J. G. TON - REFLUX OESOPHAGITIS

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ACADEMISCH PROEFSCHRIFT TER VERKRIJGING VAN DE GRAAD VAN DOCTOR IN DE GENEESKUNDE AAN DE UNIVERSITEIT VAN AMSTERDAM, OP GEZAG VAN DE RECTOR MAGNIFICUS, DR M. W. WOERDEMAN, HOOGLERAAR IN DE FACULTEIT DER GENEESKUNDE, IN HET OPENBAAR TE VERDEDIGEN IN DE AULA DER UNIVERSITEIT OP DONDERDAG 11 DECEMBER 1958 DES NAMIDDAGS TE 4 UUR

door

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geboren te

TINDJOWAN

Promotor:

Professor Doctor I. BOEREMA

Dit proefschrift werd bewerkt in de Chirurgische Universiteitskliniek van het Wilhelmina Gasthuis te Amsterdam

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VOORWOORD

Bij het verschijnen van dit proefschrift is het mij een behoefte mijn erkentelijkheid te betuigen aan allen die mijn opleiding tot chirurg hebben mogelijk gemaakt.

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ACKNOWLEDGEMENT

This study is based on 60 case histories, concerning 26 males and 34 females, of the Surgical Clinic, University of Amsterdam, (Head: Prof. I. Boerema) and most of the literature concerning the subject.
The X-ray examinations were usually performed in the Roentgen laboratory of the Surgical Clinic. Some X-rays and most of the experiments were done in the Central Roentgen Laboratory, University of Amsterdam (Head: Prof. B. G. Ziedses des Plantes).
All oesophagoscopies were performed in the E.N.T. Clinic, University of Amsterdam (Head: Prof. L. B. W. Jongkees).
Prior to the experiments, the oesophagus and cardia of two mongrel dogs were marked with metal pellets. This was done in the Laboratory for Experimental Surgery, University of Amsterdam (Head: Prof. I. Boerema).
A few drawings were done by Dr. J. H. Piers, the schematic drawings, concerning the cardia, as well as the clay models are by the author. All photography is by Mr. J. Mos.
One photograph of the perforation of an oesophageal ulcer into the aorta was kindly forwarded to us by the head of the Pathological Department of the Onze Lieve Vrouwe Gasthuis. The other photograph of perforation of an oesophageal ulcer into the left auricle of the heart was provided by the head of the Pathological Department of the University of Amsterdam, Prof. H. T. Deelman.

BOOK I

INTRODUCTION

"La lecture des travaux sur l'ulcère de l'oesophage laisse une sensation de chaos".

NEMOURS-AUGUSTE.

Chaotic indeed seems the literature concerning peptic oesophagitis.
Condensed, the main problem appears, however, to center around the behaviour of the cardia of the stomach.
About the cardia, there exists no unanimous agreement about such a basic concept as an intrinsic sphincter.
In our opinion the presence of an intrinsic cardiac sphincter can no longer be denied.
The ancient concept of the cardiac sphincter, composed of the so called collar fibres, we have revived and we feel it satisfactorily explains all the different aspects of the behaviour of the human cardia, which no other hitherto published theory could do.
QUINCKE (1879) was the first to recognize that reflux of acid gastric juice provokes the oesophageal ulcerations. It has since repeatedly been proved by experimental and clinical evidence.
It will be found that the terms "peptic oesophagitis", "reflux oesophagitis" and "peptic ulcer of the oesophagus" are used indiscriminately as we consider these to be only different aspects of the same disease.
In this treatise we endeavour to solve some mysteries of the disease, to determine which simple procedures offer a permanent cure of the disease and which operations on the oesophagus and cardia of the stomach are not complicated by reflux oesophagitis.

Chapter I

SYMPTOMS OF REFLUX OESOPHAGITIS[1])

Pain, dysphagia, haemorrhage and regurgitation constitute the characteristic tetrad. The onset of pain is related to posture.

PAIN

The tetrad of pain, dysphagia, haemorrhage and regurgitation is more or less characteristic of the disease. Of these pain is the predominant complaint, and at first often the only one. It varies greatly among individuals. At first a slight discomfort, a fullness in the abdomen, a wind around the heart are felt, especially after meals. In time the patient feels actual pain in the epigastric region or behind the sternum usually after the hot meal of the day, which lasts from a few minutes to half an hour. This pain is aggravated by lying down, stooping or lifting weights. It may become progressively worse and in rare instances unbearable.
Lasting briefly at first, it soon extends until it lasts the greater part of the day. It may become continuous. It soon becomes apparent that there is a definite relation between the onset of pain and the horizontal position of the patient. Lying down or bending forwards provokes pain. This may cause patients to wake up at night. A retrosternal burning or pressing sensation ascending behind the sternum, is experienced. This may gradually change to a boring pain felt deep in the chest. It may radiate to the back between the shoulder blades, the jaw, the neck, behind one or both ears, down one or both arms and around the right or left costal arch. The pain is very rarely referred to the lower abdomen.
At first it is relieved immediately by alkalies, later larger doses are required. Ultimately it cannot be relieved altogether. Often pain commences while actually eating or drinking.
Especially hot or very cold drinks and spicy food bring on pain. Milk, generally, passes without discomfort. Sometimes this pain becomes so severe that any attempt at swallowing whatsoever induces it.
Apart from this, patients complain of heartburn, "a burning hot pain that rises from the upper chest into the throat and may spread into

[1]) 7, 8, 26, 40, 45, 53, 88, 89, 95, 130, 137, 149, 168, 203, 224, 227, 234, 236, 239, 240, 247, 255, 286, 294, 466, 505.

the jaws and mouth and be accompanied by flushing of the face and watering of the eyes. It feels as if some acrid fluid is being forced up the gullet and the patient swallows repeatedly to keep this down". (HARMAN, 1952).

Heartburn and the above mentioned pain, localized lower in the chest, occur separately, although both are related to change in posture.

DYSPHAGIA

Dysphagia is probably a late manifestation in the natural history of the disease. Most patients with this symptom look back on many years duration of various complaints. In some cases previous symptoms may be absent, however. The onset of dysphagia may be quite sudden. One day, without warning, food becomes stuck in the lower chest. By bending backwards, inhaling deeply, drinking or repeated swallowing this sensation may be relieved. Otherwise relief may be obtained by vomiting. At first the degree of dysphagia is extremely variable, concerning mainly solids. Intermissions are frequent. It may even happen that on the same day one meal passes and the next is obstructed. Emotional disturbances tend to increase the obstruction. (93, 220). In the majority of patients periods of relative or complete freedom alternate with periods of total obstruction. As a rule dysphagia gradually increases and becomes permanent. Many patients form the habit of chewing more thoroughly, giving preference to semi-solids with increasing dysphagia.

Only in severe cases this may terminate in permanent total obstruction. The relatively easy passage of food through a severe stricture is often remarkable. For example the stricture on illustration no. XI proved only accessible to a matchstick. According to the patient, however, a 60-year-old male (H.M. 14788) the passage of food was only slightly impeded and mainly concerned solids, besides which his condition was excellent.

HAEMORRHAGE

Manifest bleeding is a common feature of the disease and usually it is recurrent. It is thought to occur in 10% to 50% of all cases. Additional symptoms are usually present although definite other complaints may be lacking. Haemorrhage as a sole symptom of the disease is rather uncommon. When it occurs, it may be difficult to trace the source of the bleeding. Upper gastro-intestinal haemorrhage due to oesophagitis without other symptoms being present is infrequent.

In the series of 673 patients, reported by JONES (1947), for example, only two cases were found.

Haemorrhage can be expected at any time in the course of the disease and at any age, e.g. it may be present at birth (371, 435, 449, 454). It varies from slight oozing to catastrophical haemorrhage. In 1933, NIELSEN reported that out of 71 patients, 19 died of haemorrhage. Fatal outcome is rare nowadays.

The aspect of the vomit, in the case of haematemesis, may be blood-

tinged, bright red or brownish. In the latter case it may appear as "coffeegrounds". Although haemorrhage usually becomes evident through haematemesis, it may become manifest by melaena alone.

REGURGITATION and VOMITING

Regurgitation and vomiting are the main characteristics of the disease in infancy and usually the principal reason for the parents to seek medical advice (23, 94, 149, 168, 240, 467). These symptoms are invariably present. They generally date from birth but may commence at the introduction of solid food. In exceptional cases they begin at a later date.

Characteristically they occur during the meal, making it a cumbersome affair every time. The frequency and the amount vary greatly at different times and in different patients. Although regurgitation, as distinct from vomiting, is not preceded by straining, contraction of the abdominal muscles and nausea, it is hardly feasable to differentiate between these two in children.

In adults, regurgitation, by which is meant an unexpected backflow of stomach contents into the mouth, is not common (282, 349). It may be provoked by change in posture although it usually occurs at meal-times. Vomiting occurs when obstruction in the oesophagus becomes severe. Some patients however induce vomiting in order to relieve the cramp-like, constricting pain in the lower chest which accompanies it. They may make a habit of this, although no actual stricture is present.

ADDITIONAL SYMPTOMS

Additional symptoms which may contribute to the clinical picture, are belching, persistent hiccoughing, loss of weight or inability to gain weight, constipation, flatulence and salivation.

Frequent belching is a common and troublesome symptom. On the other hand a rare feature may be the inability to belch at all, which results in the so-called "aerogastric bloquée" (45, 137, 239). In this situation an abundance of air is captivated in the stomach, causing severe pain, which is aggravated by every swallow.

Recurring hiccoughing, although not common, may last from a few hours to a week at a time (466, 1 case). Attacks rarely last more than a week, however.

Loss of weight may be considerable in adults, although starvation has become rare. Children with this disease are generally underweight and undersized (94, 104, 149, 168).

Constipation is usually prominent in infants and children, but less so in adults.

Flatulence and salivation are the least troublesome of all symptoms.

On page 18 a tabulation of the incidence of the various symptoms in our series and in extensive series, reported by other authors, is found.

From these symptoms alone, oesophagitis cannot be concluded because many other oesophageal diseases pose a similar syndrome. The predominant symptom of pain, for instance, appears to bear little relation to

the presence of oesophagitis. Of 61 cases with this symptom examined by HARMAN (1953), only 25 suffered from oesophagitis. On the other hand "it is common to see patients, who have oesophagitis the presence of which has been proved by endoscopy, who have no pain" (BARRETT 1950). "Il n'existe pour la brûlure aucune correspondence biologique. Il en est de même du pyrosis" (LAMBLING 1951).

It has been shown in experiments by JONES (1925), POLAND (1931), CHAPMAN (1949), WERBELOFF (1953) and MARCHAND (1955) that there is probably no specific irritant which causes the retrosternal pain. Rapid distension of the lower gullet or the cardia may produce it (KRAMER 1955), but the same sensation was recorded by rapid distension of the duodenum or the biliary tract. Transcardial reflux of gastric contents, which is considered the chief cause of the disease (chapter VIII) naturally distends the lower gullet and may thus cause pain (408, 409). But although transcardial reflux is definitely abnormal in adults, it may occur without any symptoms (283, 408, 409). Another explanation is sought in the acidity of the backflowing gastric juice. Indeed when a weak solution of hydrochloric acid comes into contact with oesophageal ulcers, it causes pain, while in normal subjects no pain is experienced (BARRETT 1950).

