THE APPLIED ANATOMY OF VOMITING

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SUMMARY

An attempt has been made to provide the anatomical background necessary for the understanding of the complex problem of vomiting. This has been dealt with in two sections: (1) active vomiting; (2) passive regurgitation. In the first section, the vomiting centre and its afferent pathways have been described, together with the detail of three "sphincters" which are concerned; these are the gastro-oesophageal, the laryngeal and the nasopharyngeal. In the second section, the various possible mechanisms for preventing gastro-oesophageal reflux have been discussed and a conclusion drawn as to the actual mechanism responsible. The intrinsic musculature of the oesophagus initiates closure of the "sphincter", and this is completed by the pressure difference between abdomen and oesophagus, which maintains apposition of the mucosal folds. When the normal mechanism is interfered with (e.g., in hiatus hernia), the action of the diaphragm may replace part of the normal mechanism.

The purpose of this paper is to attempt to describe the structures concerned in the act of vomiting. This will be considered from two aspects: firstly, true vomiting, that is the active expulsion of gastric contents through the mouth; secondly, the passive regurgitation of the gastric contents. Although both processes involve the same anatomical passages, namely the oesophagus and pharynx, the mechanisms are entirely different, as are the associated problems, and so the two kinds of regurgitation will be dealt with separately. On the one hand, there is the active process which is really part of the normal defence mechanisms of the body and, on the other, gastric reflux which is a breakdown of the normal mechanisms.

VOMITING

Vomiting is defined as the forceful expulsion of gastric and intestinal contents through the mouth. Immediately preceding vomiting are tachypnoea, copious salivation, dilatation of the pupils, sweating, pallor and rapid or irregular heartbeat, all of which are signs of widespread autonomic discharge. The problem of nausea, particularly with respect to its cause, is not relevant to the present discussion.

Any reflex must have its afferent pathway, its central connexion and its efferent pathway. In describing the anatomy of vomiting these can be broken down more specifically into a description of:

1. The vomiting centre.
2. The afferent pathways.
3. The efferent pathways and mechanics of vomiting.
4. The structure of the diaphragm.
5. The nerve supply to the diaphragm.

1. The vomiting centre.

Accurate localization of the vomiting centre is due to the work of Borison and Wang (1955). For a long time it was thought that the centre was in the midbrain in fairly close association with other vital centres, for example the respiratory. By differential destruction of brain tissue, they localized the centre to the dorsolateral border of the lateral reticular formation, lying just ventral to the tractus solitarius and its nucleus. It is now established that the region of brain lying superficial to the true vomiting centre, that is, nearer the ventricle, contains a chemoreceptor through which the vomiting centre itself is stimulated. Electrical stimulation of the dorsolateral border of the
the lateral reticular formation in experimental animals causes projectile vomiting. This begins after a short latent period during which inspiration occurs and ceases abruptly at the end of stimulation.

Some of the phenomena associated with vomiting have already been described, and it has been noted that these are the signs of a widespread autonomic discharge. The situation of the vomiting centre explains this association. Morphologically it is very closely related to neuronal loci regulating important visceral and somatic functions. These loci include: (a) the respiratory centres; (b) the vasomotor centre; (c) the salivatory nuclei; (d) the vestibular nuclei; (e) the bulbofacilitatory and -inhibitory systems.

The locus for vomiting is strategically situated in the core of these regulatory foci. A consideration of the act of vomiting shows that all the represented activities are involved in its motor expression.

(2) **Afferent pathways.**

The vomiting centre is activated only by afferent impulses which arise in many parts of the body; it is not stimulated directly by emetic substances carried to it by the blood (Davenport, 1961). Among the effective stimuli exciting afferent impulses are: tactile stimulation to the back of the throat; distension of the stomach or duodenum to a pressure of about 20 mm Hg; distension or injury of the uterus, renal pelvis or bladder; a rise in intracranial pressure; rotation or unequal stimulation of the labyrinths; acceleration of the head in any direction; and many kinds of pain, such as that attending injury to the testes. Some adults can vomit voluntarily, often by thinking of nauseating experiences, and many young children do so to express disapproval of parental actions.

