OBSERVATIONS ON THE HUMAN CARDIA AT OPERATION

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SUMMARY

The pressure required to overcome resistance at the oesophagogastric junction was measured at operation in fifteen subjects and found to be 20 cm H₂O from both oesophageal and gastric aspects. Altering the angle of entry of the oesophagus to the stomach by fundal depression or occlusion did not affect these pressures, in contradistinction to the results in the cadaver (Marchand, 1955). Dividing the vagus nerves increased the resistance to intragastric pressure to 55 cm H₂O without altering the resistance to intravesophageal pressure change. A similar effect was seen with atropine given intravenously. The results support the view of a true sphincter at the cardia with a mechanism for augmenting the resistance to gastric regurgitation.

Despite considerable investigation in the past decade, the closing mechanism at the gastro-oesophageal junction is still a controversial subject. The factors controlling regurgitation of gastric contents have been studied by several different methods and various conclusions drawn. It has been suggested that the functional alterations at the lower end of the oesophagus in man are produced by:

1. a true morphological sphincter;
2. a mucosal flap valve;
3. a valve created by the angle of entry of the oesophagus into the stomach, and whose function is intimately related to the anatomical association with the diaphragm; or
4. an intrinsic sphincter.

The cardia is of special interest to the anaesthetist who is concerned with the aetiology of regurgitation and vomiting in relation to anaesthesia, and, in particular, with preventing the passage of gastric contents into the oesophagus during induction of anaesthesia.

Using transducer manometer systems Dornhorst, Harrison and Pierce (1954) suggested that in man there was a valvular arrangement at the cardia whose sole function was to prevent gastric regurgitation, and that there was no obstruction to the forward passage of the contents of the oesophagus. Using a similar technique, however, Fyke, Code and Schlegel (1956) and later Atkinson et al. (1957a) showed that the region of the cardia behaved as a true physiological sphincter.

Though Lerche (1950) concluded that there was some thickening of the smooth muscle at the lower end of the oesophagus which might represent a sphincter, the detailed anatomical studies of Lendrum (1937) and Roux (1939) did not support this. Nevertheless a sphincter mechanism has been demonstrated in the cat cardia though here, as in man, there is no histological evidence to support it (Knight, 1934; Clark and Vane, 1961). This lack of histological evidence of a sphincter has focused attention on the valvular hypothesis (O'Mullane, 1954; Sinclair, 1959) though the anatomical support for this conclusion is tenuous. Creamer (1955) considered that the mucosal folds around the cardiac orifice formed a valve. Allison (1951) suggested that the right crus of the diaphragm, which encircles the cardia, formed a muscle sling which would alter the angle of entry of the oesophagus into the stomach, thus producing a valvelike obstruction to regurgitation. Barrett (1954) supported this view and suggested that the intraluminal projection of the mucosa at this angle was pressed against the opposite wall by intragastric pressure, thus closing the lumen. Marchand's (1955) experiments seemed to confirm this by showing that altering the angle of entry of the
oesophagus into the stomach greatly affected the intragastric pressure required to overcome the resistance at the cardia. These experiments were performed in the cadaver, however, and it seemed useful to make similar observations during operations on the stomach.

**MATERIAL AND METHODS**

Observations were made in fifteen patients undergoing laparotomy; thirteen suffered from duodenal ulcer, one from cholelithiasis and one from hiatus hernia. The anaesthetic technique employed in twelve cases was that described for the procedure of vagotomy and gastroenterostomy using electrical stimulation as a means of testing complete nerve section (Burge and Vane, 1957; Riddoch and Clark, 1962). Premedication consisted of papaveretum (10–20 mg) only and anaesthesia was induced with thiopentone (50 mg/stone (709 mg/kg) body weight), followed by gallamine triethiodide (120 mg). A cuffed endotracheal tube was passed and anaesthesia maintained by ventilation with nitrous oxide and oxygen, with supplementary injections of thiopentone and gallamine. In the remaining three patients the anaesthetic technique was similar except that atropine (0.6 mg) was included in the premedication.