NEMOURS-AUGUSTE (1951, 1953) concluded after an extensive study on the subject that there exists:

1. retrosternal pain without reflux
2. pain without oesophagitis
3. pain with reflux
4. pain with reflux without oesophagitis, and finally
5. pain with reflux and with oesophagitis.

From all these phenomena, the conclusion might be drawn, that the vagal nerves, which are the only sensory conductors of the gullet and its corresponding cerebral nuclei, do not particularly differentiate between the various stimuli received (16, 241, 348).

The other symptoms as dysphagia and regurgitation are common oesophageal symptoms. Together with pain they constitute the oesophageal triad, as proposed by THOREK. The addition of haemorrhage may, except for the disease concerned, be expected in carcinoma of the oesophagus or the cardia and in acute inflammation by other causes.

In the following tabulation the incidence of symptoms in our series is compared to that of the series of SCHMIDT (1954) and CARVER (1954).

	OUR SERIES 60 cases	CARVER (1954) 130 cases	SCHMIDT (1954) 170 cases
PAIN		91	
Retrosternal	37		72
Pyrosis	31		19
Epigastric	20		54
Pain at swallow	16		11
DYSPHAGIA	46	100	101
HAEMORRHAGE		13	
Haematemesis	20		38
Melaena	13		20
REGURGITATION	25		37
		52	
VOMITING	35		27
LOSS OF WEIGHT	30	—	10
BELCHING	22	—	13
CONSTIPATION	10	—	—
HICCOUGH	5	—	1
SALIVATION	3	—	—
FLATULENCE	—	—	13

Chapter II

EXAMINATION[1])

Diagnosis depends on roentgenology and oesophagoscopy.

Constant X-ray features are a short oesophagus, ulceration of the lower gullet and reflux.

"The role of oesophagoscopy cannot be overemphasized".
CROSS (1956)

Physical examination generally contributes little or nothing to the diagnosis. Physical signs are few, mostly comprising upper abdominal tenderness on palpation. In an acute or subacute case, the patient may run a temperature, which has been emphasized lately by HILLEMAND (1954) and LORTAT-JACOB (1951), but was already mentioned by ROTGANS in 1907.

When dysphagia is the chief complaint, examination with a soft rubber tube can be a valuable aid in the diagnosis (38). When introduced into the lower gullet, an obstruction may be encountered, which may easily yield to slight pressure, if due to muscular spasm. In typical cases the tube is gripped by spasm, sometimes rendering extraction extremely difficult (168, 400). This may be of great importance in the diagnosis as it is occasionally the only positive finding; oesophagoscopy and roentgenology being essentially negative at the time.

According to BARRETT (1950), heartburn can be initiated by drinking a 0.3% solution of hydrochloric acid, if it is due to oesophagitis. Normally this HCl solution passes the gullet without undue sensation.

Laboratory examination may show anaemia, sometimes of serious degree and occult blood in the stools (37, 95, 152, 234, 467, 476). An increased sedimentation rate of erythrocytes may occur in acute phases. Roentgenology and oesophagoscopy are essential diagnostic procedures (442).

ROENTGENOLOGY

X-ray examination usually consists of a combination of fluoroscopy and radiography in antero-posterior, right and left anterior oblique

[1]) 5, 6, 8, 23, 26, 41, 42, 47, 49, 54, 79, 80, 81, 94, 95, 103, 116, 119, 121, 123, 139, 140, 149, 164, 168, 172, 174, 177, 178, 223, 224, 225, 227, 240, 255, 270, 271, 286, 290, 296, 324, 327, 328, 329, 331, 346, 349, 350, 354, 365, 366, 405, 421, 442, 445, 463, 464, 467, 500, 504, 507, 508.

and lateral positions. Although generally done in the upright position, examination in horizontal and Trendelenburg positions is essential in this disease. In special cases bending forwards may be employed and the Valsalva test may add useful information.

For a contrast medium a watery, chocolate-flavoured suspension of barium sulphate is usually preferred. Occasionally it is advisable to repeat the examination with thicker barium paste.

ULCER NICHE

An ulcer niche is primarily sought for, but can only be demonstrated in a minority of cases. In ALLISON's (1948) series of 74 cases of oesophageal ulcer, only 5 niches were roentgenologically demonstrable. WOLF (1953) found 6 out of 29.

A higher incidence of niches is occasionally reported, e.g. THOMSEN e.a. (1949) found 21 niches out of 27 by means of X-ray alone.

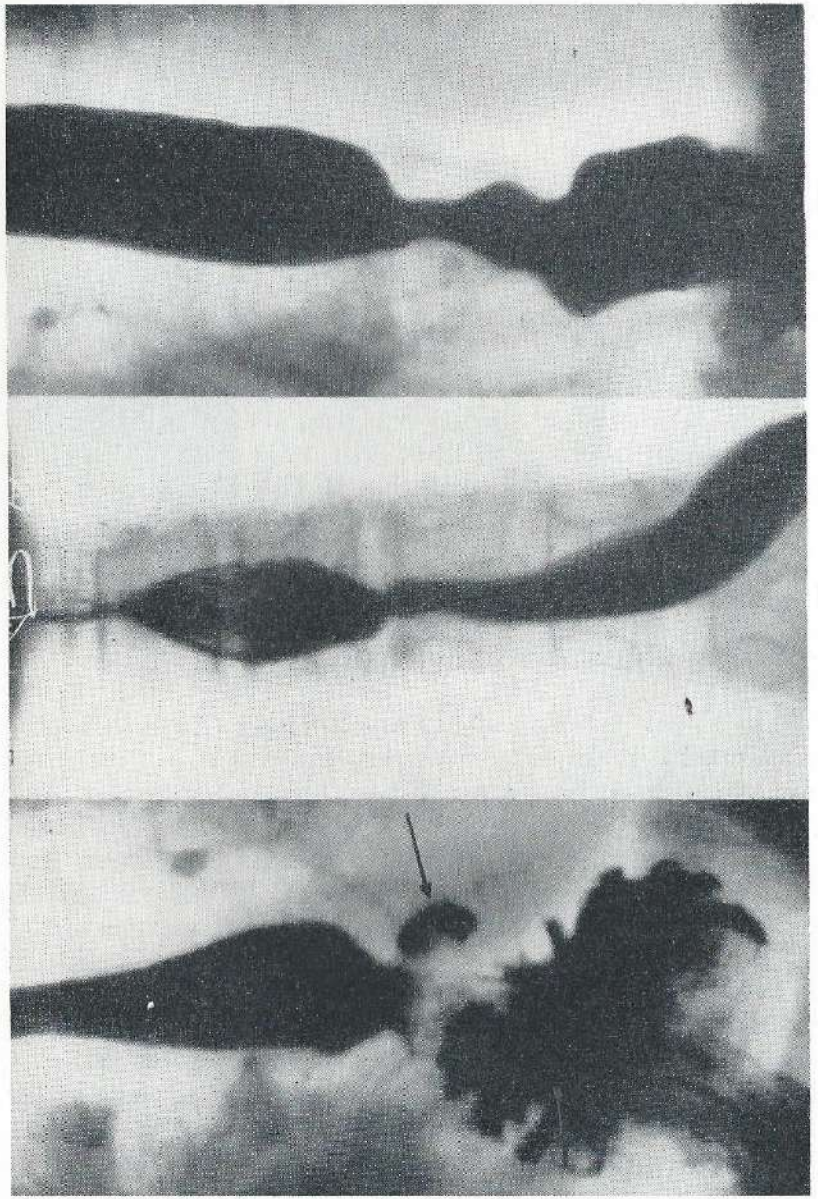
The main cause of this inability to demonstrate the oesophageal ulcer lies in the fact that manual compression of the oesophagus in the thoracic cage during fluoroscopy, as done in the demonstration of a duodenal ulcer, is impossible (286). A second factor which hides the presence of an ulcer, is spasm of the gullet which prevents the contrast medium from entering the cavity of the ulcer. Thirdly, in ulcerative oesophagitis only flat and superficial lesions are commonly present, which can seldom be visualized.

BRUNETTI (1927) is credited with the first description of the roentgen picture of the niche (172). FRIEDENWALD (1929) considered the right postero-lateral wall to be most frequently involved. BROMBART (1952) found the antero-lateral wall to be the usual locality. In reviewing the literature however, one finds that these ulcers can be located on both sides of the lower oesophagus; there is probably no predilection. The niche usually has a wide communication with the gullet and may contain a small bubble of air (80, 172, 255). Its edges may be oedematous and radiating mucosal folds may be seen around the ulcer (80).

It is generally impossible to ascertain, when a niche is present, whether it is actually situated in the oesophagus or in the herniated stomach pouch. See illustration I.

As previously stated, it is often impossible to demonstrate a niche but other changes in the mucosal pattern might be seen on careful examination. They are frequently inconspicious. It will be found that the finely plicated longitudinal mucosal folds are replaced by a blotched pattern which interrupts the transition to gastric mucosa. The contours of this image may be finely serrated. Illustration II.

On the other hand the contours may be quite irregular and have the appearance of carcinoma (8, 45, 80) Illustration III demonstrates this point. It concerns a 66-year-old man, who was suspected of having an oesophageal cancer. By subsequent resection he was proved to suffer



III *Irregular contours, simulating cancer of the oesophagus*

II *Serrated contours*

I *Ulcer niche*

from oesophagitis alone. Illustration VIII. Although familiar with the disease, we have been similarly mistaken 4 times. This pseudo-tumerous aspect is by no means rare.

In order to obtain more distinct visualization WOLF (1953) and others suggested introducing air and barium meal simultaneously into the gullet, thus procuring a double contrast roentgenogram (41).