There are two general pathways by which emetic substances or chemical changes in the body fluids affect the vomiting centre. The first is by way of a chemoreceptor trigger zone in the area postrema in the floor of the fourth ventricle. Stimulation of this zone by emetics in the blood or cerebrospinal fluid elicits vomiting. Destruction of this zone eliminates the response to centrally applied emetics and also the vomiting accompanying uraemia, radiation sickness and motion sickness. Other pathways lie in many afferent nerves, especially from the intestinal tract, which are activated by noxious substances or drugs. These afferent pathways have been very fully investigated by the use of different sorts of stimuli from different organs and dividing the appropriate afferent nerves. Borison and Wang (1953), for example, used as the stimulus oral copper sulphate. They were able to confirm the results of many other workers that the afferent impulses pass both in the abdominal sympathetic and vagus nerves. Copper sulphate in the stomach acts as an emetic, and the afferent impulses run in the visceral nerves; but, after complete denervation of the gut, absorbed copper sulphate still causes vomiting by stimulating the chemoreceptor trigger zone. After ablation of the trigger zone, even a lethal dose of copper sulphate will not cause vomiting. Either the sympathetic nerves or the vagus will suffice to transmit emetic impulses to the vomiting centre. Borison and Wang (loc. cit.) demonstrated that the vagus is the more important pathway, since no change in the emetic threshold level of copper sulphate occurred after sympathectomy alone, whereas a twofold increase in threshold occurred after vagotomy alone. In this connexion, Walton et al. (1931) made the interesting observation that, although the somatic innervation to the parietal peritoneum remained intact, peritonitis did not cause vomiting after abdominal sympathectomy and thoracic vagotomy. They arrived at the conclusion that only visceral afferent fibres are important in the act of vomiting. However, it is by no means established that somatic afferents play no part in the vomiting reflex.

(3) **Efferent pathways and mechanics of vomiting.**

The efferent pathways of the vomiting reflex are many and various, both somatic and visceral pathways being involved. It is proposed to give a general description of the mechanics of the act of vomiting and then to describe in detail the anatomy of some of the more important structures concerned.

The mechanics of vomiting may be stated as follows (Davenport, 1961):

Vomiting begins with deep inspiration. The glottis is closed and the nasopharynx partly or completely shut off. Inspiration is converted to an expiratory effort with simultaneous contraction of the abdominal muscles. Because the glottis is closed, the increase in intrathoracic and intra-
abdominal pressure is transmitted to the stomach and oesophagus. The body of the stomach and the muscle of the oesophagus relax. At the same time, a strong annular contraction at the angular notch of the stomach nearly divides the body from the antrum. While the body of the stomach remains flaccid, peristaltic waves sweep aborally over the antrum. Due to the positive intrathoracic and intra-abdominal pressure, the gastric contents are expelled out of the mouth. The oesophagus is then emptied partly by the elevated intrathoracic pressure and partly by peristaltic waves stimulated by vomitus in the gullet and the mouth. Finally, the voluntary muscles relax and respiration resumes.

The first point raised in the above description is that the act of vomiting begins with inspiration. This is confirmed by work on experimental animals by Borison and Wang (1953). They describe electrical stimulation of the vomiting centre being followed by a short latent period to allow maximal inspiration. Following this act of inspiration, respiration ceases during vomiting and is not resumed until the vomiting ends. The close proximity of the centres for vomiting and respiration in the lateral reticular formation of the brain stem indicates their close functional association. To what extent one can influence the other is not known. It may be that the vomiting centre which controls a vital reflex defence mechanism can influence temporarily the respiratory centres with the effect that vomiting will occur with the temporary cessation of respiration. Such association would be of extreme importance in the administration of drugs which, although stimulating respiration, may also excite the vomiting centre.

Action of laryngeal musculature. This will be considered from two aspects; firstly, the closure of the glottis and, secondly, the closure of the inlet to the larynx.