At operation, after opening the abdomen an occlusion clamp was placed across the pylorus. A Burge intragastric tube with an inflatable cuff was passed down the oesophagus until the tip could be felt entering the stomach. It was then withdrawn for 8 cm and the cuff inflated. A wide-bore needle was next inserted and secured in the pyloric antrum. The needle and the oesophageal tube were each connected to polythene tubing which could be joined to a water manometer as required. A side arm carried a sphygmomanometer bulb used to introduce air into the system. In this way it was possible to test the resistance of the cardia to air pressure, applied to either its oesophageal or gastric aspects. When air was introduced into the oesophagus or stomach the pressure recorded on the manometer rose until the resistance at the cardia was overcome, when it rapidly fell. The pressure attained just before this fall was described as the yield or opening pressure. The procedure is similar to that described by Greenan (1961). When pressure measurements were made, three readings from either aspect of the gastro-oesophageal junction were obtained and the mean was described as the oesophageal opening pressure (o.o.p.) or the gastric opening pressure (g.o.p.).

The resistance at the cardia was measured before and after giving the drugs, atropine, pethidine or methylamphetamine intravenously. A period of 2 minutes was allowed to elapse between giving a drug and retesting the opening pressures. The effect of vagotomy on these pressures was also studied. Measurements, comparable with those made by Marchand in the cadaver, were made after altering the relationship of the angle of entry of the oesophagus into the stomach by (1) depressing the fundus of the stomach with two large abdominal packs or (2) occluding the fundus with a clamp.

In order to demonstrate the effect on the cardia of removing the influence of the diaphragm, an experiment in the cat was performed. The cat was anaesthetized with chloralose (80 mg/kg i.v.) and the pressures required to open the cardia from either aspect were measured before and after dividing the diaphragm. The pressures were again ascertained after dividing the vagus nerves in the neck.

**RESULTS**

Table I shows the details of the patient's age, sex, diagnosis and operative procedure performed. The pressures required to make the cardia incompetent were noted in each case. The mean g.o.p. of 23 cm H$_2$O was slightly higher than the mean o.o.p. (20 cm H$_2$O) and in every case this increased resistance to intragastric pressure was found. Only in a patient with hiatus hernia was the o.o.p. found to be greater than the g.o.p. Only a small volume of air is required to be introduced into the oesophagus before incompetence occurs, though volumes of 500–1000 ml may be needed to (sufficiently) raise the intragastric pressure to a similar level producing reflux.

In three patients the effect of giving atropine was studied and in table II it can be seen that after a dose of 0.6 mg intravenously the o.o.p. remains unaltered but there is a marked increase in the resistance of the cardia to increased intragastric pressure, the mean rising from 25 to 54 cm H$_2$O. The duration of this effect was at least 30 minutes in two subjects tested. However, in three patients who had been given atropine 1 hour before operation (approximately 1½ hours before the pressure
Observations on the Human Cardia at Operation

### Table I

Opening pressures of the cardia. Age, sex, diagnosis and subsequent operation in the patients studied before atropine administration.

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>Disease</th>
<th>Operation</th>
<th>Oesophageal opening pressure (o.o.p.) (cm H₂O)</th>
<th>Gastric opening pressure (g.o.p.) (cm H₂O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>59</td>
<td>Pyloric stenosis</td>
<td>Gastroenterostomy</td>
<td>12</td>
<td>13</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>70</td>
<td>&quot;</td>
<td>&quot;</td>
<td>22</td>
<td>24</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>67</td>
<td>&quot;</td>
<td>&quot;</td>
<td>23</td>
<td>27</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>26</td>
<td>Duodenal ulcer</td>
<td>Vagotomy + gastroenterostomy</td>
<td>20</td>
<td>23</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>26</td>
<td>&quot;</td>
<td>&quot;</td>
<td>21</td>
<td>23</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>30</td>
<td>&quot;</td>
<td>&quot;</td>
<td>22</td>
<td>23</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>34</td>
<td>&quot;</td>
<td>&quot;</td>
<td>22</td>
<td>23</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>41</td>
<td>&quot;</td>
<td>&quot;</td>
<td>22</td>
<td>23</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>48</td>
<td>&quot;</td>
<td>&quot;</td>
<td>26</td>
<td>28</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>50</td>
<td>Stomal ulcer</td>
<td>Vagotomy</td>
<td>20</td>
<td>26</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>48</td>
<td>Gallstones</td>
<td>Cholecystectomy</td>
<td>19</td>
<td>21</td>
</tr>
</tbody>
</table>

Mean 20.3  23

### Table II

The effect of atropine 0.6 mg i.v. is shown on the opening pressures. There was no change in oesophageal resistance but a marked increase in the resistance to increased intragastric pressure.