ABNORMAL MOTILITY

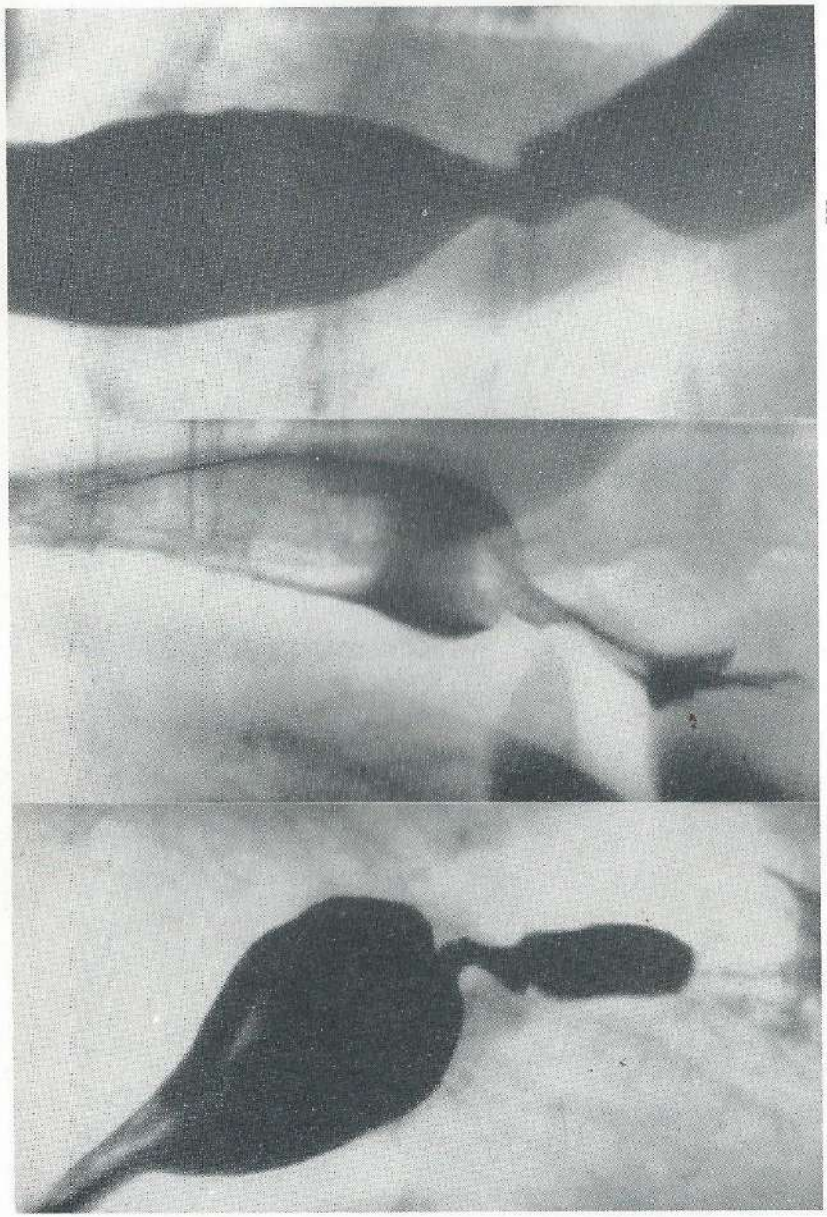
Another roentgenological characteristic is that an abnormal motility is generally present. A change in motility is first seen in a slowing down of the propulsion of the contrast medium in the lower oesophagus. In time this may increase until most or even all of the barium is retained in the lower gullet. Numerous peristaltic waves are seen above this area, evidently attempting to force the contrast medium down. The terminal end in this instance may take a rounded shape, giving the impression of a pouch. Illustration IV. It concerns a 77-year-old female (H.M. 11883) with intermittant dysphagia. It is thought that muscular spasm causes this phenomenon and there is no doubt that spasm is often the sole cause of the obstruction (467). This can be verified by instructing the patient to inhale a few drops of amylnitrite. After a few moments the spasm then slackens and on fluoroscopy there appears an abrupt passage of the contrast medium (119, 262, 463). Illustration no. V is an X-ray exposed on this moment. It concerns a 48-year-old woman (H.M. 9100) who was known to suffer from oesophagitis for 10 years. She was admitted with a sudden total obstruction, which spontaneously disappeared in a few days' time.

STRICTURE

Where there is a funnel-like stenosis with the wide end facing orally, it is usually a stricture caused by fibrosis, although a spastic factor is often present. These strictures generally have a smooth lining and a symmetrical appearance. Illustration VI. No distensibility of the stenosis can be demonstrated if it is due to stricture and no peristaltic movements are seen in this area. Sometimes there is no gradual constriction of the lumen but an abrupt weblike stenosis, e.g. illustration VIII shows such a weblike stenosis, which was proved by resection to be a rigid stricture. The stenosis is generally short but it may occasionally involve the greater part of the oesophagus. This was first described by BROWN-KELLY (1939) as "ascending fibrosis". Illustration VII.

ALLISON (1948) pointed out that mistakes are possible in estimating the length of the stenosis. If a narrow one lets through only a trickle of barium, the post-stenotic region is not filled to capacity, giving the illusion of a long stricture. By means of the TRENDELENBURG position, distention of the post-stenotic area can be achieved, revealing the true length of the stricture. It usually does not exceed 1,5 cm in length, although longer strictures are not rare.

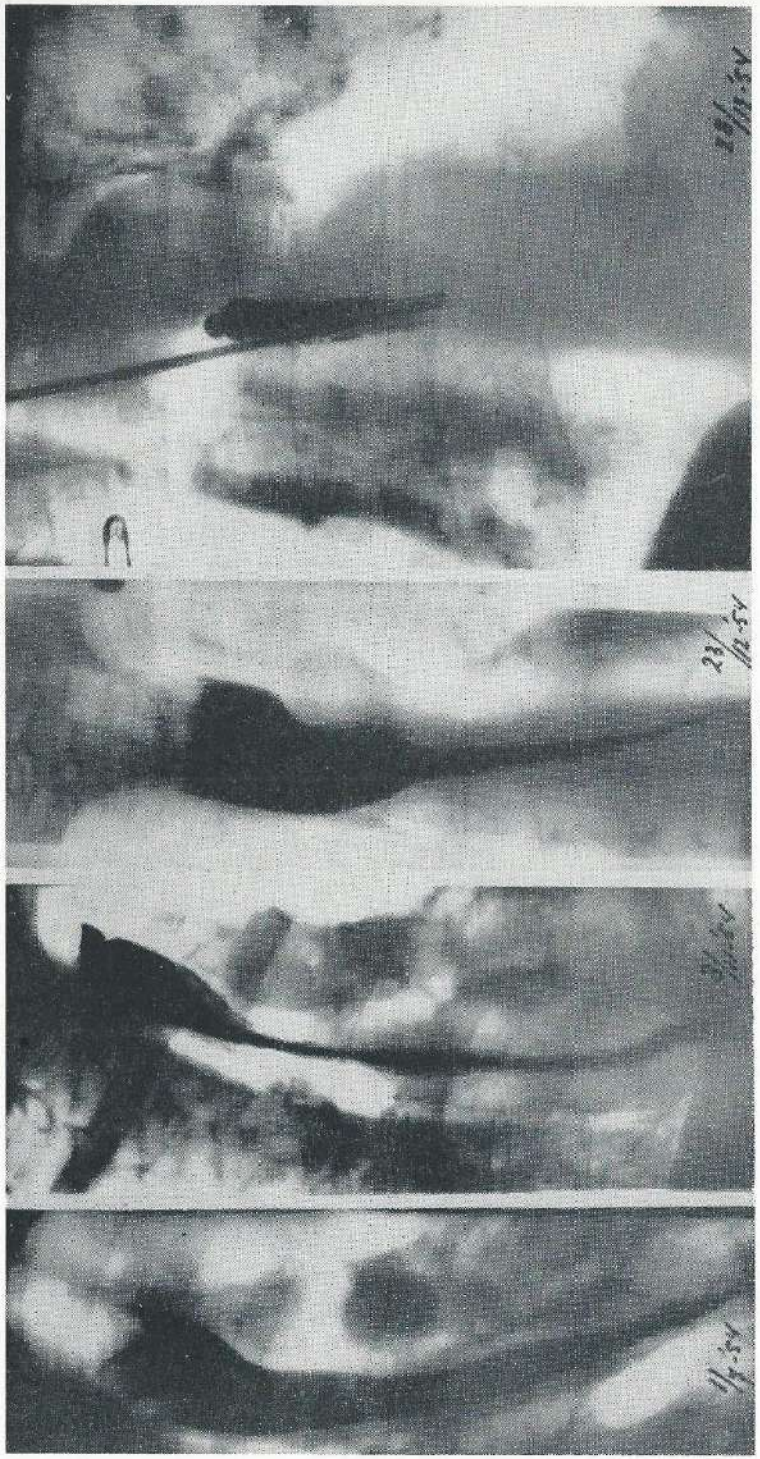
As the lower gullet is always involved, one would expect these strictures to occur in the diaphragmatic region. However, a peculiar



VI *Symmetrical and smoothly lined stricture of the oesophagus*

V *Spasm slackens, following inhalation of amylnitrite*

IV *Spasm of the gullet*



VII
"Ascending fibrosis" of the oesophagus

feature of the disease is that strictures, as well as ulcerations of the lower gullet, are generally located above the diaphragm, implying the presence of a short oesophagus.

Below such a stenosis, the rugose appearance of gastric mucosa should be apparent. It may be necessary, however, to examine the patient in Trendelenburg position, hereby filling this area with barium suspension.

During fluoroscopy when viewing the passage of the contrast medium through the stricture, the patient may complain of a retrosternal burning sensation, identical with the pain for which medical advice was sought (286, 464). This is a valuable sign, which confirms that this pathology is indeed the cause of the patient's pain.

As a rule little or no dilatation is found above the stenosis (8, 286). Generally hyperactive peristaltic movements of the oesophagus are evident. Antiperistalsis in this area is confined to the stomach as the oesophagus is thought to be incapable of this feat (117).

Occasionally X-rays are obtained showing an extraordinary picture of a corkscrew oesophagus, produced by ringlike spasms, alternating with bulging areas (45, 80, 119, 224, 251, 290, 382, 427, 478). This phenomenon, which in a lesser degree is known as "curling", "rippling" or "tertiary contractions" of the oesophagus, at first occurs periodically, but may later persist. Illustration IX.

REFLUX [1])

Apart from these signs, reflux of stomach contents into the gullet is frequently seen, alternating with respiratory movements. It is sometimes necessary to make use of the Trendelenburg position to determine this or the Valsalva test. If necessary, manual compression of the abdomen may be employed in addition. If reflux cannot be demonstrated in this way, it may be brought about by bending forwards.

In either one of the positions the gullet is abruptly filled from below. This often involves most of the oesophagus and is followed immediately by a peristaltic wave, commencing high up in the chest, which forces the barium meal back into the stomach. After a moment the cycle is repeated, repetitions occurring at an even rate as long as the patient remains in this position. Some patients already show reflux when still in the upright position. When these cases are examined in the horizontal position, the oesophagus is seen trying in vain to expell its contents by waves of peristalsis.

In many cases it is difficult to demonstrate reflux. In such cases BROMBART (1952) proposed "siphonage". After the stomach has partially been filled with barium, the patient is instructed to take a drink of water in the horizontal position. It may then be possible to demonstrate reflux.