Closure of the glottis. This is achieved by the approximation of the vocal folds due to the action of three muscles, namely the interarytenoid and the two lateral crico-arytenoid muscles. The interarytenoid muscle consists of transverse fibres which connect the posterior and part of the medial surfaces of the arytenoid cartilages to each other. Contraction of the muscle draws the arytenoid cartilages upwards along the sloping shoulders of the cricoid lamina, approximating them without rotation.

The lateral crico-arytenoid muscles arise from the upper border of the cricoid arch, and the fibres pass upwards and backwards to be inserted into the muscular processes of the arytenoids. The action of the two muscles, in drawing the muscular processes forwards, is to cause medial rotation of the arytenoids and hence approximation of their vocal processes, with resultant adduction of the vocal cords.

As part of the intrinsic musculature of the larynx, these muscles are supplied by the recurrent laryngeal nerve.

Closure of the inlet to the larynx. The aryepiglottic muscle has been described as the sphincter of the laryngeal inlet. The aryepiglottic fold contains this muscle, which runs from the side of the epiglottis to the apex of the corniculate cartilage and then continues obliquely, crossing its fellow on the posterior aspect of the transverse interarytenoid muscle, to be attached to the muscular process and posterior surface of the opposite arytenoid cartilage. These oblique fibres are often described as a separate muscle, but, from the functional point of view, it is better to think of this muscle-complex as a complete sphincter of the inlet to the larynx. Its contraction draws the two arytenoids towards each other and pulls them forward, so that they come into contact with the cushion of the epiglottis. The muscle complex is supplied by the recurrent laryngeal branch of the vagus nerve, whose fibres are, of course, not true vagal fibres, but are given to the vagus by the cranial accessory nerve, and arise in the nucleus ambiguus.

Closure of the nasopharynx. There is also a sphincter-like mechanism to close off the nose from the pharynx. Two muscles are responsible for this action. The palatopharyngeus and the levator palati. The anterior head of the palatopharyngeus arises from the posterior border of the hard palate, and the lateral fibres of this origin form a separate muscle bundle, which arises from the lateral aspect of the posterior border of the hard palate and passes posteriorly to encircle the pharynx within the fibres of the superior constrictor. Contraction of these fibres produces a ridge in the posterior wall of the pharynx known as Passavant's Bar. The soft palate is pulled up
against this ridge by the second muscle, the levator palati. This muscle arises from the quadrato area at the apex of the petrous temporal bone, anterior to the carotid foramen, and is inserted into the nasal surface of the palatine aponeurosis between the two heads of the palatopharyngeus. The two levator palati muscles in passing down to the palate are directed forwards and medially, and together form a U-shaped muscular sling. There are thus two U-shaped slings encircling the nasopharynx. The two levators constitute a sling in a downward and forward direction, and the lateral fibres of the palatopharyngeus form a sling running horizontally and backward to clasp the lower parts of the levators. Contraction of both slings shortens them, makes them interlock and thus closes off the nose from the pharynx.

The nerve supply to both the levator palati and the palatopharyngeus is from the pharyngeal plexus, that is, from the pharyngeal branch of the vagus to that plexus. Again, these are not true vagal fibres, but originate in the nucleus ambiguus. (4) Structure of the diaphragm.

Any discussion both on vomiting and gastro-oesophageal reflux must include a mention of the diaphragm. Two points in the anatomy of the diaphragm are of special interest, because there has been much argument about them. These are, firstly, the detailed structure of that part of the diaphragm surrounding the oesophageal hiatus and, secondly, the nerve supply of the crura.

Oesophageal hiatus. For a long time the classical description of the oesophageal hiatus of the diaphragm has been that it is surrounded by fibres of the right crus. This is still the description in many textbooks. Collis et al. (1954a) described the anatomy of the crura based on fifty dissections, and produced some extremely good photographs of these dissections. They found that the classical story does not hold true in every case; in 66 per cent of the dissections, the right crus did in fact form the boundary of the oesophageal hiatus; of the remaining 34 per cent the left crus contributed to a varying degree to the left border of the hiatus in 32 per cent, and was entirely responsible for surrounding the hiatus in 2 per cent of cases. This last was in subjects without situs inversus when one might expect a reversal of the normal pattern of the crura. Carey and Hollinshead in 1955 followed up this work with a further study of the oesophageal hiatus and were able to confirm the above findings. These observations are extremely important, particularly in relation to surgery of this region when one considers that many of the theories about diaphragmatic action and operative procedures have been based on the classical description still given in the current edition of some textbooks of anatomy that “the oesophageal hiatus is elliptical in shape and is formed by the splitting of the fibres of the right crus”. This apparently is true in only two-thirds of cases. The part played by the diaphragm in preventing gastro-oesophageal reflux will be dealt with later.