<table>
<thead>
<tr>
<th>Case</th>
<th>Opening pressures before atropine (o.o.p.) (cm H₂O)</th>
<th>Opening pressures after atropine (g.o.p.) (cm H₂O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>22</td>
<td>24</td>
</tr>
<tr>
<td>7</td>
<td>22</td>
<td>23</td>
</tr>
<tr>
<td>9</td>
<td>26</td>
<td>28</td>
</tr>
</tbody>
</table>

Pressures were measured before and 10 minutes after abdominal vagotomy. Prior to vagotomy it was the practice to stimulate the vagal nerves with an electrode encircling the lower end of the oesophagus. The oesophageal tube was passed into the stomach for this purpose and after inflating the stomach with air, the demonstration of gastric contractions confirmed an intact functional nervous pathway. Following vagotomy it was found (table IV) that while there was no change in the pressure required to overcome the cardia from the oesophageal aspect, once again there was a marked increase in the pressure required to cause incompetence from the gastric aspect. Atropine given after vagotomy did not further influence the opening pressures (table IV) except in one case. In this patient (Case 10), who had an anastomotic ulcer following a Polya gastrectomy, the g.o.p. was unchanged after vagotomy but increased by 10 cm H₂O on subsequent administration of atropine.

### Table III

The effect of adding atropine to the premedication. Age, sex, diagnosis and operation in three cases to whom atropine 0.6 mg was given with papaveretum 10 mg 1 hour before operation.

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>Disease</th>
<th>Operation</th>
<th>Oesophageal opening pressure (o.o.p.) (cm H₂O)</th>
<th>Gastric opening pressure (g.o.p.) (cm H₂O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>F</td>
<td>51</td>
<td>Duodenal ulcer</td>
<td>Partial gastrectomy</td>
<td>12</td>
<td>23</td>
</tr>
<tr>
<td>13</td>
<td>M</td>
<td>43</td>
<td>Duodenal ulcer</td>
<td>Partial gastrectomy</td>
<td>13</td>
<td>27</td>
</tr>
<tr>
<td>14</td>
<td>M</td>
<td>60</td>
<td>Duodenal ulcer</td>
<td>Partial gastrectomy</td>
<td>21</td>
<td>30</td>
</tr>
</tbody>
</table>
TABLE IV

Opening pressures (cm H$_2$O) were obtained before and after vagotomy. There was a marked increase in the resistance of the cardia to increased intragastric pressure. The subsequent administration of atropine had no further effect except in Case 10. Oesophageal opening pressures were unaltered.

<table>
<thead>
<tr>
<th>Case</th>
<th>Normal opening pressures (o.o.p.) (g.o.p.)</th>
<th>After vagotomy (o.o.p.) (g.o.p.)</th>
<th>After vagotomy and atropine (o.o.p.) (g.o.p.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(cm H$_2$O)</td>
<td>(cm H$_2$O)</td>
<td>(cm H$_2$O)</td>
</tr>
<tr>
<td>5</td>
<td>16</td>
<td>23</td>
<td>20</td>
</tr>
<tr>
<td>6</td>
<td>21</td>
<td>21</td>
<td>19</td>
</tr>
<tr>
<td>8</td>
<td>22</td>
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<tr>
<td>10</td>
<td>20</td>
<td>26</td>
<td>22</td>
</tr>
<tr>
<td>11</td>
<td>19</td>
<td>21</td>
<td>18</td>
</tr>
<tr>
<td>Mean</td>
<td>20</td>
<td>23</td>
<td>20</td>
</tr>
</tbody>
</table>

TABLE V

The effect of pethidine was to cause a small increase in the gastric opening pressures, an effect which persisted. A similar effect seen after methylamphetamine lasted only a few minutes. Oesophageal opening pressures were unchanged.