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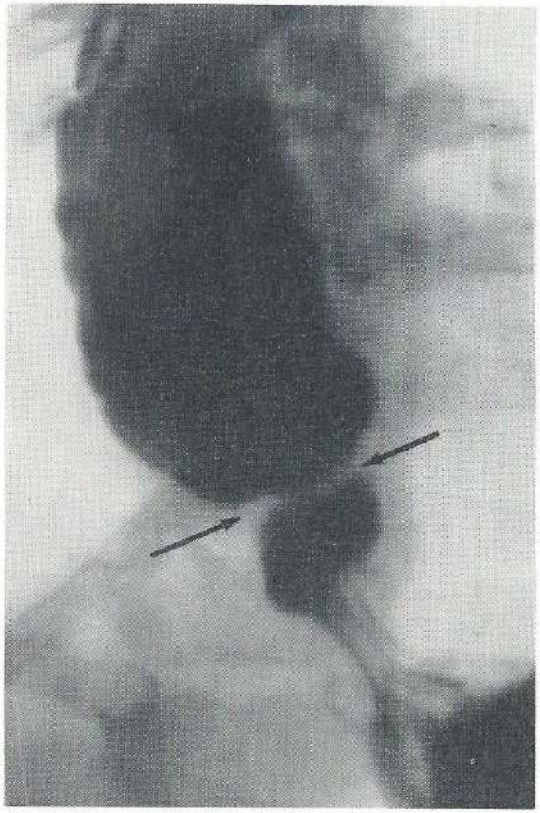
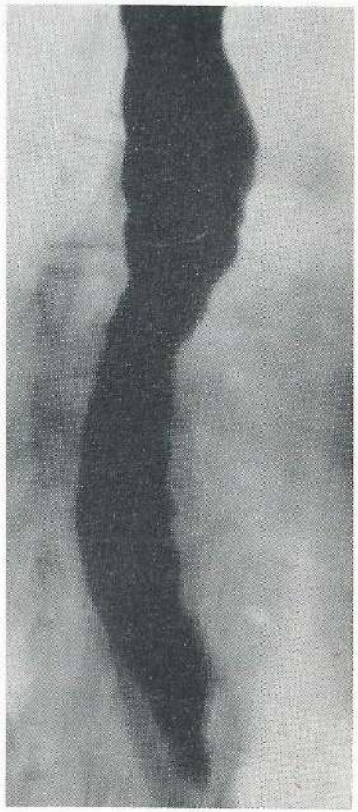
1) 5, 23, 227, 283, 405, 408, 409.

Although X-ray examination is of great importance, patients may be encountered, suffering from oesophagitis proved by other means, who have no roentgenological evidence of the disease (26, 80, 399). Reversily roentgenological evidence alone cannot always exclude the presence of other oesophageal diseases, for instance cancer (45, 80, 152, 191).



VIII
Weblike stricture of the oesophagus



IX
"Tertiary contractions" of the oesophagus

OESOPHAGOSCOPY [1])

The diagnosis of oesophagitis can only be accurately determined by direct visualisation of the oesophageal mucous membrane. We feel that as it is essential to obtain a satisfactory view of the region involved, the use of general anaesthesia should be considered, especially in the case of a restless patient.

The proximal part of the gullet, when viewed through the scope, generally appears healthy. Towards the lower end the mucosa becomes

[1]) 11, 37, 42, 53, 95, 121, 168, 178, 203, 204, 205, 247, 248, 447.

pinker and thicker and the passage of the scope may be impeded by redundant folds. The mucosa is congested and bleeds easily on contact with the scope or a swab. Ultimately shallow ulcers, usually covered with a yellowish slough may be seen. When this slough is swabbed away an intensely red, bleeding, granular base remains. These ulcers are usually surrounded by a pink margin. Although the bleeding is generally diffuse, one may on occasion see a small artery spurting blood (97). In more profound cases the entire lower gullet ulcerates. Transition to normal mucosa is often irregular and ulcerations may stretch in long shoots upwards. In chronic cases solitary round or oval ulcers are not uncommon. The surrounding mucosa is usually scarred and whitish in appearance.

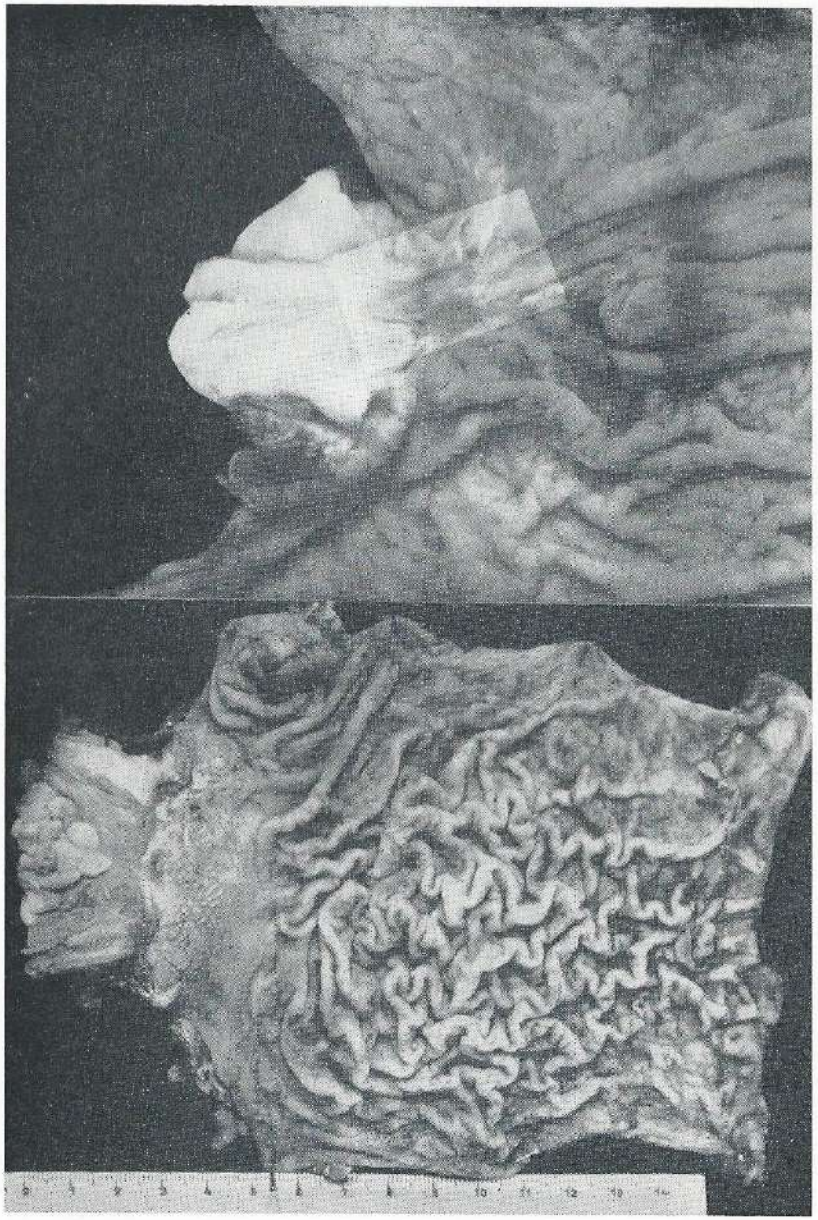
The cardioesophageal junction is nearly always found to be more proximal than one would expect as it is then situated above the diaphragm. In exceptional cases it may even reach as high as the aortic arch (37, 66). A typical feature of the disease is that the scope can be pushed into the stomach without deviation. Due to these factors one is liable to pass the cardia accidently. The stomach is however easily identified by the rugose appearance of the gastric mucosa.

Characteristically the cardia is patulous. As a result the view may be obscured by continous reflux of stomach contents. In a severe case the abounding gastric mucosa may join the oesophageal inflammation but usually the inflammation ends abruptly at the transition to gastric mucosa.

The passage of the scope may be obstructed by constriction of the oesophagus. This constriction is generally concentrical with a funnel-like inlet, although an abrupt obstruction is not uncommon. It is usually situated in the lowermost part of the oesophagus, but it may be encountered higher. It is almost always above the diaphragm. There may be ulcerations in the stenosis itself. Usually it is impossible to look through the stenosis without dilating it first. Beyond the stenosis the stomach is seen. The gullet, proximal to the stenosis, is never greatly dilated.

The stenosis may be a stricture or caused by spasm. Usually only by repeated examinations can this be determined.

Occasionally such a bleeding, ulcerating impediment in the oesophagus appears cancerous. In such cases a biopsy is clearly indicated.



XI *This stricture (in rectangle) was barely accessible to a matchstick. The patient complained of only slight dysphagia*

X *Flat ulcerations of the lower oesophagus, sharply limited below by gastric mucosa. Illustration no. III is an X-ray taken before resection of this specimen*

Chapter III

MORBID ANATOMY[1])

The lower gullet is the site of the inflammation.
Gastric mucosa forms the lower limit.
Acute and chronic inflammation are simultaneously present.

Essentially the disease is confined to the oesophagus, the lesion is sharply limited below by gastric mucosa. The distal part of the oesophagus is always involved. Generally less than the lower third of the oesophagus is involved although occasionally half and rarely the entire oesophagus may be diseased. The lowermost lesions are the severest. Ulcerations are of many irregular and different shapes and sizes — frequently linear in shape and situated lengthwise in the oesophagus.

As a rule acute and chronic inflammation are found simultaneously implying repeated attacks (PETERS 1955). In the acute stage the epithelial lining is usually shed to some extent, even the greater part of the tube may be stripped of epithelium. Shallow ulcers may be seen with brownish or greenish bases and ragged edges. In the more severe case the whole surface appears raw and haemorrhagic. In extreme cases the lowermost part of the oesophagus may disintegrate completely and appear blackish. This has been termed "intra-vital softening" or "oesophago-malacia".

The muscular wall may be affected and the inflammation may spread to the mediastinal tissues. Perforation need not necessarily be present for acute mediastinitis to occur, although when severe and progressive, this is usually the case.

The chronic stage is characterized by regeneration of the surviving epithelium and fibrosis. According to PETERS (1955), some ulcers at this stage have a lower margin which is fairly abrupt and a proximal margin, which is long, flat and terraced with a dirty brown base. Others are smooth, circumferential and whitish. Increasing thickness of the oesophageal wall by active fibrosis advances with time and a stricture develops. These strictures are generally funnel-shaped, the

[1]) 33, 47, 53, 88, 89, 201, 204, 232, 346, 351, 373, 378, 384, 389, 428, 451, 482, 484, 506.

inlet pointing orally, although strictures, which begin abruptly, are not uncommon. Usually the strictures are short, not exceeding 1,5 cm in length but, of course, depending on the severity, these may be much longer and may even involve the greater part of the oesophagus.
Microscopically the lesion is generally more extensive than would be suspected by naked-eye inspection. QUINCKE (1879) was the first to note the similarity of the microscopic aspect to peptic lesions elsewhere. Later HAMPERL (1934) proposed the name "peptic oesophagitis", because oesophageal ulcerations have all the anatomical characteristics of a chronic ulcer of the stomach or duodenum (121, 178, 238, 299). Although fibrinoid necrosis, which is found in many cases, is definite proof of acid peptic activity, it need not be present for the diagnosis of peptic activity. In the course of the disease, this characteristic feature usually disappears and nonspecific, granulating ulcerations and fibrosis are mainly present.
This inflammatory fibrosis is usually confined to the submucosa; only in severe cases is the muscular wall involved. Accordingly peri-oesophageal involvement of the mediastinal tissues is uncommon.

