THE OESOPHAGEAL CARDIA AND REGURGITATION*

BY

R. N. SINCLAIR

Victoria Infirmary, Glasgow, Scotland

An understanding of the mechanisms governing the integrity of the cardia is of immense importance to the anaesthetist. The cardia is intimately concerned with the vital problems of vomiting, regurgitation, and aspiration of gastric contents into the lungs, and the lethal potentialities of these complications are only too well known. If any proof be needed, the Report on Deaths Associated with Anaesthesia (Edwards et al., 1956), published under the aegis of the Association of Anaesthetists, reveals the serious menace of regurgitation and aspiration. There cannot be an anaesthetist who has not experienced trouble and anxiety—and often—from these hazards. This being so, it might be interesting to present various views and findings which relate to this problem.

Having referred to the importance of cardia, it may seem odd to say that it is doubtful whether, in point of anatomical fact, it exists at all. In 1719, Claude Helvetius, French philosopher and physician, described a sphincter—the so-called cardiac sphincter—at the gastro-oesophageal junction. Since then, despite the most careful search, nobody has been able to provide any concrete evidence of the existence of this anatomical sphincter, at least not in any demonstrable fashion. It has been curiously controversial—a few anatomists have agreed that there is a morphological sphincter, but the majority have denied it, and in the meantime many ingenious theories have been put forward to account for the undoubted sphincter-like function at the lower end of the oesophagus. The position today is only gradually becoming more clear than when Helvetius first described a cardiac sphincter. There does seem to be an obvious everyday need to postulate such a control. Regurgitation from stomach to oesophagus would be continuous if there were not some arrangement to resist the force of intraperitoneal and intragastric pressure.

The cardia is usually taken to mean the localized region at the junction of the oesophagus with the stomach. There is, however, no clear-cut demarcation between oesophagus and stomach, as the change from the squamous epithelium of the oesophagus to the columnar epithelium of the stomach may not correspond with the gross anatomical appearances. Normally, the cardia is within the abdomen, and in this position acts as a competent valve, but if the cardia be in the chest, as in sliding hiatus hernia, this competence is diminished. It would seem, therefore, that the functional efficiency of this “sphincter” is very dependent on its anatomical shape and position. The competence is such that, in the normal position, the cardia yields readily to quite low pressures on the oesophageal side, while it is able to resist pressures up to 80 mm Hg on the gastric side. Indeed, even in the cadaver, the cardia can usually be shown to be competent. The physiological mechanism for opening the cardia is by reflex, on the act of swallowing or by the arrival of a peristaltic wave at the cardia.

There are four main theories to account for the competence of the cardia to prevent reflux.

(1) The anatomical sphincter.
(2) The intrinsic, functional sphincter.
(3) The extrinsic, diaphragmatic pinchcock.
(4) The valvular mechanism, related to the oblique angle of entry of oesophagus to stomach.

(1) The Anatomical Sphincter was described by Helvetius as a circular thickening of muscle fibres around the oesophageal orifice, and this observation appears to have been repeated by others off and on over the course of the years. Lerche

* Based on a Presidential Address to the Scottish Society of Anaesthetists, April 1958.
an American writer, went so far as to describe two sphincters; but it is a strange fact that modern anatomists appear unable to find any such structure. From the practical standpoint, and in view of the historical disagreement on the subject, one can take it that there is no real sphincter in the anatomical sense.

(2) The Intrinsic, Physiological Sphincter. The following observations strongly suggest the existence of such a functional sphincter. First, even when the cardia is above the diaphragm in sliding hiatus hernia, some sphincteric activity can still be detected at the cardia. Second, with corrosive burns of the oesophagus the distal few centimetres tend to escape, which suggests protective spasm in this area. Third, achalasia of the cardia is said to be due to the inability to relax of an otherwise normal sphincter mechanism. Many patients with achalasia who have had a Heller's operation (division of the muscular coats down to the mucous membrane) subsequently develop symptoms of reflux. Finally, while experimental proof of an intrinsic functional sphincter has been elusive through the years, the most recent experimental data continue to favour such a concept. For example, quite recent contributions by Atkinson and Rowlands (1957) and Fyke et al. (1956) have demonstrated, by means of combined manometry and radiology, a zone of high pressure interposed between stomach and oesophagus. This pressure is consistently higher than the gastric pressure, and is said to be typical of a sphincter anywhere. Here is the paradox—a physiological sphincter without an anatomical sphincter!

