FLATULENT DYSPEPSIA*

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'Flatulent dyspepsia' is one of the commonest ailments of man, ranking with 'rheumatism' and 'headache' in its ubiquity; and, like them, it is not a disease but a manifestation of an underlying condition which may be trivial, serious or fatal. It does, however, form a well-knit symptom-complex which may be defined as upper abdominal discomfort related, though sometimes vaguely, to meals and associated with heartburn, eructations and acid regurgitation. In severe cases angina-like chest pains, high backache and dysphagia may also be experienced. The causes of this syndrome are many and may be listed as:

1. Normal or Physiological Conditions
   (*a*) Pregnancy
   (*b*) Obesity
   (*c*) Gastric distension—gluttony, aerophagy, etc.
2. Disease States
   (*a*) Hiatus hernia
   (*b*) Peptic ulceration of stomach and duodenum
   (*c*) Cholecystitis and cholelithiasis
   (*d*) Appendicitis
   (*e*) Abdominal herniae
   (*f*) Abdominal tumours—fibroids, pancreatic cysts, etc.

Though flatulent dyspepsia may be common to all these varying conditions, it is often possible to distinguish the cause clinically because of the presence of additional symptoms more or less specific to the underlying condition.

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Flatulent dyspepsia is a non-specific syndrome as far as disease states are concerned, yet it is highly specific to its basic underlying cause. Acid eructations, heartburn and flatulence, when present together, denote only one thing—gastro-oesophageal regurgitation. It is interesting to determine why affections of so many different organs should cause gastro-oesophageal regurgitation and for this it is necessary, in the first place, to understand the mechanism of reflux of stomach contents into the oesophagus.

The Mechanism of Prevention of Regurgitation

Because the abdominal pressure is positive and the thoracic pressure negative, the pleuroperitoneal pressure gradient constantly tends to displace stomach contents into the oesophagus. In spite of this, continuous regurgitation is in some way prevented. It is commonly agreed that this is mainly accomplished by the oblique entry of the oesophagus into the stomach, which acts as a flap-valve when the stomach distends. Several experiments on young male cadavers were designed to confirm this belief (Marchand, 1955):

The pylorus was cannulated and the stomach distended with water from a reservoir which was raised alongside a measured rule until the water was seen to escape from the oesophagus.

The experimental findings illustrated in Fig. 1 clearly confirm the importance of the mechanical arrangement of the fundus. When the fundus is excluded from the rest of the stomach (Fig. 1B), the resistance offered by the cardia to regurgitation is diminished threefold.

When the left leaf of the diaphragm is removed to allow the

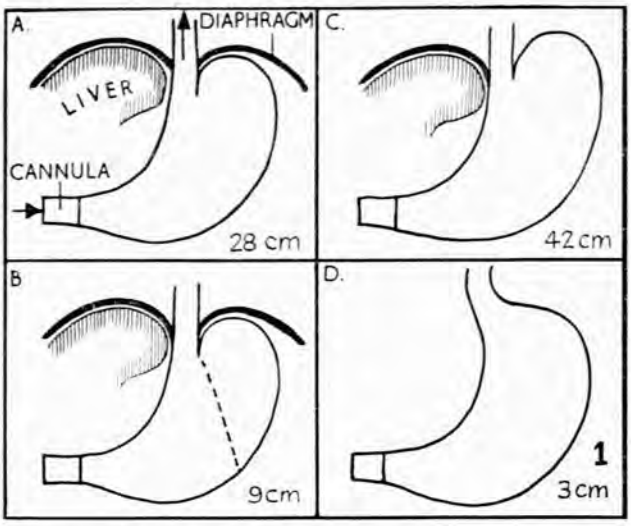

Fig. 1. Illustration of experiment designed to show the importance of the normal acute angle of entry of the oesophagus into the stomach in preventing reflux of stomach contents (see text). The pressures, in cm. of water, denote the force at which reflux occurs under the experimental condition illustrated. The dotted line in B illustrates position of the clamp used to obliterate the fundus of the stomach. In D the liver and diaphragm were completely removed.

fundus to bulge upwards without hindrance, the pressure necessary to induce regurgitation is increased by half again (Fig. 1C).