Chapter IV

NATURAL COURSE[1])

The natural course of the disease is well-known due to the fact that many physicians have been unfamilair with the clinical entity. The onset of the disease may occur at any time, although it generally occurs in the extremes of life, either during early childhood or after middle-age. In extensive series of patients SCHMIDT (1954) and ALLISON (1949) reported 70% and 90% resp. as being past the age of fifty. 73% of our cases were past this age. Taking into account that many children are probably referred to a pediatrician and therefore not included in these series, they are definitely in the minority. The average patient is past 50 years of age. Only THOMSEN reported the unusual incidence of 11 patients under 10 years of age in a series of 27.
The onset of the disease can often be traced to an operation, a confinement or some serious disease; the relation frequently not being clear. In 25% of our cases the onset was in this postoperative period.
The course of the disease is usually benign and protracted, as seen for instance in the series of SWEET (1954) where 40% of patients suffered complaints for more than 5 years, and 14% for more than 10 years. In our series the duration of complaints exceeded 1 year in 75%, 5 years in 30% and 10 years in 18% of all cases. The longest recorded was 55 years, the shortest a few hours.
Acute oesophagitis is by no means rare, however, and instances have been known where the disease took a lethal course in a few hours time.
Most patients recall no outstanding symptoms at the initial stage of the disease, and sought no medical treatment. They, on their own accord, follow a soft, bland diet in order to relieve the vague discomfort in the upper abdomen. At this stage there is no acute suffering and patients manage without great inconvenience. When dysphagia occurs it may be compensated temporarily by masticating thoroughly

[1]) 8, 9, 36, 37, 40, 42, 47, 53, 69, 83, 84, 90, 93, 94, 95, 102, 103, 104, 105, 108, 114, 129, 130, 137, 149, 152, 178, 195, 196, 203, 229, 234, 235, 286, 294, 299, 321, 334, 375, 378, 407, 410, 442, 449, 451, 454, 466, 471, 504.

and by washing down the food with liquids. In the long run, patients prefer semi-solids.

Postures, that provoke pain, for example bending forwards, sleeping supine etc. are soon recognized and consequently avoided. Therefore many patients sleep propped up in bed. Spontaneous intermissions are the rule and relapses are not necessarily worse than earlier attacks. This chronic, intermittent course is reminiscent of a gastric or duodenal ulcer, although according to HARMAN (1952) it does not appear periodically over weeks or months as in the case of these ulcers. This may go on ad infinitum. This fact is demonstrated by BROWN-KELLY (1953) in his report on 6 patients. After a 20 years interval, they were all in good health and neither physically nor mentally impaired; five of them even served in the Forces. Therefore it is probably true, that a large number of patients, although not treated, do not become worse. A small number of patients, however, tend to become progressively worse, thereby offering a challenge to the medical profession. These patients suffer much pain and the passage of food becomes gradually impaired. Obstruction is generally due to a stricture, which is caused by chronic inflammation. It usually takes a few years for these strictures to develop, although a severe stricture may develop in a few months and in rare instances even in a few weeks time (8, 36, 42, 53, 84, 129). For example one patient in our series, a 36-year-old male (H.M. 12146) developed a stricture shortly after subtotal gastrectomy for duodenal ulcer. On illustration VII this progress is evident.

While a stricture is developing, the passage of food may be temporarily blocked by spasm. This spasm, which is frequently influenced by emotional factors, may on one day obstruct even the passage of fluids but may allow all food to pass freely the next day (9, 93). In childhood, oesophageal strictures are considered to be rarely of a serious degree (GROSS 1953); the incidence being estimated at less than 10% (CARRE 1952).

Ultimately no food or fluids pass the gullet and starvation becomes imminent. In 1933 HINDSE-NIELSEN collected 6 cases, who died of starvation, from the literature referring to 71 cases. At present, death due to starvation is exceptional, but chronic malnutrition may make these patients prone to infection and in this way death may still take its toll.

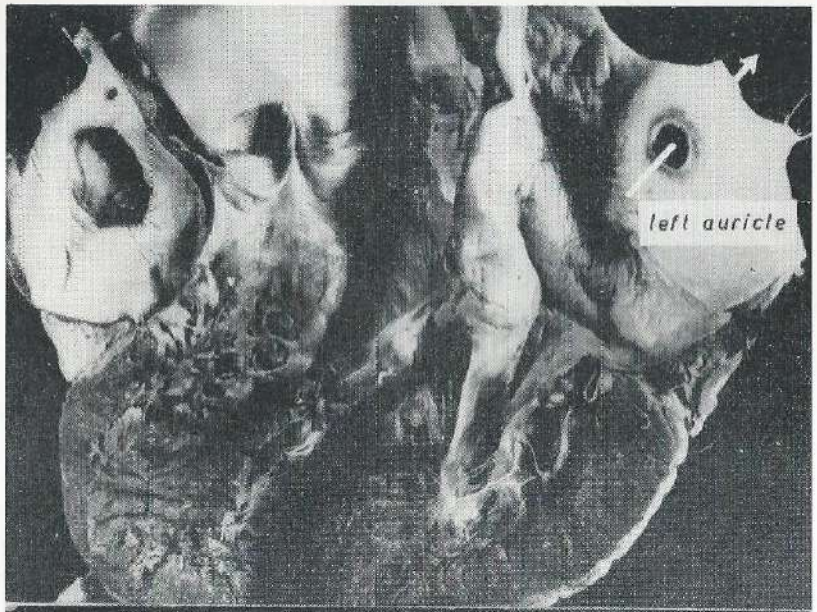
Haemorrhage may be the first symptom of the disease, particularly in infancy. It has been known to occur in the newborn with fatal outcome (371, 435, 449, 454). Excessive bleeding without any foreboding, is a rarity in the case of adults. In our series, only one patient, a 23-year-old male (H.M. 11691), experienced excessive bleeding without any other symptoms being present. He required a vast number of blood-

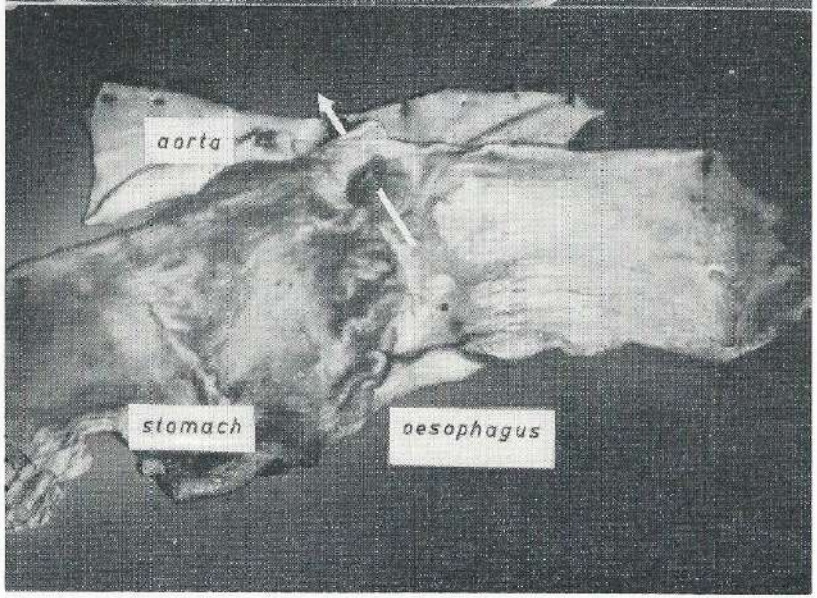
transfusions, 65 in all! We were able to recognize the disease because an oesophago-gastrostomy had previously been done and indeed the presence of ulcerative oesophagitis was confirmed by resection.

RIPLEY (1952) reported a similar case where 100 bloodtransfusions were given. With the routine use of bloodtransfusions, fatal haemorrhages have become rare. The treatment of profusely bleeding patients, however, is troublesome. Naturally a peptic oesophageal ulcer, perforating into the heart or aorta, will cause immediate death (299, 321, 375, 437, 451, 501). Prior to fatal haemorrhage, small haematemeses usually occur. In our series two patients died from this cause. One, a male 65 years of age (H.M. 11062), developed oesophagitis soon after a proximal gastric resection for cancer. He was admitted shortly before his death with severe retrosternal pain and recurrent haematemeses. He died suddenly of severe haemorrhage. The cause, the perforation of an ulcer into the left auricle of the heart, is evident on illustration no. XII (187). The other patient, a 60-year-old male (H.M. 8943) was admitted elsewhere with recent, recurrent haematemeses, five years after a proximal gastric resection for cancer. His sudden death was caused by the perforation of an oesophageal ulcer into the aorta. (Illustration XIII.) Manifest bleeding is present in 10%—50% of all cases. Recurrences are common. In our series the incidence was 38%.

Repeated small haemorrhages tend to make a patient anaemic in the long run (152, 234, 286, 294). This anaemia may dominate the clinical picture, when no other signs of the disease are evident. Our series included only one example of this. This patient, a female, 59 years of age (H.M. 13242) suffered from severe anaemia for 2 years, the cause of which was discovered to be oesophagitis. By treating the oesophagitis the anaemia was cured.

Perforation of an oesophageal ulcer is a catastrophy, which almost without exception terminates in death within a few days. Since ROTTER'S report in 1850, many instances of perforation have been published, although it seems sufficiently rare to warrant publication in each instance. Perforation is generally considered an uncommon complication. It occurs only when the disease takes a fulminating course. In exceptional cases, the course of the disease may be so rapid that perforation occurs a few hours after the onset of the disease (105). The cases of CUSHING (1932) and others are illustrative of this fact (264, 303, 314, 503). In these extreme cases the disease may simulate spontaneous rupture (20, 105, 300, 323, 339, 390, 444, 492). They are very uncommon. On the contrary, penetration of an oesophageal ulcer into the mediastinum is a fairly common occurrence. According to ALLISON, "boring pain felt deeply behind the sternum and radiating to the back usually indicates the spread of inflammation from the





XII

Perforation of an oesophageal ulcer into the left auricle of the heart (indicated by arrow)

XIII

Perforation of an oesophageal ulcer into the aorta (indicated by arrow)

submucosa of the oesophagus to the deeper layers and the mediastinum". The sequence of this may be a mediastinal absces. This absces may secondarily perforate into the pleural space, the bronchial tree, the peritoneal cavity or back into the oesophagus again (33, 108, 137, 151, 385, 439, 468, 501). Perforation of an oesophageal ulcer into the mediastinum becomes apparent by rapidly increasing pain in the upper abdomen and behind the sternum, radiating to the back, the neck and shoulders (105, 167, 300). It is usually accompanied by vomit, which may have an aspect of coffee-grounds. Soon the patient becomes restless with respiratory difficulties, tachypnoe, cyanosis. He runs a high temperature, the pulse rate increases, unconsciousness follows and subsequent death. On the X-ray of the thorax, perforation may become apparent through air bubbles in the mediastinum. When the diagnosis is in doubt FINCHER (1949) advises the introduction of iodized oil into the gullet. The diagnosis is confirmed when the oil is seen to diffuse into the mediastinal tissues. In a long-standing case, perforation of the oesophagus into the mediastinum is prevented by surrounding inflammatory fibrosis. It takes place into the left or right pleural space instead (334, 389, 407, 410, 451). The usual signs of pleural effusion are then found.

Where a peptic oesophageal ulcer perforates into the peritoneal cavity, the physical signs are identical to those of any perforation into this cavity (90, 108, 178, 407, 451). An extraordinary case was reported by BRANDSMA (1947). Perforation of an oesophageal ulcer had taken place into the retroperitoneal tissues. Subsequent breakdown of the serosal lining was responsible for generalized peritonitis. A peculiar feature found at laparotomy was a chocolate-like discolouration of the peritoneum and subserosal tissues around the stomach, spleen, pancreas and left diaphragm. The author proposed the name "brown belly" for this.