<table>
<thead>
<tr>
<th>Case</th>
<th>Opening pressures (cm H$_2$O)</th>
<th>After pethidine 5 mg i.v. (o.o.p.) (g.o.p.)</th>
<th>After pethidine 10 mg i.v. (cm H$_2$O)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(o.o.p.)</td>
<td>(g.o.p.)</td>
<td>(o.o.p.)</td>
</tr>
<tr>
<td>1</td>
<td>12</td>
<td>13</td>
<td>14</td>
</tr>
<tr>
<td>3</td>
<td>23</td>
<td>27</td>
<td>16</td>
</tr>
<tr>
<td>4</td>
<td>20</td>
<td>23</td>
<td>25</td>
</tr>
<tr>
<td>5</td>
<td>16</td>
<td>23</td>
<td>18</td>
</tr>
</tbody>
</table>

Two patients were given pethidine (25-50 mg) intravenously, which caused a slight lowering of the o.o.p. and no real change in the g.o.p. (table V). Two patients who were given methylamphetamine (5-10 mg) showed an increase in the g.o.p., but this effect only lasted for a few minutes.

In two patients pressures were obtained before and after two large abdominal packs had been introduced to depress the gastric fundus, thereby altering the angle of entry of the oesophagus into the stomach (fig. 1). There was no significant change in the pressure required to force the cardia from the gastric aspect. Similarly (fig. 3) there was no change in the pressure required to produce incompetence when the fundus of the stomach was excluded by a clamp. In none of these cases was there any alteration in oesophageal opening pressures. These results should be compared with those of Marchand in the cadaver (fig. 2 (2)).

In the patient with hiatus hernia, a female aged 60 years, the position of the herniated portion of the stomach resulted in an abnormally acute angle of entry of the oesophagus (fig. 4). Here, however, the normal relation of pressures between o.o.p. and g.o.p. was reversed but returned to normal after restoring the anatomical relationships. The findings suggested that mechanical factors in this instance were important in reducing the degree of competence of the cardia. Though not strictly comparable with Marchand's experiment in the cadaver (fig. 2 (3)), where the left dome of the diaphragm was removed, nevertheless the results were the opposite of those said to occur when the angle of entry of the oesophagus to the stomach is made more acute.

The importance of the diaphragm in the mechanism of the human cardia is difficult to establish by experiment. Figure 5 shows an experiment in the cat, which has a gastro-intestinal system whose function closely resembles that in man. Dividing the diaphragm has no effect on the resistance to increased intragastric pressure. It can also be seen that vagotomy greatly increases the resistance, as was found to occur in the patients at laparotomy.
The gastric opening pressures measured in two patients (A) before and (B) after depressing the fundus of the stomach with two large abdominal packs (P). There was no significant change in the pressure required to open the cardia; the pressures were 24 and 25 cm H$_2$O before and 25 and 27 cm H$_2$O respectively after fundal depression. Oesophageal opening pressures were also unaffected.

C = clamp occluding pylorus.

Marchand's experiments in the cadaver (1955). Intra-gastric pressures were raised through the cannula (C) and the pressures required to open the cardia with alteration of the oesophago-gastric angle produced by various manoeuvres were noted.

D indicates diaphragm.

(1) Normal anatomical position. Pressure required to open cardia, 28 cm H$_2$O.
(2) Abolition of the normal oesophago-gastric angle by a clamp excluding the fundus of the stomach. Pressure now 9 cm H$_2$O.
(3) Exaggerate the oesophago-gastric angle by removing the left leaf of the diaphragm. Pressure required 42 cm H$_2$O.
(4) Both leaves of the diaphragm are removed and the cardia opens with a pressure of only 3 cm H$_2$O.

Adapted from Marchand (1955) by kind permission of the author and the editor of the British Journal of Surgery.

The gastric opening pressures in two patients (A) before and (B) after occluding the fundus of the stomach with a clamp (C$_2$). These pressures were 22 and 23 cm H$_2$O before and 22 and 25 cm H$_2$O respectively after occlusion. No significant change was found and the oesophageal opening pressures were unaffected.

N = wide bore needle.
The opening pressures of the cardia in a patient with hiatus hernia (H).

