at operation, most of the time the stomach is not herniated into the chest. Herniation is intermittent and only occurs when the subject gets into certain positions or increases intra-abdominal pressure. This form will be missed at fluoroscopy unless the appropriate challenge is applied. Sometimes part of the stomach remains in the chest when the subject stands up, and more herniates when he lies down or bends over. This is continuous herniation which is infrequently missed at fluoroscopy.

Reflux is the flow of gastric contents into the oesophagus and the capacity to reflux depends on the competency of the anti-reflux mechanism which may be intermittently or continuously incompetent.

Intermittent incompetence means that, most of the time, the antireflux mechanism prevents reflux when challenged, but intermittently and momentarily the mechanism fails and reflux may then occur if circumstances are right. We know that reflux occurs because we can taste gastric contents or we can detect it with a pH electrode in the oesophagus, but unless we happen to X-ray the subject at the precise moment that the antireflux mechanism becomes incompetent, we shall not see barium enter the oesophagus. Intermittent incompetence with reflux occurs when the subject is standing, sitting, or lying, and is not provoked by lying or rolling over or bending. When symptoms occur as a result of the reflux, they are particularly troublesome after fatty foods and for this reason I have called intermittent incompetence the 'Hot Fat Heartburn syndrome'.

If the antireflux mechanism is continuously incompetent it will fail at any time if challenged, so

that barium is seen to reflux if the patient is examined by an appropriate technique. Because continuous incompetence allows reflux to occur when we lie down or bend over I have called it the 'Recliner's Reflux syndrome'.

Intermittent incompetence is a problem because we cannot demonstrate it with barium and we must depend on the recognition and reproduction of the symptom pattern to make a diagnosis. Reflux from intermittent incompetence can produce unpleasant symptoms and recognizable histological change in the oesophageal epithelium.

The Antireflux mechanism

(a) *The abdominal segment hypothesis.* When the stomach is in its normal position the lower half of the cardiac sphincter is in the abdomen (Fig. 1a). This sphincter normally squeezes with a force of 5-15 cm water. An increase of abdominal pressure squeezes the stomach and also squeezes the intra-abdominal part of the sphincter so the sphincter is not challenged. If the whole stomach wall contracts when the antro-pyloric segment is closed so that intra-gastric pressure increases and exceeds the squeeze of the sphincter, or if the sphincter squeeze has fallen to or below the squeeze of the stomach, the sphincter may then be opened and stomach contents can flow into the oesophagus. This might be the mechanism of intermittent incompetence. The challenge to the sphincter comes from stomach squeeze and has nothing to do with posture or intra-abdominal pressure. Anything which influences sphincter squeeze, such as the concentration of serum gastrin or secretin, might act in this way and changes in the concentration of these two polypeptides may be the mechanism of the 'Hot Fat Heartburn syndrome'. Anti-cholinergic drugs reduce sphincter squeeze and may thereby increase the likelihood of reflux, whatever they may do to the amount of acid secreted. Cholinergic drugs like bethanecol or neostigmine logically might be more useful. Metoclopramide, although it slightly increases sphincter tone, has not so far been demonstrated consistently to prevent the symptoms of reflux in a complaining group of patients, but it does also increase the magnitude of gastric contractions,

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FIG. 1. Hiatus hernia reflux syndrome.

and might thereby increase the possibility of reflux. Further studies are necessary.

When herniation occurs the sphincter is wholly within the chest and the hiatus is occupied by a tube of stomach. If the hiatus is small its walls will squeeze and empty the tube of stomach, especially when the diaphragm contracts to increase intra-abdominal pressure. Abdominal pressure squeezes the stomach but also squeezes the emptied tube of stomach which acts rather like the flutter valve of the old-fashioned BMR machine. Scarcely any stomach contents flow from abdomen to thorax and intra-abdominal pressure is not transmitted to the intra-thoracic part of the stomach. I have called this the competent hiatus (Edwards, 1961) and it is important in radiology because the herniation may be missed if barium does not flow from abdominal to thoracic stomach when the patient is tipped head-down (Fig. 1b). This is a common radiological finding in patients with the capacity to herniate a loculus up to 3.5 cm diameter. When the hiatus is competent the sphincter squeeze only has to withstand the pressure within the intra-thoracic stomach and gross reflux is not a problem. In these circumstances barium frequently does not reflux from the intra-thoracic stomach.

If the hiatus is large the neck of stomach passing through is not squeezed and there is free flow of gastric contents from abdominal to thoracic stomach and transmission of intra-abdominal pressure to challenge the sphincter. I have called this the incompetent hiatus (Edwards, 1961) (Fig. 1c). The battle between sphincter squeeze and stomach squeeze will cause intermittent incompetence with symptoms predominantly related to time after food and type of food, as in the Hot Fat Heartburn syndrome, but in addition, the sphincter squeeze has to battle with intra-abdominal pressure which is transmitted directly to it. If abdominal pressure, caused by lifting or bending or the weight of abdominal contents when lying down, is greater than sphincter squeeze the sphincter will be opened and reflux could occur.