Hydro-pneumo-pericardium is a clinical entity which is found when an oesophageal ulcer penetrates the pericardium. (302, 448). According to SHACKELFORD (1931), who reviewed 77 cases, a loud, metallic, splashing sound, synchronous with the heartbeat, is typical. This may be heard with the unaided ear at a distance of several feet. On percussion, a characteristic sign is a high-pitched tympanytic area over the precordium which shifts with changes in posture. Confirmation of the diagnosis may naturally be provided by X-ray examination. SCHALM (1950) observed in one case that shock was evident in the sitting position, caused by heart tamponade of accumulated fluid. On lying down the fluid was drained back into the oesophagus and all signs of shock disappeared.

As previously stated, perforation of the oesophagus is generally lethal. A few exceptions to this rule have been reported by DICK (1942),

BARRETT (1947), LYNCH (1949), HOOVER (1952) and CANTOR (1953). BARRETT (1950) and BELSEY (1953) expressed doubt as to whether an oesophageal ulcer could perforate at all. According to BELSEY "In contradistinction to peptic ulceration of the gastric mucosa reflux oesophagitis does not lead to perforation of the organ . . .". By many meticulously documentated reports this has been proved to be untrue. In two of our cases perforation was definitely situated in the oesophagus as proved by microscopical examination.

The development of cancer due to chronic oesophagitis is considered to be rare.

The following cases have been reported:

1 case by ORTMANN (1901)
3 cases by BENEDICT (1946)
4 cases by SMITHERS (1950)
1 case by DAWSON (1950)
9 cases by RESANO (1950).

Chapter V

INCIDENCE

Reflux oesophagitis is a common disease

The incidence of reflux oesophagitis is higher than generally realized, because many cases are discovered during oesophagoscopy, who otherwise have no complaints.

In 1929 JACKSON reported the, at the time, remarkable incidence of 88 oesophageal ulcers or the scars thereof during 4000 oesophagoscopies. Apparently it is even more common than this as BENEDICT (1954) reported 60 cases found during 1000 oesophagoscopies. These endoscopies were performed for symptoms referring to the gullet.

Oesophagitis is common as a cause of oesophageal obstruction. In a series of 507 cases reported by ALLISON (1949), 105 were due to this cause. In a series of 1340 reported by BENEDICT (1954), the incidence was 119.

Estimation of the incidence of the disease from series of autopsies, is rather difficult, due to the fact that the lower oesophagus rapidly disintegrates after death as a result of autolysis. This explains the divergent incidence reported by different authors, listed below. The high incidence of approximately 7% reported by BUTT is probably due to the special pain the author took to examine the oesophagus shortly after death.

In 1906	TILESTON	reported 6 cases	(0.13%)	in a series of 4496
1911	GRUBER	15	(0.35%)	4208
1929	STEWART	1	(0.01%)	10000
1935	BARTELS	82	(1.37%)	6000
1936	BUTT	213	(7.02%)	3032
1946	EMERY	5	(1.5%)	324
1955	PETERS	116	(0.56%)	20000
1955	LODGE	25	(5.0%)	500

The disease shows a slight preference in affecting the male, as statistical evidence by the following authors shows:

BUTT	1936	136 males	—	77 females
ALLISON	1948	34	—	29
BENEDICT	1954	60	—	40
CARVER	1954	73	—	57
SCHMIDT	1954	101	—	69
PETERS	1955	65	—	49

In adding these figures a 3 to 2 ratio evolves. In our series females were prevalent in a ratio of 34—26. A female preponderance was also reported by BEACONSFIELD (1953).

Chapter VI

CONCOMITANT DISEASES AND CONDITIONS[1])

Reflux oesophagitis is seldom an isolated disease.

Reflux oesophagitis, although itself a disease, is commonly accompanied by other affections. In recent years, surgeons have become familiar with this troublesome disease which frequently complicates surgery of the cardia of the stomach. In this respect proximal gastric resection and oesophago-gastrostomy for achalasia (HEYROWSKY, GRONDAHL) have become notorious.

The most constant companion of oesophagitis is the diaphragmatic hernia through the oesophageal hiatus, especially the "sliding type". The association is reported in more than 75% of patients. BENEDICT (1954) and SCHMIDT (1954) reported an even higher incidence, that of 85% and 93% resp. Vice versa in the case of hiatal hernia of the "sliding type", the presence of oesophagitis is equally common, i.e. 70% (ALLISON 1951) and 60% (BELSEY 1953).

Of special interest is the hiatal hernia of the "sliding" type with the short oesophagus and oesophagitis, which entity was for the first time described by HAROEN (1934). This concurrence is so pertinent that you never meet either alone (ALLISON 1948).

A similar association is that of the disease with a condition known as "the oesophagus with gastric mucous membrane" (ALLISON (1953). This association is by no means rare, e.g. ALLISON (1953) in a series of 125 cases reported an incidence of 21. It is difficult, sometimes impossible, to differentiate between this association and the previous one of the "sliding" hernia with a short oesophagus. It was first reported by BOSHER (1951).

According to BARRETT (1955) this can be determined by the difference in blood supply. This seems rarely feasible, however.

Oesophagitis is frequently accompanied by peptic affections of the stomach or duodenum, although the reported incidence is divergent: CARVER (1954) reported 10%; BUTT (1936) and SCHMIDT (1954) and

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1) 6, 7, 8, 11, 32, 33, 35, 42, 47, 48, 58, 60, 88, 89, 95, 102, 121, 135, 148, 179, 203, 210, 227, 247, 349, 350, 361, 379, 383, 384, 397, 416, 426, 447, 449, 452, 457, 498, 507.

CROSS (1956) reported 20%; BRINKMAN (1941); BENEDICT (1954) and STEWART (1955) 40%; WINKELSTEIN (1954) 85%.

On account of the frequent association between oesophagitis and hiatal hernia on the one hand, and gastric or duodenal ulcer on the other hand, all three diseases may occur simultaneously. This triad was first described by BENEDICT (1948). CARVER (1958) recently reported an incidence of 20% of the triad of Benedict in a series of 150 patients. In our series the incidence was 10%.

To a lesser degree oesophagitis is accompanied by cholecystopathia. The association was found in 19% of cases in SCHMIDT's series. In our series it amounted to 15%.

Relation of oesophagitis with acute affections of the brain was first reported by ROKITANSKY in 1841. In 1932 CUSHING considered a clear-cut relationship to exist between acute oesophagitis and affections of the interbrain. Later publications by MASTEN (1934), BUTT (1936), FINCHER (1949), KING (1953), MACIVER (1955) and WHITBY (1958) have since proved that diverse affections of the brain may be associated with acute oesophagitis.

The following are the various affections reported:
tumor, meningitis, cerebral haemorrhage, cerebral oedema, encephalitis, cerebral absces, following endocranial operations, and following head injuries.

A remarkable fact is that oesophagitis may be the direct sequence of an operation, generally laparotomy (33, 88, 89, 222, 312, 371, 383, 384, 452). It has been known, however, to occur after thoracotomy, operations on the extremeties etc. It was first noted by VON EISSELSBERG in 1899. In 1936 BUTT and VINSON classified 75% of their series of 213 cases, as belonging to this category. In recent years the occurrence is reported less frequently. BENEDICT (1954) reported only 22% to belong in the post-operative group. In our series it amounted to 10%.

During pregnancy oesophagitis may develop. VINSON (1921) was the first to describe this. In 1937 he found 13 in a series of 186 cases. HOOVER (1952), HILLEMAND (1954), LUND (1954) and MARCHAND (1955) have reported further cases. The occurrence is not frequent, however, and does not rely on the presence of a hiatal hernia (MARCHAND 1955). In 4 cases of our series oesophagitis developed during pregnancy.

A great many other diseases have been mentioned associated with oesophagitis by various authors (33, 88, 89, 135, 179, 215, 247, 389, 426, 452, 471). The coincidence is probably by chance.

Chapter VII

DIFFERENTIAL DIAGNOSIS

Although the tetrad of pain, dysphagia, haemorrhage and regurgitation with their relation to posture, characterizes the disease, it is not wholly pathognomic.

The oesophageal triad dysphagia, retrosternal discomfort and regurgitation is present in most afflictions of the oesophagus (THOREK). The addition of haemorrhage is possible in some diseases while it may be lacking in oesophagitis.

CANCER

Oesophagitis should mainly be differentiated from carcinoma of the gullet and the cardia. They may resemble each other in practically every respect and only biopsy can verify the diagnosis. Besides carcinoma and inflammation may occur together.

ACHALASIA

Reflux oesophagitis may have certain features in common with achalasia, especially when the latter does not show much dilatation of the gullet (416). In both affections, the X-ray picture may show a funnel-like constriction of the distal part, but the constriction due to oesophagitis lies above the diaphragm. Achalasia is nowadays considered to be a primary oesophageal muscular dystonia, the predominant feature being a flaccid dilatation (216, 278, 363, 386, 440, 460, 463, 488, 490). Because of this, the oesophagus contains fluid and gas and shows little or no peristaltic activity. On the contrary, when due to oesophagitis, dilatation is usually absent even in advanced cases, and on fluoroscopy hyperperistalsis is revealed. When in an early case doubt arises as to the identity of the disease, an injection of 6—10 mg of mecholyl may settle the issue. According to KRAMER and INGELFINGER (1949) marked spasm of the oesophagus results in the case of achalasia in addition to severe substernal pain (MOERSCH (1952) and HIGHTOWER (1955)).

SCLERODERMA[1]

Patients with scleroderma may suffer from obstruction. This is caused by hypotonia of the oesophagus. In this disease the cardia is usually open and reflux oesophagitis complicates the disease. We observed one case. Skin lesions usually betray the presence of scleroderma.

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[1]) 68, 121, 143, 258, 278, 391, 431, 463.

LOWER OESOPHAGEAL RING

SCHATZKI (1953), INGELFINGER (1953) and BUGDEN (1956) reported a few cases with a so-called "lower oesophageal ring", by which is meant a concentric membrane caused by neither fibrosis, nor inflammation.

Oesophagoscopy settles the issue.

EPIPHRENIC DIVERTICULUM

Obstruction of the lower oesophagus may be caused by an epiphrenic oesophageal diverticulum (127, 193, 280, 297). The cause of this is probably concurring cardiospasm, according to KAY (1953).

When on the alert for the condition, the diverticulum, usually located in the posterior side, will be revealed by the X-rays. These epiphrenic diverticula are uncommon. E.g. G. HARRINGTON (1949) treated only 8 patients as opposed to 216 with pharyngo-oesophageal diverticulum.

VARIOUS AFFECTIONS

Acute oesophagitis may be caused by infectious diseases as for instance variola, typhoid fever, diphtheria, scarlet fever and others. Pemphigus of the oesophagus and thrush oesophagitis are other rare affections, as is acute oesophageal ulcer with "intranuclear inclusion bodies", reported by HARTZ e.a. (1941) (46, 509).

When differentiating between affections of the oesophagus, manometry in addition to roentgenology and oesophagoscopy, may add valuable information (122, 123).

BOOK II

"That one part of the body should be at permanent hazard from the secretions of another seems an oddity of nature". (PETERS 1955).