(5) Nerve supply of crura.

The fact that the phrenic nerve supplies the diaphragm with motor fibres is well established, but recently there has been some doubt raised as to whether the crura also have this same nerve supply. Last (1956) states that the crura are supplied by the intercostal nerves because they are part of the prevertebral musculature. This is a convincing argument if the crura be considered as arising from the anterior longitudinal ligament and inserted into the central tendon. On the other hand, the detailed structure of the crura suggests that it would be better to consider them as arising from the central tendon and having an insertion into the vertebral region (Collis et al., 1954b). It then might be expected that the crura would have the same nerve supply as the remainder of the diaphragm. In 1954, Collis et al. performed detailed dissections of the termination of the phrenic nerves in fourteen cadavers. A summary of their findings is as follows:

(a) The nerve supply to the left crus is from the posterior branch of the left phrenic nerve.

(b) The left phrenic nerve also supplies the part of the right crus which lies to the left of the oesophageal hiatus.

(c) The right phrenic nerve supplies the right portion of the right crus and the muscle of Low, when present.

That the phrenic nerve is the sole motor nerve to the diaphragm is confirmed by Botha (1957a). He based his views on dissection in man and animals and on experimental work in animals, in which he stimulated the different branches of the
phrenic nerve. He also states that the phrenic nerve supplies ipsilateral fibres of the oesophageal hiatus, regardless of which crus gave origin to the fibres.

More detailed work was carried out in 1958 by Ogawa et al., who worked with freshly killed dogs. They stimulated the phrenic nerve, then dissected the nerves and then stimulated them again more peripherally. They confirmed the previous findings that the phrenic nerve supplies the crura, but also found that, in 30 per cent of cases, the left posterior branch of the left phrenic nerve supplied fibres to the right side of the hiatus. This, they say, may account for the lack of paralysis of the crura when the phrenic nerve is cut, which is one of the arguments supporting the view that the lower intercostal nerves play some part in the motor supply of the crura. The lower intercostal nerves do, of course, provide some sensory innervation to the peripheral parts of the diaphragm.

GASTRO-OESOPHAGEAL REFLUX

The nature of the mechanisms preventing gastro-oesophageal reflux has been a matter of dispute since at least the eighteenth century. Controversy still exists concerning both experimental observations and their interpretation. There is general agreement that a closing mechanism of some sort must exist. This is apparent from an appreciation of the pressure gradient between the stomach and the oesophagus. The intraluminal pressure of the stomach is from 10 to 25 mm Hg higher than that in the oesophagus on inspiration. If there were no mechanism to close the lumen between the two cavities, reflux would readily occur whenever favoured by gravity.

Since 1719, when Helvetius described a sphincter at the cardia, various theories have been advanced to explain the mechanism which prevents reflux. Many of the older ideas are now thought to be incorrect, but some are relevant to this present discussion and may well help to explain the closure mechanism. Vantrappen et al., in 1960, reviewed the work done on this subject, examined the different theories with their experimental support, discussed the interpretation of the observations and suggested other hypotheses.

Several ideas have been put forward as to the factors responsible for preventing reflux and they are as follows:

1. Anatomical sphincter.
2. Flap valve mechanism.
3. Diaphragmatic action.
5. Physiological sphincter.
6. Mechanical factors.

(1) Anatomical sphincter.