The liver tunnel, the crura, and the pressure of surrounding organs, are important in maintaining the relative positions of the stomach and oesophagus and the efficiency of the cardio-oesophageal angle (Fig. 1D).

During barium examination of a patient with hiatal hernia, a condition in which the gastro-oesophageal angulation is abolished, it is often possible to demonstrate a weak barrier to regurgitation from the intrathoracic stomach. Johnstone (1946) believes that this is evidence for an intrinsic sphincter at the cardio-oesophageal junction. This view is supported by the following experiments.

Twenty normal young adults were subjected to controlled abdominal compression in the head-down position by means of a balloon incorporated in a corset. (Marchand, 1952). The stomach was filled by drinking watery barium and the balloon was then gradually inflated until regurgitation was seen or until a pressure of 150 cm. of water was reached. The following general conclusions were made:

1. The higher the pressure at which reflux was produced, the more forcible was regurgitation. For instance, pressures of 60 cm. of water seldom caused barium to regurgitate into the cervical oesophagus, whilst pressures of 150 cm. of water usually did so and frequently forced the barium into the mouth.
2. Regurgitation is facilitated by the act of swallowing. If a constant abdominal pressure is maintained whilst the stomach is distended with barium then, at the very instant of swallowing, the barium already in the stomach regurgitates powerfully.
3. Regurgitation is sometimes seen when an oesophageal peristaltic wave reaches the cardia. This observation was reported by Johnstone (1951) and is to be distinguished from the reflux which occurs at the inception of a swallowing effort.

These observations illustrate two fundamental points:

1. The force which causes regurgitation is the intragastric pressure.

2. The acute oesophago-gastric angle is reinforced by some active intrinsic mechanism which can be inhibited by swallowing; in other words an intrinsic sphincter is present.

The acute angle of oesophageal entry into the stomach and the weak inherent sphincter are the immediate factors which prevent free gastro-oesophageal regurgitation. The normal angle of entry, however, is maintained intact by surrounding structures such as the liver and more particularly the diaphragm. The prime need is that the cardia remains at diaphragm level and this depends largely upon the preservation of the optimal size and shape of the oesophageal hiatus. The normal hiatus is elliptical in shape. This shape is maintained by the direction of the crural muscle fibres, the anterior decussation of these fibres and the ligaments and membranes of the diaphragm (Fig. 2). The most important of these

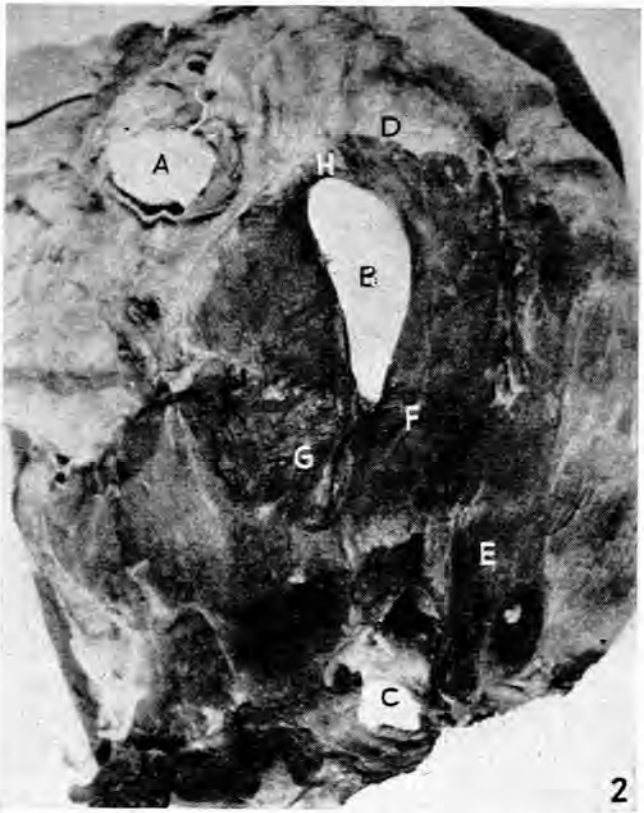

Fig. 2. Dissection of hiatal structures viewed from below. Note that the elliptical shape of the hiatus arises naturally from the origin and insertion of the hiatal limbs, which in this case are derived entirely from the right crus. A=Inferior vena cava. B=Oesophageal hiatus. C=Aorta. D=Transverse membrane of central tendon of diaphragm. E=Left crus. F=Left hiatal limb of right crus. G=Right hiatal limb of right crus. H=Anterior decussating muscle fibres.