(A) Before repair the normal o.o.p.:g.o.p. ratio was reversed (25:19 cm H₂O) but neither was abnormally low.
(B) After repair the ratio returned to normal (20:30 cm H₂O), the gastric opening pressure being significantly increased.

The effect on intragastric pressure of dividing the diaphragm, and of vagotomy, in a chloralosed cat.

(A) Normal.
(B) There is no change in the pressure required to cause incompetence of the cardia after dividing the diaphragm.
(C) and (D) There was increased resistance at the cardia to intragastric pressure change after (C) right vagotomy, (D) left vagotomy.

Oesophageal opening pressures were unaltered throughout.
As in man, there was no alteration in the pressure required to overcome the resistance from the oesophageal aspect. In these animals when the effect of the diaphragm in augmenting sphincter resistance was looked for, it was found in two instances that dividing the diaphragm lowered the resistance to gastric reflux (Clark and Vane, 1961).

DISCUSSION

Several workers have measured the resistance of the cardia in man. Mikulicz (1903) introduced fluid into the oesophagus and measured the rise in pressure before it passed into the stomach. Using water, a pressure of 2-6 cm H₂O was found, but beer or soda water achieved a pressure of 20-40 cm before the cardia yielded. Hurst (1934) stated that the cardia could support a column of water 8 inches high while Jutras, Levrier and Longtin (1949), from radiological studies, suggested the cardia could resist a force of 5 grammes from the oesophageal aspect.

Dornhorst, Harrison and Pierce (1957) calculated the yield or opening pressure of the cardia to be 5 cm H₂O but pointed out that this was affected by the normal pressure gradient between the pleura and the peritoneum. This gradient, which may be as high as +15 cm H₂O, was considered in the studies of Fleshier and associates (1958) which showed that the cardia could resist a mean pressure of 12.75 mm Hg (17.4 cm H₂O) from the oesophageal aspect. This, however, was only 8.6 mm Hg (11.4 cm H₂O) above the mean oesophago-gastric gradient but it was emphasized that in every case when the oesophagus emptied this was initiated by peristalsis.

In our experiments the cardia resisted a mean pressure of 20 cm H₂O. The normal thoraco-abdominal pressure gradient was altered since the abdomen was open and, therefore, the oesophagus was influenced only by the negative intrathoracic pressure. We did not, however, observe any tendency for the cardia to empty during expiration.

The pressures required to make the cardia incompetent from the gastric aspect are perhaps of more practical importance. Marchand (1955) found a mean of 28 cm H₂O in the cadaver, but Atkinson and Summerling (1959), who repeated these experiments, found that a pressure of only 5 cm H₂O was required to show incompetence at the gastro-oesophageal junction. They pointed out that after death the normal angle of entry of the oesophagus into the stomach is lost and showed that if this is restored by manipulation then the mean pressure required to force fluid past the cardia is 23 cm H₂O. This pressure is similar to that found in our patients at operation, and O'Mullane (1954) also found that the cardia resisted a force of 10–19 cm H₂O applied from the gastric aspect. Recently Greenan (1961), in experiments similar to ours, found a resistance of 26.7 mm Hg (36.3 cm H₂O). The similarity between results in the patient at operation and in the cadaver, where the angle of entry is restored, perhaps suggests that mechanical factors are of importance in the competence of the cardia. Nevertheless, the fact that the cardia can resist pressure change from either aspect favours the existence of a sphincter mechanism. The fact that the sphincter is, apparently, more powerful in resisting intragastric pressure change does not detract from this suggestion since the mechanism producing incompetence may be different. It is known that the cardia opens when the upper oesophagus is distended (Atkinson et al., 1957b) and does so reflexly before the simultaneously initiated peristaltic wave has reached the lower oesophagus. In achalasia when oesophageal reflexes are deranged or lost there is evidence that the cardia is normal (Olsen et al., 1957). Failure of the oesophagus to empty in this condition may be due to loss of reflex control although the oesophagus would still yield when it had attained its uninhibited yield or opening pressure.