Predictions about treatment can be made from this hypothesis. The management of intermittent incompetence should concentrate on increasing sphincter squeeze and decreasing stomach squeeze, together with a decrease in the noxiousness of what refluxes. Postural treatment is of limited value because the symptoms are not consistently provoked by bending or lying. Medical measures are usually quickly successful and surgery is inappropriate unless there is inflammatory oesophagitis. On the other hand the management of continuous incompetence which is a mechanical problem, must be by mechanical method; either by preventing or avoiding challenge to the sphincter, or by repairing the hernia in such a way that the sphincter segment will be retained within the abdomen. Medical measures cannot cure the mechanical defect.

(b) *The sphincter alone hypothesis.* Another hypothesis postulates that reflux is prevented solely by the squeeze of the sphincter whether it is in the abdomen or in the chest (Lind, Warrian and Wankling, 1966; Cohen and Harris, 1971). The sphincter is claimed to respond to a challenge to open it by contracting and the contraction response is proportional to its basal tone. If the basal tone is high the contraction response will be higher than the challenge, but if the basal tone is low the response will be less than the challenge and reflux will occur. One prediction of this hypothesis is that to determine whether reflux is occurring it is only necessary to measure basal sphincter tone. This prediction has not been fulfilled. Another prediction is that herniation has nothing to do with reflux and therefore there is no point in repairing a hernia because the sphincter will not work any better in the abdomen than it did in the thorax. In my own experience of 230 peptic strictures every one of them had radiological evidence of reflux and of the capacity to herniate; and of the several thousands of patients with symptoms of reflux that I have screened, less than 1% have had visible reflux of barium without visible evidence of herniation at the same time. I suspect that con-

tinuous incompetence relates to the capacity to herniate. Moreover surgeons do sometimes succeed in stopping continuous incompetence by repairing a herniation and creating an intra-abdominal segment of oesophagus.

Neither hypothesis is wholly satisfactory.

Pathological changes. 'Oesophagitis' is a word used by pathologists to indicate erosion of the epithelium, hyperaemia, oedema, infiltration with neutrophils, lymphocytes and plasma cells and fibrosis in the submucosal layers. Although the word is commonly used by physicians to indicate that the patient has symptoms of gastro-oesophageal reflux, the correlation between symptoms, endoscopic appearance and histological changes is poor. This lack of correlation seems likely to result from an illogical choice of criteria because inflammation represents a severe degree of damage to the epithelium, whereas symptoms and changes in the endoscopic appearance can occur without inflammation.

Recently Ismail-Beigi, Horton and Pope (1970) have described early changes in the squamous epithelium in association with symptoms of reflux. Normal oesophageal squamous epithelium has a thin basal zone of several layers of rounded basophilic cells with dark nuclei which occupy not more than 10% of the total thickness. Beyond this layer the cells are progressively flattened to form the stratified squamous layer. The subepithelial tissue has papillae which extend not more than two-thirds of the way to the surface of the epithelial layer and contain capillaries and nerve endings. The earliest changes described by Ismail-Beigi *et al.* (1970) are: (1) hyperplasia of the basal cell layer which is increased to 15% or more of the total thickness with a correspondingly thinner squamous layer; (2) the papillae are elongated and extend beyond the normal two-thirds of the distance to the surface. At this stage there is no increase in the round cell or neutrophil content of the epithelium or subepithelial layers and none of the classical signs of the inflammatory process. These earlier changes do not justify the term 'oesophagitis'; there is an intact epithelium, and healing is to be expected.

The next stage in the damaging process seems to be a further thinning of the epithelial layer and extension of the papillae so that eventually the epithelium is eroded to expose the capillaries and the subepithelial tissue. Bleeding, ulceration, and provocation of the inflammatory response may then develop.

It seems likely that the texture and colour of the mucosa seen down the endoscope will be related to the character of the squamous epithelium and the thickness overlying the capillaries of the papillae, as well as the degree of engorgement of these capillaries. It also seems likely that the character and thickness

of this epithelium will determine how readily hydrogen ions, heat and alcohol stimulate the nerve endings.

Diagnosis of the more severe changes is important because healing is not predictable and a stricture may develop if submucosal inflammatory changes proceed to fibrosis. Early fibrotic changes may possibly stop and even soften if noxious reflux is stopped by an effective operation, but once the fibrotic changes are mature the stricture remains until it is excised. Bougienage never relieves the dysphagia.

The cause of symptoms

(a) Herniation. The movement of stomach in and out of the chest is not appreciated by the patient during fluoroscopy, nor is the flow of barium from stomach to oesophagus. If most of the stomach is in the chest, torsion may occur and be associated with bouts of lower chest or upper abdominal pain which characteristically and dramatically switch on and off.