(3) The Extrinsic, Diaphragmatic Pinchcock. This theory is perhaps the most intriguing, though no longer acceptable. It is based on the idea that during contraction of the diaphragm, the crura exert a gripping action on the distal end of the oesophagus, and so effect closure. It was thought that this was the reason why a barium swallow is delayed at the diaphragm during inspiration, a fact long recognized by radiologists, but a more satisfying explanation of this phenomenon will be given in a later paragraph. Professor P. R. Allison (1951), whose work on reflux oesophagitis is responsible for so much of the recent revival of interest in this subject, has stressed the importance of the sling-like action of the right crus of the diaphragm, which sweeps over the terminal oesophagus at the angle it makes with the stomach. Professor Allison supports the theory that, when the diaphragm contracts, this right crus exerts a pull which increases the angulation at the oesophago-gastric junction.
and tends to close the lower end of the oesophagus. More recently Braasch and Ellis (1956), in an important experimental study on dogs, have shown that the distal segment of the oesophagus relaxes rather than contracts at inspiration. In point of fact, eructation and reflux occur only during inspiration, which would seem to contraindicate a pinchcock action. In this connection, on the occasions when one has been able to view this area for oneself through the oesophagoscope, it was found that, with the patient intubated with a well-fitting cuffed tube and breathing spontaneously, if one deliberately obstructed the respiration for a few breaths, the cardia could clearly be seen to open with attempted inspiration, in an obliquely linear fashion, or more widely. It was quite a definite and constant finding and is illustrated in the two coloured illustrations drawn from selected sequences from a cine-film shot via the oesophagus (figs. 1 and 2). This "opening" effect during obstructed inspiration was very striking, and one could clearly see the dangers of the accidental performance of this manoeuvre should the stomach be distended with fluid or the intragastric pressure high. It certainly gives the impression of a diaphragmatic pull, and suggests that contraction of the diaphragm actually opens the cardia rather than closes it—at any rate when respiration is obstructed. It is interesting to note that on direct examination at operation or via the oesophagoscope, neither a sphincter nor a valve can be identified, nor, during normal quiet respiration, can any cardiac opening be seen. The so-called "rosette" appearance described by some oesophagoscopists is probably no more than the slight narrowing of the oesophagus as it passes through the diaphragm.

(4) The Valvular Mechanism of cardiac competence is based on the oblique angle of entry of the oesophagus to the stomach. It is readily understood by reference to figure 3. The explanation of this valvular mechanism seems to be in the anatomical features, notably the acute or eccentric entry of oesophagus to stomach. The essence of this is that the intragastric pressure in itself will tend to assist closure. It is also suggested that an effective flap-valve is developed at the left lip of the oesophageal opening, and this is able to seal the cardia. These mucosal flap-valves have been observed in some animals and are said to be especially well developed in the guineapig. It seems likely that in the human subject, however, these flaps are functional rather than anatomical. Dornhorst et al. (1954) believe that the muscularis mucosae, which is well developed in the lower portion of the oesophagus, is able to pull the lax mucosa into a valvular form.

![Fig. 3](image-url)
Perhaps the most interesting of recent work on this subject comes from an essay by Paul Marchand (1954). Marchand showed the importance of the anatomical shape and position of the cardia by the following experiments on cadavers. He measured the intragastric pressures required to overcome the cardiac resistance under varying experimental conditions (fig. 4).

To go back now to the question of why a barium swallow is delayed at the diaphragm during inspiration. The barium flows quite freely during expiration, but on inspiration there is a delay at the cardia, while the oesophagus just above this level dilates up into a temporary bulge. This is called by the radiologists the "phrenic ampulla", and is a constant and normally occurring rhythmical dilatation and delay during inspiration.