Although it is now accepted that there is no true anatomical sphincter at the cardia, this has not always been the case. In some histological sections, there is an apparent increase in the circular muscle fibres at the cardia, and it is this finding which has been the basis of the controversy. Following the work of Lendrum in 1937, this description of the muscle layers at the gastrooesophageal junction may now be given. The outer longitudinal muscle layer of the oesophagus passes through the gastro-oesophageal junction without causing a thickening at the cardia and without dipping into the circular layer. The inner circular layer also continues down to the stomach, where it becomes continuous with the middle layer of the muscle coat of the stomach which encircles the entire organ. The oblique fibres from the stomach form a U-shaped band that borders the cardiac incisura and extends downwards, one arm on the anterior and one on the posterior surface of the stomach. These oblique fibres blend with the circular layer of the cardia. It is probably these fibres which have given rise to so much controversy in the past.

(2) Flap valve mechanism.

The idea that the angle made by the oesophagus with the fundus of the stomach which encircles the entire organ. The oblique fibres from the stomach form a U-shaped band that borders the cardiac incisura and extends downwards, one arm on the anterior and one on the posterior surface of the stomach. These oblique fibres blend with the circular layer of the cardia. It is probably these fibres which have given rise to so much controversy in the past.
gus and the right wall of the fundus of the stomach. These experiments have been repeated under more controlled conditions and, in recent years, a distinction made between the results obtained in cadavers and those in living animals or human subjects (Nauta, 1956). One point brought out in these later experiments is that the so-called flap valve mechanism is only effective if the cardia be in its normal anatomical surroundings. Particularly important is support to the right side of the lower oesophagus.

It was with this background that the basis for many of the operations for repair of hiatus hernia was established. Collis (1957) describes the operation for hiatus hernia with short oesophagus, and his main thesis is the re-establishment of the gastro-oesophageal angle. He also ensures that the right crus is active; thus the operation involves at least two of the possible mechanisms for preventing reflux, and, by altering the immediate relationships of the lower end of the oesophagus, it may be that other mechanisms are involved as well. One cannot therefore quote this operative technique and its success in support of the theory that reflux from the stomach into the oesophagus is prevented by the flap valve mechanism.

(3) Diaphragmatic action.

There are three ways in which the diaphragm could help to prevent gastro-oesophageal reflux. They are:

(a) Pinchcock action.
(b) Maintenance of the oesophagogastric angle.
(c) By the attachment of the oesophagus to the diaphragm.

(a) Pinchcock action. By this is meant the actual nipping of the lower oesophagus by the diaphragm. This idea grew up following the classical description of the oesophageal hiatus as being surrounded by fibres of the right crus. In 1930, Whillis wrote for his M.D. thesis "some observations of the anatomy and physiology of the lower end of the oesophagus". The anatomical contribution to this was a study of the oesophageal hiatus. Whillis described the margins of the hiatus in two layers, the right lying inferior to the left. Both these margins are concave and he describes their action to be like that of a shutter in a camera. When the diaphragm contracts, the concavity of the two edges bordering the hiatus will straighten, thus narrowing the opening and compressing the oesophagus. Although there is no doubt as to the accuracy of the anatomical findings in this work Whillis's conclusions are not so readily acceptable in view of more recent experiments. According to Whillis, the shutter mechanism of the hiatus would nip the oesophagus when the diaphragm contracted, that is, during inspiration. Creamer (1955) has found that reflux only occurs during inspiration, which is directly opposed to the above view. Braasch and Ellis (1956) performed some experiments on dogs which are relevant to the present discussion. They found that interference with the crural mechanism, either by destruction or displacement, produced no reflux, but the moment they interfered with the cardia by either Heller's or Wendel's operation (in both of which the muscle of the cardia is divided) reflux occurred. They inferred that the diaphragm is not responsible for preventing reflux, but that there must be some intrinsic sphincter mechanism.