latter are the phreno-oesophageal ligament and the transverse membrane of the diaphragm. So long as this shape is maintained and the size remains sufficient only to transmit the oesophagus, the hiatal limbs will provide direct support to the gastro-oesophageal ligament (Marchand, 1957). Should diaphragmatic support be lost by widening of the hiatus, the phreno-oesophageal ligament must bear the full brunt of the abdominal pressure. The cardia will be displaced upwards and the acute angle of entry of the oesophagus into the stomach will be altered.

THE MECHANISM OF REGURGITATION

Theoretically, regurgitation could be due to one of two basic causes:

1. Increase of the intragastric pressure above levels normally withstood by the resisting mechanism.
2. Weakening of the anatomical sphincter mechanism.

1. *Increase of Intragastric Pressure*

Regurgitation can be induced simply by elevating the intragastric pressure. This may be due to active or passive causes.

Passive Causes. Cannon (1911) has shown that the stomach, like the bladder, accommodates for its increasing contents by reflex relaxation of its muscular walls, so that excessive pressure rise is prevented. However, considerable and sharp elevation in tension is caused by postural changes such as stooping, bending forwards, or lying supine. Fig. 3 illustrates graphically the rise in pressure within the stomach of a subject who adopts the 'head-down' position. This is an entirely physical phenomenon caused by the direction of the thrust of the abdominal contents affected by gravity.

Gastric distension also affects the intragastric pressure, and this can be proved by simple experiment. A subject is given graduated amounts of watery barium to swallow against a constant abdominal compression force of 120 cm. of water (by means of the balloon technique described above) and the effects are then viewed by radioscopy. In a series of 10 young adults, no one regurgitated after 500 ml. had been swallowed, 3 regurgitated after 1000 ml. had been taken and 5 others after 1500 ml. The effect of gastric distension has been recorded manometrically and Fig. 4 shows the immediate rise in gastric pressure caused by swallowing an effervescent powder.

Intraperitoneal pressures are passively transmitted to the stomach and when the peritoneal capacity is decreased, as in pregnancy or adiposity, slight postural changes will cause an inordinate rise in the intragastric tension. The effects of diaphragmatic contraction also cause a considerable pressure variation within the stomach and with deep inspiration the pressure gradient between stomach and oesophagus may be very considerable (Fig. 5). With straining, both pleural and peritoneal pressures rise so that the gradient at first remains constant. Only on very forceful exertion does the pressure gradient across the diaphragm rise sharply. (Marchand 1955 and 1957).

Active Causes. Gastric contractions can also cause a sharp increase in the intragastric pressure particularly if the stomach is distended. Normally it is the empty stomach which is the most active, whereas the full stomach is at rest (Alvarez, 1948). When gastric contractions are associated with pyloric closure the intragastric pressure may rise violently and cause regurgitation. This is what happens during vomiting.

2. *Weakening of Anatomical Sphincter Mechanism*

Disruption of the normal oesophago-gastric angle occurs classically in sliding hiatus hernia. In this condition the cardia is displaced upwards into the chest and the normal anatomical angle is abolished. The tonic contraction of the terminal oesophagus still provides a weak defence, and an increase in gastric tension following postural change, deep inspiration, etc., is usually necessary to cause regurgitation. The tonic closure of the terminal oesophagus seems just capable of withstanding the normal pleuroperitoneal pressure gradient.

DISCUSSION

The results of the experiments and observations here described are supported by clinical experience. It is almost