Chapter VIII

AETIOLOGY

Reflux of gastric or other digestive juice is the main cause of the disease.

Concerning the aetiology there is practically unanimous agreement, that inflammation of the lower gullet is caused by the action of the gastric juices. In 1879 it was QUINCKE, who first recognized this action of the gastric juices on the lower gullet in histological specimens. This publication referred to three autopsy cases with, what he called, "ulcus oesophagi ex digestione". In recent years accumulated experimental and clinical evidence has proved that the oesophageal lesions are indeed mainly caused by gastric juices, but that the oesophagus may also be damaged by other intestinal juices.

In experiments FERGUSON e.a. (1950) demonstrated that the gullet of cats and dogs, when perfused with gastric juice, developed a severe inflammation, often in less than an hour. He proved besides that the higher the acidity, the more severe was the oesophagitis.

In experiments on dogs by RIPLEY (1950), KIRILUK (1951, 1954) and ELLIS (1954, 1956), it was demonstrated that when the cardia of the stomach was either resected or bypassed, thus allowing free access of gastric contents to the oesophagus, all dogs developed oesophagitis. It was ascertained besides that the oesophagus, as compared to the jejunum, is by far the most susceptible to the destructive properties of the gastric juice. Similar observations have been made in the case of human beings, where the cardia was resected for cancer, for example, or bypassed as in the ill-fated procedures of HEYROWSKY or GRONDAHL for achalasia. From 65 of such cases, RIPLEY (1952) reported that 40 developed severe oesophagitis. Many other authors have made similar observations (36, 73, 233, 256, 262, 294, 344, 362, 504).

Furthermore oesophagitis is common in the condition where the oesophagus is lined with gastric mucosa. The proximal oesophageal epithelium is continuous with the lower part, lined with gastric mucosa, which produces acid gastric juices that affect the upper part. (13, 37, 307). Before this entity became known, it was for some time a controversial point whether ectopic gastric mucosa in the gullet could be the cause of the disease (95, 246, 465, 515). Ectopic islets

of gastric mucosa, which were the first reported by SCHMIDT in 1805, are commonly present in the oesophagus. RECTOR and CONNERLY (1941) found these islets in 118 cases out of 1000 routine autopsies on children (26 of these containing perietal cells). It was thought that the oesophageal ulcers might originate in the same way as in a Meckel diverticulum of the small gut, where such ectopic gastric islets also occur.

In experiments on dogs, ARROYAVE (1950), transplanted a small patch of gastric mucosa, 1½ cm. in diameter, with its nerve and blood supply both intact, into the oesophagus. All these dogs, indeed, developed an oesophageal ulcer. But ulcers in islets of gastric mucosa in the human gullet are extremely rare. Besides which the ectopic islets are generally located in the proximal segment of the gullet, in contrast to the disease concerned. We agree with BARRETT (1950) who stated that "it is known that they secrete acid, but I cannot believe that the tiny volume of acid, diluted by pints of saliva would be likely to harm the lower reaches of the gullet". It must be concluded that the gastric juices which ravage the lower gullet are derived from the stomach.

These gastric juices gain access to the gullet through the cardia which normally prohibits this. This transcardial reflux of gastric juice into the gullet, made possible by an ineffective cardia, is the main feature of the disease. It was first seen by ROBINS en JANKELSON in 1926 during fluoroscopy. In 1953 transcardial reflux as the principal cause of oesophagitis was confirmed by AYLWIN, by means of continuous suction during the night on a small polythene catheter which had been introduced into the oesophagus. He proved in 50 cases that there existed a direct correlation between the severity of the inflammation and the amount of acid in the nightly samples. In the 19 control cases no gastric juice could be recovered.

Direct correlation between the severity of the inflammation and the acidity of the gastric juice has been demonstrated in experiments on dogs (154, 155, 165, 265). In a series of patients reported by WINKELSTEIN (1953) it indeed transpired that 85% had increased gastric acidity. WANGENSTEEN and others (121, 129, 178, 417, 505, 506) accordingly consider that reflux oesophagitis "might well be a consequence of acid peptic digestive mixture in the same manner that duodenal ulcer is".

Not in all cases, however, is gastric acidity increased and in our series for instance, in only 18% did free acid exceed 40 clin. units. In 6 cases no free acid was present at all. In two of these a total gastrectomy had previously been done. Similar cases were reported by BENEDICT, BARRETT and others (90, 95, 294).

Clearly other secretions caused oesophageal ulcerations in these cases.

This has been proved in the experiments on dogs and cats by CROSS (1951), who perfused the gullet with bile and pancreatic juice. In all cases severe oesophagitis developed. This clinical and experimental evidence proves that excoriation of the oesophageal lining is due to reflux of various digestive juices.

As every endoscopist knows, oesophageal mucosa shows a marked tendency to recovery. FRIEDENWALD (1928), who attempted to provoke oesophagitis by wounding the mucosa, only succeeded when it was followed by prolonged perfusion of HCl 1%. This adds weight to the clinical experience that oesophageal inflammation is due to continued *long-term* bathing by digestive juices (372).

The mechanism of reflux, however, requires explanation. Another remarkable point is the fact that reflux does not necessarily lead to oesophagitis (174). ALLISON (1953) estimated that in 75% of adults, reflux produces oesophagitis.

As in the case of reflux, continual vomiting may also cause damage to the oesophageal lining. And indeed cases have been known where oesophagitis originated as a result of vomiting during pregnancy, migraine, seasickness (372) or other diseases where there is severe vomiting, (124, 144, 313, 449). These cases are exceptional however. Considering that vomiting is a common occurrence, this is remarkable. Of special interest in this respect, for instance, is the absence of oesophagitis in the case of the most aggravated pyloric stenosis (178, 505, 506).

VINSON (1921, 1936) and BUTT (1936), who first reported such cases, were at the time convinced that vomiting was a major cause of the disease. They realized, however, that vomiting could not be the sole cause and looked for another factor, which they thought to be the inlying nasal gastric tube. Indeed FARRIS (1956) recently pointed out that such tubes may harm the oesophagus, but the decubitus was generally in the proximal segment of the gullet. No great aetiological value can be attached at present to the use of gastric tubes (144, 361). This is illustrated by 100 patients examined by BENEDICT (1954) of whom only 5 had ever had a gastric tube. VINEBERG (1933), BUTT (1936), AYLWIN (1953) and SWEET (1957) hold that another factor responsible, may be a change in the buffering capacity of the oesophageal secretions and the saliva which normally protects the oesophageal mucosa. They pointed out that stimulation of the vagal nerve is responded to by a copious serous flow with a poor buffering quality. It is possible that by stimulation of the vagal nerves, susceptibility to oesophagitis is increased.

Other factors, i.e. excessive use of alcohol, tobacco, focal infection and vascular disturbances, as suggested by various authors, we do not consider to be of aetiological importance (247, 371, 428, 472).

Chapter IX

THE CLOSING AND OPENING MECHANISM OF THE CARDIA

A cardiac sphincter prohibits reflux.
Relaxation of the cardiac sphincter is effected by contraction of the lower oesophagus.

In the preceding chapter the main cause of oesophagitis was seen to be incompetence of the cardia. There is still a lack of knowledge however, concerning the opening and closing mechanism of the cardia. For instance about the nature of a cardiac sphincter, there exists no unanimous agreement. The need to account for the function of the cardio-oesophageal junction and the absence of an anatomical sphincter is the root of this controversy (301, 310, 498).

JACKSON 1922 (247) and SAUERBRUCH 1932 (420) sought the solution in a sphincter extrinsic to the cardia. During fluoroscopy delay of barium suspension at the level of the diaphragm during inspiration was interpreted by these authors as a pinchcock-action of the diaphragm. ALLISON (1951) has in recent years become the leading exponent of this concept and explains the action of the diaphragm as follows: when the oesophagus comes through the hiatus ". . . . the canal takes a bend forwards and to the left and this bend is lassoed and maintained by the right crus of the diaphragm, which hitches it down to the lumbar spine". "When the right crus of the diaphragm contracts its action on the cardia is twofold: first, it compresses the walls of the oesophagus from side to side and second it pulls down and increases the angulation of the oesophagus".

We do not believe in a diaphragmatic pinchcock.

If it exists:

1. the sphincter should be ineffective when the hiatus is enlarged, but every surgeon has encountered the enlarged hiatus during laparotomy without any evidence of reflux (171, 210).
2. reflux of the contents of the herniated gastric pouch should occur in every hiatal hernia, but reflux occurs only in a minority. HARRINGTON (discussion COLLIS 1957) for instance, recently estimated that about 17% of all diaphragmatic hernias are complicated by oesophagitis.

3. the amount of reflux should increase with the size of the hernia, but the relationship appears to be an inverse one (47, 48, 123, 186, 315, 370, 402).
4. incompetence should result when the left diaphragm is paralysed, but paralysis of the left diaphragm has been advocated as therapy for a troublesome hiatal hernia (18, 206, 210, 320, 364, 374). It should be pointed out that although the right crus of the diaphragm forms the main part of the muscular sling around the hiatus, it is innervated by the left phrenic nerve (110, 359). During general anaesthesia paralysis of the diaphragm can be obtained by muscle relaxants. When a full stomach is then manually compressed during laparotomy, no reflux can be instigated (364).
5. which structure protects the abdominal oesophagus from destruction by gastric juice? ALLISON's (8) explanation that "there is in life no such thing as the abdominal oesophagus" is not convincing, as every surgeon, who is familiar with the hiatal region, will agree (210). Furthermore the abdominal oesophagus has been identified by means of roentgenology. By marking the hiatus, MONGES (1955) and MULLER BOTHA (1957) clearly demonstrated the cardia to be at a lower niveau than the hiatus. If there should still be doubt as to existence of an abdominal oesophagus, it can be remarked that in the operation of BOEREMA for hiatal hernia, an abdominal oesophagus is expressly created. Oesophagitis has never resulted from this precedure. Similar observations were also made by BRAASCH (1956) and MEISS (1958), who also created abdominal oesophagi in dogs.
6. why is the action of the diaphragm on the oesophagus then not felt during laparotomy when a finger is inserted into the hiatus and the diaphragm functions normally? (70, 142).

From these observations, the conclusion may be drawn, that the diaphragm does not play any role in the all-important function of the cardia of limiting reflux. It has recently been proved by the efforts of DORNHORST (1954), CREAMER (1955), FYKE (1956), ATKINSON (1957) and FLESHLER (1958) by means of manometry, with roentgenology combined, that the sphincter acts independently of the diaphragm. DORNHORST (1954) stated the pinchcock "to be based on an ignorance of the pressure changes involved and a misconception of the respective roles of gravity and propulsion waves in swallowing of liquids in the upright posture".