The fact that a barium swallow may be held up at the level of the diaphragm on inspiration has been interpreted as an argument in favour of the constriction of the diaphragm. That the delay may occur well above the apparent diaphragmatic shadow does not necessarily invalidate this argument. Botha (1957b) attached metal clips to the hiatal wall at operation and showed that, in some positions, the diaphragmatic hiatus projected above the diaphragmatic shadow. The delay in the passage of barium does not necessarily indicate a pinchcock action of the diaphragm. Intra-abdominal pressure increases on inspiration and reaches a level well above intrathoracic and intra-oesophageal pressures. This pressure differential is sufficient to explain the delay of passage of barium from the oesophagus into the stomach. In 1959, Creamer et al. measured these pressures within the oesophagus and stomach and found that the pressure within the oesophagus and stomach and found that the lower part of the oesophagus did not open until the intra-oesophageal pressure was greater than the intra-thoracic pressure. They also found that the delay in the passage of barium on inspiration was only 0.125 second. This they consider much too rapid for muscle contraction and must be due to some mechanical effect.

(b) Maintenance of the oesophagogastric angle. Although it is not true to say that the lower oesophagus is always surrounded by fibres of the right
crus, it is a fact that the hiatus is bounded by fibres derived from one or both crus. When the crus contract, they must have some effect on the oesophagus, and this can be seen radiologically. There is no doubt that the line of the lower oesophagus is altered during contraction of the diaphragm. In considering the anatomy of repair of hiatus hernia with oesophageal reflux, Allison (1951) emphasizes the action of the crus. He describes it, not as a pinchcock, but as a sling, and likens this mechanism to that of the pubo-rectalis part of the levator ani which helps in the sphincteric control at the opposite end of the intestinal tract. In 1957, he described an operation for hiatus hernia with short oesophagus, and his main point is the re-establishment of the oesophago-gastric angle. In this operation, he ensures that the right crus is made efficient.

Relevant to this discussion is the work of Nauta (1956), although this was carried out only in dogs and not in the human subject. He considers that the role of the diaphragm in the closing mechanism between oesophagus and stomach is a passive one, namely, to maintain the oesophago-gastric angle. The absence of reflux after destruction of the action of the crus (Braasch and Ellis, 1956) indicates that this is not the sole mechanism of competence at the cardia, but it almost certainly plays a subsidiary role, and may be of importance when some of the other mechanisms are disturbed.

(c) The attachment of the oesophagus to the diaphragm. This is not strictly an action of the diaphragm, but it is best considered here. Mention has already been made and will be made later of the pressure gradient between abdomen and thorax. The higher pressure within the abdominal cavity would tend to extrude the lower oesophagus through the hiatus, were it not anchored there. Allison (1951) places great emphasis on the integrity of this anchorage, the oesophagophrenic ligament, in describing the operation for repair of sliding hiatal hernia.

(4) Mucosal valve.

Closure of the cardiac orifice by folds of gastric mucosa which have the appearance of a rosette was suggested by Magendie in 1833. Until recently the evidence in favour of this mechanism was indirect. Dornhorst et al. (1954) studied the closing mechanism of the oesophagus by intraluminal pressure measurements, radiologically, by direct palpation of the cardiac region during the course of gastric operation, and by observation of the gastro-oesophageal angle during regurgitation. They found no evidence in favour of a pinchcock action of the diaphragm or of a flap valve mechanism. In spite of this, there was a definite valve-like resistance to retrograde flow and they concluded that this is due to a mucosal valve. Such indirect evidence is of little value, but support for this idea comes from the work of Botha in 1958. He carried out extensive experiments on cadavers, several species of animals and human subjects during life. By careful palpation of the cardiac orifice during the course of gastric observations, he found evidence of occluding folds in every patient. Botha considers that the mucosal folds and the internal sphincter together form the closing mechanism between stomach and oesophagus. As will be seen later, this may not be the whole story, but it is interesting to note that the mucosal folds may be responsible for a watertight closure. One further observation by Botha was that, after a tube had been introduced through the cardia, these folds became patulous and inefficient. This possible contributory factor to gastro-oesophageal reflux could be important in these days of frequent gastric intubation.