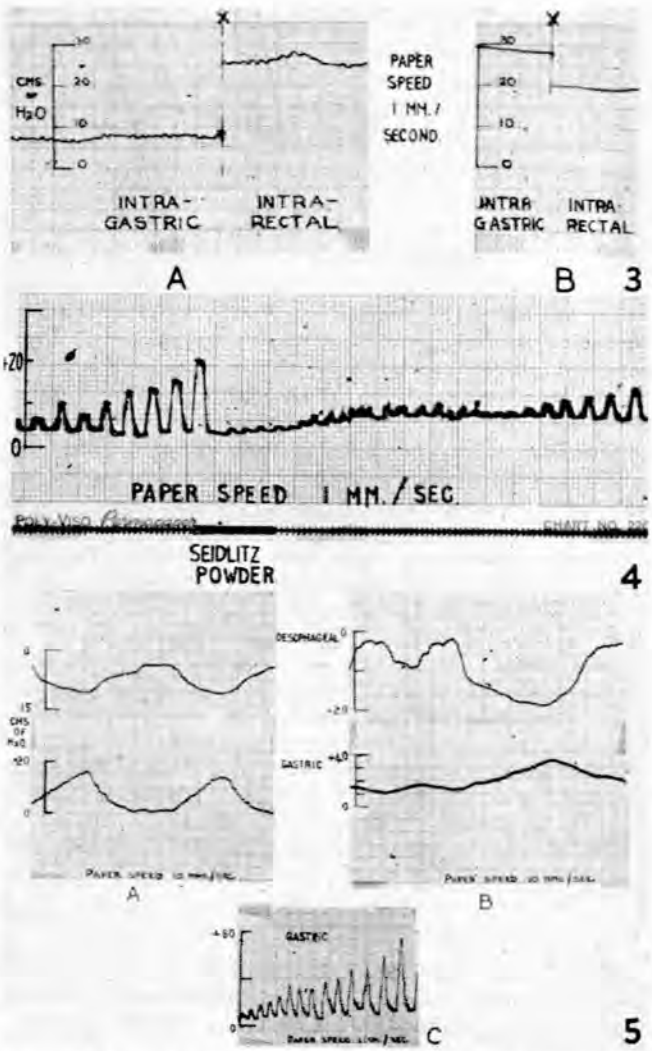

Fig. 3. Immediately consecutive intra-gastric and intra-rectal pressure tracings. A. With the subject erect, the gastric pressure is 18 cm. of water less than in the rectum. B. With the subject inclined head-downwards at an angle of 45° from the horizontal, the intra-gastric pressure is 8 cm. of water higher than the rectal. Reproduced from *Thorax* (1957): **12**, 189.

Fig. 4. Gastric pressure tracing illustrating the effect of distention with gas. The second Seidlitz powder was swallowed whilst the continuous line by the time-marker was being written. There is an immediate rise in the basal gastric pressure. The subject progressively increased the depth of inspiration before and after the powder was taken. Reproduced from *Thorax* (1957): **12**, 189.

Fig. 5. Simultaneous intra-gastric and intra-oesophageal pressure. A. Normal respiration. B. Deep respiration. C. Only gastric pressure recorded, with progressively deepening inspiration. With quiet respiration a pressure gradient of 30 cm. of water exists across the diaphragm. This rises to 60 cm. of water on deep breathing. Reproduced from *Thorax* (1957): **12**, 189.

invariable to find, when investigating a case of flatulent dyspepsia, either that there is some cause for an enhanced regurgitating force or some cause for a weakened defence. Pregnant women constantly complain, and well they might for, in addition to a rapidly growing abdominal tumour which raises the weight and pressure of the abdominal contents, they suffer the effects of softening and laxity of the collagen fibres in their muscles and membranes. As a consequence, the support which the hiatus affords the cardia is weakened and hiatus herniation is not uncommon.

The second large group of sufferers are previously vigorous men who abandon active sport in their thirties and then gain weight. In the presence of a still strong abdominal musculature the accumulation of abdominal fat causes a marked rise of the basal abdominal pressure and the development of flatulent dyspepsia. Not infrequently the inexorable pressure disrupts the hiatus, resulting in hiatus herniation, though they may be saved from this, and cured of their dyspepsia, by losing abdominal muscle tone and increasing their abdominal capacity so that a sagging or rotund paunch develops.

Our wasp-waisted grandmothers paid the price of fashion in flatulent dyspepsia; their repressive corsets left little room for a well-filled stomach. All of us have suffered, at least occasionally, either because we have filled our stomachs to drum-like tension in an act of gluttony, or because we have eaten a particular food which causes our pylorus to close in protest. Similarly duodenal ulceration, cholecystitis and appendicitis cause flatulent dyspepsia because of the associated pyloric spasm. The reason for symptoms in people with sliding hiatus hernia is obviously the loss of the normal acute oesophago-gastric angulation, and it is interesting to note that, with the development of an oesophageal stricture, which physically limits regurgitation, symptoms of flatulence improve. Also sufferers from the less common para-oesophageal hernia do not complain of flatulent dyspepsia, for their acute angle of entry of the oesophagus into the stomach is retained.