(b) Reflux. When gastric contents reach the mouth without being propelled by contraction of the abdominal walls in vomiting, the antireflux mechanism must have failed and we call the process regurgitation or rumination according to the circumstances. Although the taste may be unpleasant, heartburn may not occur. A common paraesthesia in this syndrome is a sensation of a lump or ball in the throat or that food is 'sitting like a lump of lead' in the neck or chest. The characteristic feature of this pseudodysphagia is that it disappears during eating and drinking or after taking antacid, and tends to appear 10–110 min after a drink or a meal. It probably arises from stimulation of the lower oesophageal mucosa.

Heartburn seems to result from the combination of two events. Firstly the mucosa must be hypersensitive, possibly because the epithelial layer is thinner or more pervious or the stimulus is applied for an unusually long time. Secondly the stimulus has to be sufficiently vicious. Hypersensitivity seems greatest to hydrogen ions and the pH of the refluxing material must be low enough. Possibly some other component of gastric contents potentiates the action or increases the permeability of the mucosa, but highly acid citrus fruit juices often sting as they go down the oesophagus. Hypersensitivity to heat and alcohol rarely occurs without hypersensitivity to acid, and probably represents a greater degree of damage to the mucosa. A hot or alcoholic drink burns or stings as it goes down.

Since reflux does not produce symptoms unless the mucosa is abnormal and the refluxing material sufficiently noxious it is possible to have reflux from either intermittent or continuous incompetence and

not have any symptoms. It is also possible to have symptoms and no radiologically visible reflux; and to have an abnormal mucosa and not have any symptoms because what comes up or goes down at that particular time does not stimulate the nerve endings. Heartburn and hypersensitivity to acid occur in people with only minor histological changes and these symptoms should not be interpreted as indicating inflammatory 'oesophagitis'.

Hypersensitivity to stretch is present when a solid bolus hurts as it goes down but its passage is not delayed and there is no radiological or endoscopic evidence of stricture formation. This symptom is not common and has to be sought for. It seems to precede stricture formation and probably indicates a moderate degree of inflammatory change in the sub-mucosa without much increase in its resistance to stretch. It should represent a reversible phase of inflammation. Discomfort or pain with delay in or obstruction to the passage of a solid bolus indicates a more severe degree of inflammation with an increased resistance to stretch which is likely to result from the presence of fibrous tissue rather than muscle contraction, because the muscle of the sphincter segment contracts and relaxes apparently normally in response to a swallow, but the inflamed wall does not stretch as much as usual. Liquids can be drunk at a normal speed, but a solid bolus impacts according to its size and consistency. Stricture segments may be hypersensitive to acid, heat and alcohol but sometimes the mucosa of the segment heals and re-epithelialization occurs. This may be the reason that although stricturing persists, the symptoms of heartburn may disappear in patients in whom free reflux of barium can be demonstrated radiographically.

Ring strictures have a different symptom pattern. Although they obstruct the passage of solids, the mucosa is commonly not hypersensitive to heat or alcohol and sometimes not hypersensitive to acid. Such information as there is on the histological changes in the epithelium of the ring stricture suggests that there is usually no inflammatory response.

The role of radiology in the diagnosis of these syndromes

The radiologist cannot confirm or deny symptoms of reflux since the symptoms may arise from intermittent incompetence which by definition is not demonstrable by radiology. The demonstration of the capacity to herniate stomach and of continuous incompetence does not prove that the symptoms arise from reflux, since both herniation and reflux may occur without symptoms being present. The main roles of radiology are firstly to detect the presence of oesophageal, gastric or duodenal ulcer or neoplasm, or radio-opaque gall stones or other pathology which may be contributing to the patient's ill health; and secondly to demonstrate the size of the loculus which herniates in response to a standard challenge, the size of the hiatus through which it herniates, and the severity of the mechanical defect which allows reflux to occur. Free flow of gastric contents up the oesophagus cannot be stopped by medical means but an appropriate operation may produce some relief and the radiological information is helpful to the surgeon.

What the radiologist sees depends on what he does and the technique of examination is important (Edwards, 1969). The capacity to herniate may not be demonstrated unless the subject drinks barium while tipped 10° head-down, or rolls onto his right side, or bends over to touch his toes. In my own experience it is extremely uncommon to see radiographic evidence of reflux without the capacity to herniate stomach.

Table 1 shows the frequency of the postulated combinations of hiatal hernia, radiographic reflux, symptoms, and histological evidence of inflammatory oesophagitis.

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TABLE 1. Frequency of postulated combinations of hiatal hernia radiographic reflux, symptoms and oesophagitis

	Frequency															
	U	C	U	U	U	C	R	C	R	R	?	?	R	C	?	C
Capacity to herniate	+	+	+	+	+	+	+	+	—	—	—	—	—	—	—	—
Radiographic reflux	+	+	+	+	—	—	—	—	+	+	+	+	—	—	—	—
Symptoms	+	+	—	—	+	+	—	—	+	+	—	—	+	+	—	—
Histologic oesophagitis	+	—	+	—	+	—	+	—	+	—	+	—	+	—	+	—

C, common; U, uncommon; R, rare; ?, doubtful that it exists.

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