(5) Physiological sphincter. It has already been stated that there is no evidence for the presence of a true anatomical sphincter at the lower end of the oesophagus. The work of Rash and Thomas (1962), however, does suggest that the muscle in this region may have a special function. They describe the autonomic nerves in the mammalian oesophagus, stomach and duodenum, with emphasis on the variation at the two junctional regions. They did not actually perform neuron counts, but the relative density of the plexuses and of the size of ganglia were observed. They confirmed the work of Irwin (1931) and Matsuo (1934), who calculated that the density of the myenteric neurons in the oesophagus reaches a maximum just above its lower third; from this point downwards, there is a gradual decrease in the number of cells until just above the gastro-oesophageal junction. From here, they increase again in number over the stomach to reach a maximum over the pyloric region, before a progressive decline in their number occurs along the duodenum. It is suggested that this concen-
tration of neurons over the lower oesophagus and at the pyloric sphincter is associated with the specialized activity of these regions.

There is no doubt that the physiological characteristics of the gastro-oesophageal junction are those of a sphincter mechanism. The older data suggestive of a sphincteric action include the demonstration that the cardia is normally closed, but that this closure can be made incompetent by reflex means, as evidenced by regurgitation, and that various gastric stimuli increase the resistance to gastro-oesophageal reflux. Direct observation of the cardia by oesophagoscopy and through gastrostomy shows that this segment of the oesophagus is normally closed, but the early work gives no indication as to the mechanism of this closure. It could result from either intrinsic tonic contraction of the oesophageal musculature or as a result of extrinsic factors. It is established that reflex opening of the closed segment of the oesophagus occurs under certain stimuli. These include swallowing or distension of the oesophagus. Vagal stimulation also lowers the threshold of intragastric pressure necessary to produce reflux. The resistance of the cardia to reflux can also be increased by reflex means. Distension of the gastric wall, irritation of the gall bladder and distension of intestine all produce an increase in the tone of the cardia, as measured by alteration of the intraluminal pressure.

Fyke, Code and Schlegel (1956) have demonstrated a zone in the lower end of the oesophagus where the resting pressures are higher than in the gastric fundus regardless of the position of the subject. This region has been termed the "high pressure zone". The pressures in this segment, which extends from 1–2 cm below the diaphragm to 1–2 cm above it, are such that, during both phases of respiration, there is an area of high pressure between the stomach and the oesophagus. The pressure in this zone falls 1.5–2.5 seconds after the onset of deglutition, thus abolishing the barrier. This decrease in pressure persists for 5–6 seconds until a peristaltic wave reaches the segment.

This high pressure zone could be due to tonic contraction of the circularly arranged muscle fibres in this segment of the oesophagus, or it could be attributed to external compression. The diaphragm is the only organ capable of participating in such a mechanism, and it can be shown not to be responsible. Physiological studies in patients with sliding hiatus hernia have indicated that the high pressure zone, when present, is found at the junction of the oesophagus and the herniated stomach, not at the level of the diaphragm (Atkinson et al., 1951). Further evidence in favour of an intrinsic sphincter mechanism is provided by observations on patients with scleroderma who, despite the normal anatomical relationship between oesophagus and diaphragm, have a decrease in, or absence of, a high pressure zone. This physiological alteration is a result of sclerodermatous changes in the oesophagus. Gastro-oesophageal reflux is exceedingly common in this group of patients.

(6) Mechanical factors.

If the intrinsic sphincter mechanism of the lower end of the oesophagus were solely responsible for preventing gastro-oesophageal reflux, there should be a close correlation between the pressure exerted by the sphincter and the occurrence of reflux. This is not always the case. Edwards (1961), in a symposium on the mechanism at the cardia, states that, in his experience, the association of gastro-oesophageal reflux and poor sphincter tone is not a close one. There is, then, some other factor concerned and this is probably mechanical.

It is possible to put forward a mechanical theory of the antireflux mechanism which is compatible with a wide range of clinical and radiological observations (Edwards, 1961). Two important points must be made regarding the detail of the anatomy of the oesophageal hiatus region as a preliminary to this theory, which is based on the idea put forward by Creamer et al. in 1959.