CAUSES OF FLATULENT DYSPEPSIA

From this brief consideration of the factors which produce, and those which prevent, gastro-oesophageal regurgitation, the following classification of the causes of flatulent dyspepsia may be made:

1. Conditions associated with *weakening of the resisting sphincter mechanism*:
   - (*a*) Hiatus hernia
   - (*b*) Hypotonicity of muscle and laxity of membrane—as seen in debilitating conditions and senility.

2. Conditions associated with *diminution of the abdominal capacity*
   - (*a*) Obesity
   - (*b*) Pregnancy
   - (*c*) Abdominal tumours
   - (*d*) Gastric distension—gluttony, aerophagy.
   - (*e*) Constricting abdominal garments.

3. Conditions associated with active *increase in the intragastric pressure*
   - (*a*) Pyloric spasm. This is caused by such conditions as peptic ulceration of the duodenum, gall-bladder disease, appendicitis, etc.
   - (*b*) Gastric irritation, caused by dietary indiscretion, etc.

During the course of 1 year, whilst engaged in general surgical duties at the Johannesburg General Hospital, I fully investigated every one of my out-patients who complained of flatulent dyspepsia. There were 72 patients in all. Cholecystitis or cholelithiasis was the cause in 22, duodenal ulcer in 15, hiatus hernia in 9, 'right inguinal fossa' pathology in 7, gastric ulcer in 4, obesity in 5 and ventral hernia in 3; 7 patients were not categorized. Thus, in my experience of hospital patients who suffer from flatulence and indigestion, 30% have gall-bladder disease, 20% duodenal ulcers and 12% hiatus herniae. The symptoms are the same because their basic cause is gastro-oesophageal regurgitation. This is not to say that the various conditions cannot be clinically distinguished; indeed in most instances each condition adds its own peculiar facet to the dyspepsia. With gall-bladder disease the role of fatty foods in the production of dyspepsia and the right hypochondrial pain provide strong clues; with duodenal ulceration, the hunger pains and the relief afforded by food are characteristic; whilst postural intensification of dyspepsia is strongly suggestive of hiatus hernia. However, in all these conditions reflux dyspepsia is not infrequently the major complaint, and the final differentiation must then depend upon special investigations. Of course the vast majority of sufferers accept their discomfort as a more or less normal visitation and are never seen in hospital practice. They are in the general practitioner's own particular domain, for temporary relief is readily afforded by antacids such as Amphogel, Rennie's tablets, bismuth and a host of others. These act by neutralizing the irritating enzymes so that the oesophagus, though still the recipient of stomach contents, gains a temporary respite. If, however, one appreciates the basic factors underlying the complaint, management can be far more confident and effective. Most practitioners readily recognize cases where cholecystitis and peptic ulceration are responsible, for here specific symptoms shout the diagnosis. Hiatus herniation, however, is very frequently treated lightly and, in my series of 92 cases, the average time that elapsed between the onset of symptoms and definitive diagnosis was 12 years. The first duty of the doctor called to advise a patient with habitual dyspepsia is to make a diagnosis and, where a postural history is obtained, a barium meal with abdominal compression or with a Trendelenburg tilt of at least 60° from the horizontal must be done.

Treatment should depend on the cause. An obese patient will be cured by weight reduction and nothing else. A diseased gall-bladder, an ulcerated duodenum, or an abdominal tumour, must be effectively managed. A pregnant woman must wait her delivery, though dietary management, to prevent overeating and gain in weight above that normally expected, will ensure greater comfort. With hiatus herniation the management is a matter of judgment for here, in addition to the disruption of the defensive mechanism, an excessive extruding force, namely obesity, is frequently present. Weight reduction is of great benefit and should be insisted upon even when operation is contemplated. While this is not the place for a discussion of the indications for operation on hiatus herniae, this can be said: Operation will cure the dyspepsia, but it must not be considered by the patient as a licence to indulge the palate and increase the girth.

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