The suggestion was that diaphragmatic pinch was responsible. A better explanation is that, with the difference of pressure ordinarily developed between thorax and abdomen during inspiration, this pressure differential could in itself be quite sufficient to account for any delay. On deep inspiration the negative pressure in the pleural space becomes more negative, while the positive pressure in the abdomen becomes more positive, and these pressures are transmitted directly to the oesophagus and stomach. In other words, at the end of inspiration there is a high pressure in the stomach and a low pressure in the oesophagus. One can readily see that such a pressure differential would constantly tend to produce regurgitation, but normally this is easily resisted by the competent cardia. However, with obstructed respiration, or with an already distended stomach, or both, it is quite another matter as very high pleuroperitoneal pressure...
The differences are set up. A glance at figure 5, taken from Dornhorst (1954), shows this quite clearly. Reference has already been made to this pleuroperitoneal pressure gradient as being a force constantly tending to favour regurgitation, especially during inspiration. This is exactly the means by which cows ruminate, or chew the cud. From time to time they inspire happily against a closed glottis and quietly regurgitate the ingesta back into the mouth. Veterinary surgeons say that a cow with a tracheotomy opening has the greatest difficulty in regurgitating at all. Theoretically at least, therefore, a preliminary tracheostomy would presumably lessen the chances of regurgitation and aspiration under anaesthesia. Some people with sliding hiatus hernia are, of course, ruminants too, because of the ease with which they regurgitate—and by the same mechanism of inspiring against a closed glottis.

There is a further anatomical point of practical concern to the anaesthetist. The lower half of the oesophagus is composed of unstriped, involuntary muscle, while the upper half is composed of striped muscle. Muscle relaxants, therefore, have no effect on the cardia and lower oesophagus, but, on the other hand, curare does relax and paralyse the upper oesophagus and the cricopharyngeal sphincter. In fact, vomiting and regurgitating should probably be regarded as a two-stage affair. Firstly, the cardia is overcome and stomach contents fill the oesophagus—and the oesophagus can hold a surprising volume of fluid, as much as a pint. Secondly, the cricopharyngeal sphincter relaxes and the fluid may then reach the pharynx and mouth, or even the air passages and lungs. In intestinal obstruction, for example, fluid may be already lodged in the oesophagus but only visibly regurgitated when the cricopharyngeal sphincter is relaxed. This relaxation of the cricopharyngeal sphincter occurs relatively early and suddenly when muscle relaxants or intravenous anaesthetics are used. In the case of inhalation anaesthesia the relaxation is more gradual, and this is really the basis of the safety of the straight "gas–oxygen–ether" technique, which frowns on the use of muscle relaxants or intravenous anaesthetics until the trachea is safely intubated with a cuffed tube.

It should be realized that regurgitation, unsuspected and covert, is possibly a lot more common than one may appreciate. Culver and Beecher (1951) in America investigated the incidence of regurgitation and aspiration in 300 unselected surgical patients, and Weiss (1950) did the same

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**Fig. 5**

Müller's manoeuvre. Inspiration against a closed glottis (Dornhorst, 1954).
with 112 cases. The technique of investigation was the same in both cases. A capsule containing 4 ml of 0.25 per cent Evans' Blue dye was swallowed with a little water half an hour before induction of anaesthesia. The anaesthetic was thiopentone, nitrous oxide, oxygen, ether. At the conclusion of the operation the pharynx, larynx, trachea and main bronchi were examined for the presence of the blue dye. The results were striking and in the two series agreed so closely that it will be sufficient to summarize the figures. Very briefly, 25 per cent of the cases regurgitated to the pharynx, and in 19 per cent of the cases the dye was seen below the larynx. It would seem, therefore, that in nearly one case in every five, during routine surgery and in the absence of frank vomiting, fluid stomach contents find their way into the lungs. If the dye was found below the larynx at all, it was most commonly seen in both the trachea and right main bronchus. Two further interesting points emerge from these figures: (a) that the incidence of aspiration to the bronchi was not markedly less when an endotracheal tube (presumably uncuffed) was used; (b) that the over-all incidence of regurgitation was 22 per cent with an experienced anaesthetist and 30 per cent with an inexperienced anaesthetist.

It might be of interest to discuss a few points on the practical anaesthetic management of the typical case which might give trouble, the surgical patient with intestinal obstruction. First of all let us remind ourselves of the factors which aggravate gastric retention—pain and anxiety, circulatory collapse, salt depletion, morphine, food and drink, and reverse peristalsis. All these factors should be considered and dealt with as best as may be.