The first point is that the lowest 2–3 cm of the oesophagus is below the level of the diaphragm, that is, it is intra-abdominal and therefore subjected to intra-abdominal pressure. The second point is the attachment of the oesophagus to the diaphragm by the phrenico-oesophageal ligament. This is described as an elastic membrane which springs from the pillars and fascia of the diaphragm, dividing at the hiatus into an ascending and descending part. The ascending part joins with the oesophageal wall 1–2 cm above the hiatus, the descending is enmeshed with adipose
tissue cells, forming a subdiaphragmatic fat ring. This ligament becomes thinner in old age, and torn and attenuated in hiatus hernia, so that it may not be easily defined in the dissecting room or operating theatre. It may, however, be extremely important in preventing the oesophagus from sliding through the hiatus.

As a result of manometric studies (Edwards, 1961) it has been found that the abdominal pressure exerted on the stomach is +10 cm H₂O whilst the intrathoracic pressure acting on the portion of the oesophagus above the diaphragm is −5 cm H₂O. During quiet respiration, these pressures vary by a few centimetres of water, inspiration producing a rise of pressure below the diaphragm and a fall above it. This observation leads to the formulation of the mechanical theory. When a flaccid tube passes through a slit opening between two compartments at different pressures, the difference in pressure tends to evert and extrude the tube and its contents into the compartment at lower pressure. It can be argued that the oesophagus fits this description. The portion of the tube subjected to the higher pressure will tend to be extruded and everted, unless it be first closed, in which case the higher pressure will maintain the closure even more securely. In the normal subject, the oesophagus may behave as a flaccid compressible tube. Peristaltic waves empty it and approximate its walls, whilst the intrinsic sphincter keeps the walls approximated between swallows. The difference in pressure between the abdomen and thorax completes the closure mechanism, and the phrenico-oesophageal ligament prevents the oesophageal "tube" from being extruded into the thorax.

It is important to restate that the lowest 2–3 cm of the oesophagus and its "sphincter" are intra-abdominal, thus any increase in intra-abdominal pressure will be transmitted to the outside of this sphincteric segment, as well as to the stomach. The effects of the pressure at these two sites may not be identical because of the difference in surface areas over which the pressure is being exerted. However, it does seem that the only pressure which the normally situated sphincter has to withstand is slightly more than the difference between intragastric and local intra-abdominal pressures. The normal sphincter appears adequate to resist this distending force, thus keeping the mucosal surfaces of the intracrural part of the oesophagus in close apposition. If the anchorage of the oesophagus to the hiatus be intact, the hiatus of normal size and the musculature of the oesophagus normal, then no reflux will occur. If, however, the hiatus be disturbed or the intrinsic sphincter ineffective, then it is probable that reflux may occur with smaller increases in intra-gastric pressure than normal.

DISCUSSION

Several mechanisms have been suggested to explain the absence of gastro-oesophageal reflux despite a negative pleuro-peritoneal pressure gradient. These include an anatomical sphincter, a flap valve mechanism, the action of the diaphragm, mucosal valve, physiological sphincter and mechanical factors. It seems certain that no single mechanism is responsible for preventing reflux, and the latest work suggests the following theory. A watertight closure of the lower end of the oesophagus is maintained by the apposition of mucosal folds. This closure is brought about by the pressure difference between abdomen and oesophagus (on the principle of the flaccid tube), once the intrinsic musculature of the oesophagus has initiated the closure. The action of the diaphragm may be of importance when there has been an upset of the normal mechanism, for example in hiatus hernia. This subsidiary action may then replace some part of the normal mechanism.

REFERENCES


THE APPLIED ANATOMY OF VOMITING


II PORTUGUESE-SPANISH CONGRESS OF ANAESTHESIOLOGY

The II Portuguese-Spanish Congress of Anaesthesiology will be held in Lisbon from June 10 to 14, 1963.

The main subjects for discussion are as follows:
1. Correction of the metabolic acidosis due to anaesthesia.
2. Prolonged respiratory resuscitation.
3. Resuscitation of the seriously injured.
4. Symposium on halothane.

There will be sessions for presentation of free papers and films. A social programme is being arranged.

Further information can be obtained from:
SECRETARIA, RUA DO ALECRIM, 53-2º, LISBON, PORTUGAL.