The cardia can become incompetent from reflexes initiated in the stomach. This is supported by the fact that the gastric opening pressure is increased after vagotomy or by giving atropine. It may also perhaps explain the results noted by Greenan (1961) who found that depressing the fundus by hand reduced the gastric yield pressure from 26.7 to 7.5 mm Hg (36.3 to 10.1 cm H₂O). Barrett (1952) has also made this observation. Greenan considered that his results supported the findings of Marchand (1955) in the cadaver but our experiments with occluding the fundus by a clamp, as in Marchand's procedure, show that the experiments in the cadaver do not obtain in patients under anaesthesia. Similarly, depressing the gastric fundus with abdominal packs did not affect the gastric opening pressures.
The most striking results obtained were those after vagotomy or after giving atropine. The increased resistance at the cardia, which is of unknown duration, might be explained by loss of reflex opening or possibly as a result of removing the inhibitory effect of the vagus. The effect has been seen in animals (Clark and Vane, 1961) where it has been shown that stimulation of the vagus at high frequency causes relaxation of the cardia. It seems unlikely that this could be accounted for by any changes in the angle of entry of the oesophagus into the stomach, and some other active mechanism is responsible. Since no change was found in the ability of the cardia to resist pressure change from the oesophageal aspect, attention must be directed to structures other than the actual sphincter muscle.

Nauta (1956) drew attention to the remarkable folds of gastric mucosa which surround the cardia and Botha (1958a, b) suggested that these might exert their influence in the mechanism of the cardia. The mucosal rosette formed at the cardia is supported by the muscularis mucosa and it moves independently of the outer muscular coat. Hughes (1957) showed that in the human foetus the muscularis mucosa had only longitudinally disposed fibres. Creamer (1955) suggested that the muscularis was thicker in the region of the cardia and Botha (1958b) concluded that they might form a sort of plug to the cardia from the gastric aspect. It is possible that the resistance to reflux after atropine or vagotomy may be due to some influence exerted on this component of the sphincter. If the mucosal folds are dependent upon the underlying mucosa for their movement and if the movement is paralyzed by atropine or vagotomy, then by the mere bulk of tissue the folds could effectively augment resistance to reflux without interfering with oesophago-gastric flow.

The part which the diaphragm plays in preventing regurgitation is difficult to assess but Atkinson and associates (1957b) have shown that the cardia may be competent even when it is dissected into the thorax. In the cat the cardia functions as a sphincter when the diaphragm has been removed (Shenk and Frederikson, 1958) though in some animals it is capable of augmenting the resistance to gastric regurgitation, and it seems probable that this function exists in man.

These results show that the mechanism at the cardia cannot be regarded as a simple valve. The evidence favours a sphincter whose function in preventing gastric regurgitation is augmented by unknown factors. There is a factor concerned with vagal innervation, for when vagal tone is abolished resistance is increased, at least temporarily. This active nervous component is clearly of importance to the anaesthetist as are the reflexes associated with regurgitation. Further studies of regurgitation and the influence of drugs on the cardia are indicated. The feature which is perhaps of most practical importance is the effect of atropine in greatly increasing the resistance at the cardia.

ACKNOWLEDGMENTS

We are grateful to Mr. N. J. Logie, Consultant Surgeon, for allowing us to make these observations on patients in his care. We record our indebtedness to Dr. H. Thomson, Dr. J. McClay and Dr. W. Leishman who took part in recording some of the observations.

REFERENCES


OBSERVATIONS ON THE HUMAN CARDIA AT OPERATION


La pression nécessaire pour surmonter la résistance à la jonction oesophago-gastrique fut mesurée au cours de l'intervention chez 15 sujets et fut de 20 cm H₂O aussi bien du côté oesophagien que du côté gastrique. La modification de l'angle d'entrée de l'oesophage dans l'estomac en déprimant le fond gastrique ou par l'occlusion n'affecta pas cette pression, à l'opposé des résultats obtenus sur le cadavre (Marchand, 1955). La section du vague augmenta la résistance à la pression intragastrique à 55 cm H₂O sans modifier la résistance au changement de la pression intra-oesophagienne. Un effet semblable fut observé après l'administration intraveineuse d'atropine. Ces résultats appuient l'idée d'un véritable sphincter au cardia avec un mécanisme vagal pour augmenter la résistance à la régurgitation gastrique.

ZUSAMMENFASSUNG