With regard to premedication, perhaps morphine and such drugs are best withheld altogether from these patients and atropine alone given, as the following case illustrates. This was a man, aged 60 years, whose abdomen was much distended but who had not been vomiting. He was described as being apparently in reasonably good condition, and morphine 15 mg with atropine 0.6 mg was ordered. Half an hour later, while the patient was still in the ward and before even a gastric tube had been passed, he regurgitated, asphyxiated, and died. It is suggested that he might well have coped with this incident had it not been for the morphine.

Pre-operative gastric suction in these cases should preferably extend over an hour or two, either continuously or intermittently. The one-shot suction effort is hardly adequate. Recently the writer has been using a No. 12 gastric tube with an inflatable cuff in the oesophagus. Although, of course, the idea of blocking the oesophagus is not new, it was thought that a tube for the combined purposes of routine gastric aspiration and oesophageal blocking at the time of induction would be advantageous. Such tubes were made on request by M.I.E. Ltd. The tube is passed into the stomach in the ordinary way for pre-operative suction, and the cuff is inflated to block the oesophagus just immediately before induction of anaesthesia. Once induction is completed and a cuffed tube is in the trachea, the oesophageal cuff is deflated. The cuff is situated in the middle of the tube, i.e. 15 inches from either end. In use the cuff should lie about the middle of the oesophagus, or at a level in the oesophagus corresponding to just below the bifurcation of the trachea. It is inflated with 10 ml of air. The oesophagus is very distensible, and the risk of damage with this cuff seems remote. One or two patients have complained of slight substernal discomfort but mostly there is no discomfort at all, and there has been no damage following use of the tube.

It seems quite as logical to block the oesophagus in order to prevent stomach contents coming up, as to block the trachea to prevent stomach contents going down. The withdrawal or even change of position of a gastric tube should be avoided while the patient is unconscious, as the movement of the tube through the oesophagus moves the loose mucosa and favours regurgitation. Another little point worth remembering is that giving the patient a mouthful of water to swallow just before induction of anaesthesia helps to empty the oesophagus—a useful safety device.

The actual induction of anaesthesia is probably best carried out on the operating table, and in the head-up position to seek the assistance of gravity. Suction apparatus must, of course, be to hand. It is a matter of individual preference which anaesthetic agents are used, but it is certainly a mistake to think that anaesthesia must almost
necessarily be induced with thiopentone, come what may. As an alternative, half a dozen or so breaths of cyclopropane mixture followed by a relaxant is about as quick and on the whole safer. A straightforward “gas-oxygen-ether” induction without relaxants until after the trachea is intubated also has its advocates. It has already been pointed out that the safety of this method depends upon the ability of the anaesthetist to intubate the trachea under light anaesthesia and before the cricopharyngeal sphincter has relaxed.

It is obviously better to try to prevent aspiration of fluid into the lungs than to have to cope with this mishap after its occurrence. When, however, fluid has entered the air passages, the effectiveness of bronchoscopy as a resuscitative measure may be disappointing—not, of course, the immediate bronchoscopy necessitated by a massive flooding, but rather bronchoscopy after the inhalation of relatively small amounts of acid stomach contents. In this event, bronchoscopy may serve only to increase the spasm, and simple suction with a soft rubber catheter through the endotracheal tube may in the end be just as effective and is less disturbing.

SUMMARY AND CONCLUSIONS

The present accepted concensus of opinion regarding the modus operandi of cardiac competence may be summarized as follows. The greatest stress is laid on the eccentric angle of entry of the oesophagus into the stomach, and the flap-valve formed at the left lip. In addition, there is almost certainly a truly inherent activity at the cardia, which has the physiological characteristics of a sphincter. The diaphragm is not thought to have any part in maintaining cardiac competence, and indeed overaction of the diaphragm can result in incompetence. There is certainly no demonstrable anatomical sphincter. The pleuroperitoneal pressure differential is a force constantly tending to favour regurgitation. Finally, inspiration against a closed glottis opens the cardia, thus making regurgitation most likely. All of which brings us back—as so often happens—to that most basic and fundamental of all anaesthetic principles—keep a clear airway and never allow obstructed respiration.

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