Other extrinsic sphincter mechanisms, except for MOSHER'S "liver tunnel" (336), which need not be taken seriously, cannot be imagined. Without an anatomical intrinsic sphincter, by which is meant a thickening of existing circular muscles, the clue to the cardiac function, in the opinion of many authors, is the oblique entrance of

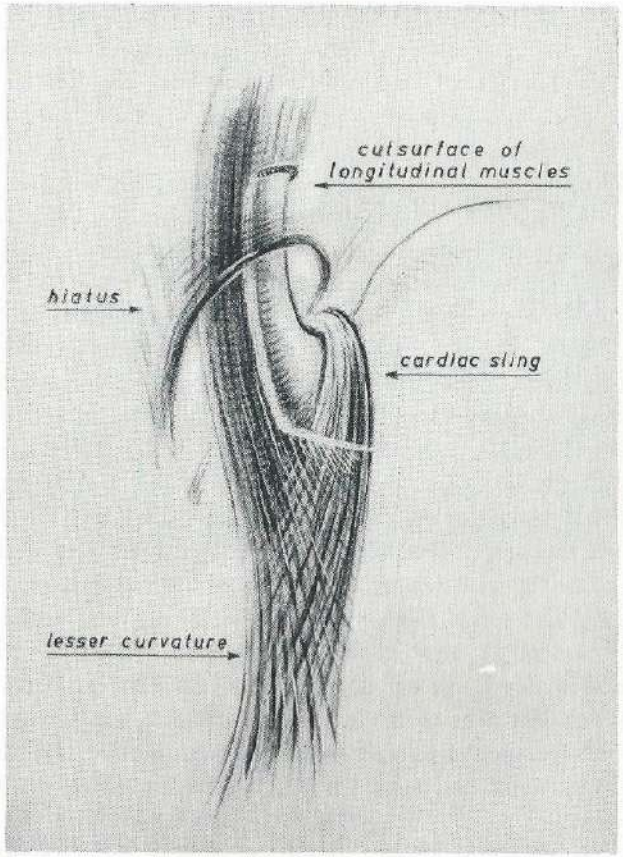
the oesophagus into the stomach. This favours the existence of a valve. It was originally described by BRAUNE in 1875 and VON GUBAROFF in 1886 and is still held in esteem by many (40, 42, 139, 296, 310). It has been made plausible in experiments by COLLIS (1954), that an increasing cardiac notch is the more effective in preventing reflux, although later MEISS (1958) could not confirm this in experiments on dogs.

A valve is not altogether in accordance with the cardiac behaviour, however. The act of vomiting and belching still requires explaining. The normal cardia, as proved by manometric studies, can withstand retrograde pressure exceeding 80 mm of mercury (142). In belching the cardia opens imperceptibly, however, and the intra-gastric pressure rises only trivially during the act of vomiting (24, 117, 142). Moreover this valve should be dependent on an acute angle of entry of the oesophagus into the stomach, but as MULLER BOTHA (1958) recently pointed out, the angle of entry varies from very acute to very obtuse, without reflux necessarily resulting. From these observations a valve, as the sole mechanism preventing reflux, becomes improbable. The extrinsic cardiac sphincter and valve, as only means of preventing reflux thus excluded, there only remains the possibility of an *intrinsic sphincter*. In experiments on dogs HOAG (1954) and MEISS (1958) proved that when the oesophagus and the cardia or part of it are anastomosed to the rest of the stomach, no oesophagitis results. This sharply contrasts with the accepted notion that oesophagitis follows oesophago-gastrostomy done without benefit of the cardia. In the thesis of NAUTA (1955) it was furthermore proved by means of roentgencinematography on dogs, that a definite sphincter cardiae exists, which lies next to the cardiac mucosa. It was proved besides that no oesophageal sphincter exists, which notion was originally favoured by PAYNE and POULTON (1927), LERCHE (1936) and others (262, 379).

In man the sphincter cardiae has been demonstrated by means of manometry combined with roentgenology. In the pressure reading a zone of high pressure was clearly seen in the cardiac region, suggesting a sphincter. This sphincter appears to be independent of externally visible kinking and successfully prevents the escape of liquids and air from the stomach, even when under high pressure (DORNHORST 1954).

It was originally thought by DORNHORST that the behaviour of the cardia, its yielding to the slight pressure of forward flow and its resistance to backflow, favoured the existence of a valve. At the same time he realized that the valve should be capable of active opening, as it was also recorded that preceding or during the act of eructation or vomiting, the pressure increased only slightly.

A high pressure zone, indicating a sphincter not exceeding 3 cm in length was consistently found in the cardia by INGELFINGER (1954), FYKE (1956), ATKINSON (1957, 1958), MULLER BOTHA (1957, 1958), FLESHLER (1958) and CODE (1958) in similar proceedings.
The presence of a sphincter has recently been proved, moreover by

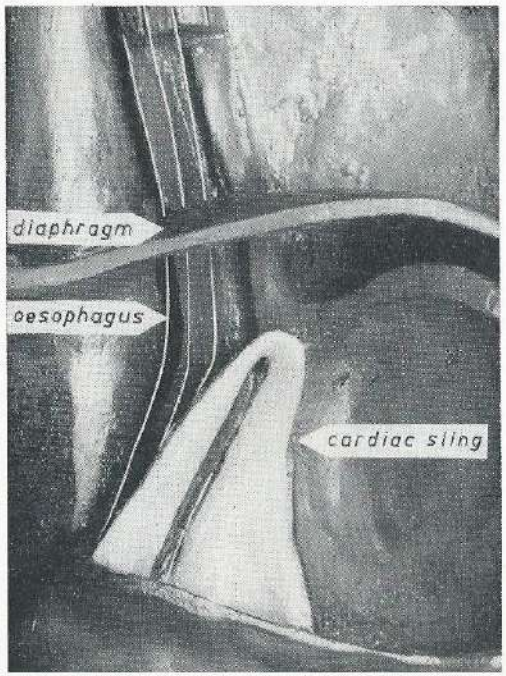


XIV

The collar fibres, which constitute a sling around the gastroesophageal junction. The longitudinal muscular coat of the oesophagus fans out along the lesser curvature of the stomach

FLESHLER who showed that the cardia, firstly can withstand a force exerted from above and secondly exhibits reflex motility in response to a relatively simple stimulus. Firstly: the oesophagus was gradually filled with barium suspension through a small catheter. As long as peristalsis was not initiated by distension (118), the cardia could resist a hydrostatic force, measured by the column height of barium suspension.

Secondly: when a balloon placed in the mid oesophagus was rapidly inflated, pressure in the gastroesophageal junction fell before the responding peristaltic wave arrived. This same phenomenon of decreasing pressure in the sphincter zone before the bolus arrived at the cardioesophageal junction was also seen in pressure recordings of the other authors. From this evidence the presence of a physiological sphincter can no longer be denied.

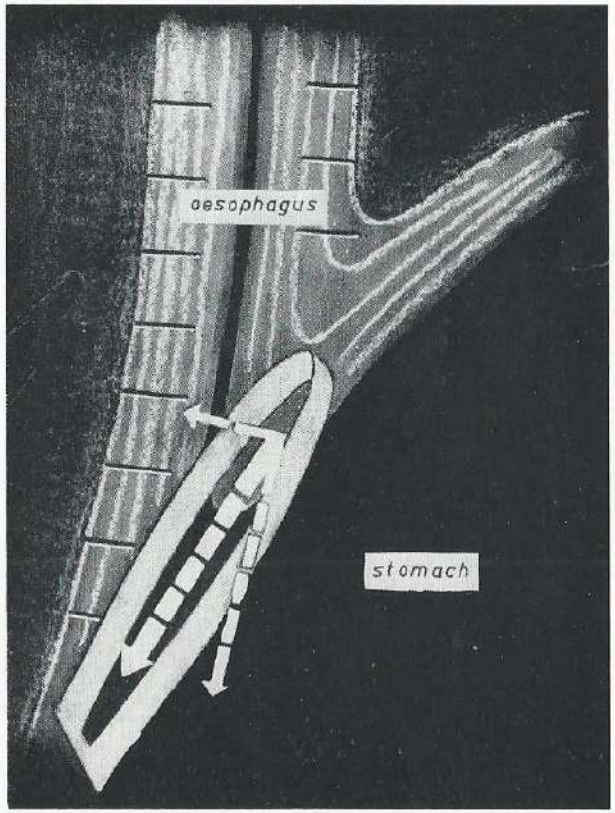


XV

Clay model, depicting the anatomical features of the cardiac region. The anterior wall of the stomach and lower oesophagus are partially removed. The cardiac sling is painted white. Silver wires indicate the longitudinal oesophageal muscles

CLERF (1933), NAUTA (1955) and MULLER BOTHA (1958) have seen the action of this sphincter from within the stomach through an endoscope and deduced that this sphincter lies next to the cardiac mucosa. NAUTA and MULLER BOTHA consequently hold the muscularis mucosae responsible. We cannot believe, however, that these relatively weak muscles can in any way withstand the great forces involded. Besides a better solution was proposed more than two centuries ago by HELVETIUS (in 1719). This sphincter is composed of the collar fibres or oblique fibres of the gastric muscular wall. (NAUTA accredited the description to BLANKAART (1696) and VERHEYEN (1711)).

These fibres which constitute a sling around the entry of the oesophagus into the stomach, lie on the lateral, anterior and posterior side of the gastroesophageal junction, run partly parallel to the lesser curvature and fan out into the body of the stomach (121, 137, 272, 293, 301, 321). See illustration no. XIV.



XVI

Schematic drawing, illustrating the action of the cardiac sling, which can be analysed in two forces: one acts downwards, parallel to the lesser curvature, producing a mucosal flap and one at right-angles to this force approximating this flap to the medial oesophageal wall

It can easily be imagined that by contraction of the muscle fibres, a mucosal flap is developed. Besides it should be noted, that the anterior and posterior cardiac mucosae are hereby brought together, thus forming a slit-like aperture. This is evident on illustration no. XI of a clay model constructed in accordance to the normal human anatomy, depicting the features of the cardiac region. The action of the muscle sling can be analysed in two forces (Illustration XVI).

One acts downwards and parallel to the lesser curvature, thus producing the cardiac notch and at the same time forming a mucosal flap at this site. The other, at right-angles to this force, approximates this flap to the medial oesophageal wall.

The oesophageal lumen at rest is tonically closed, so very little force is necessary to keep this flap approximated to the medial wall (121, 225, 296, 348, 419).

A very efficient sphincter is thus provided, which derives its strength from its valve-like structure. A watertight and airtight seal exists as long as the muscular sling insures the existence of a flap.

If this sling of collar fibres represents the sphincter cardiae, which in our opinion satisfactorily explains the hitherto encountered phenomena, it then remains to be explained how the opening mechanism functions.

According to STARLING's law of the intestine, a wave of inhibition precedes a wave of contraction in peristalsis (78, 224). In pressure readings of the act of deglutition, no decrease in pressure, aborally from the peristaltic wave, was ever recorded, however (24, 117, 123). Furthermore it was definitely established by FLESHLER that the cardiac sphincter relaxed in response to dilatation of the mid oesophagus, before the peristaltic wave arrived at the gastroesophageal junction. It thus becomes improbable that STARLING's law of the intestine can be applied to the opening mechanism of the cardia. FINDLAY (1931) and others (51, 276, 287) observed that during deglutition the cardia is pulled up, shortly before the bolus passes.

As early as 1904, MAY observed (in STARLING's laboratory), in experiments on rabbits, cats, dogs and monkeys that the cardia relaxed when the oesophagus shortened by contraction.

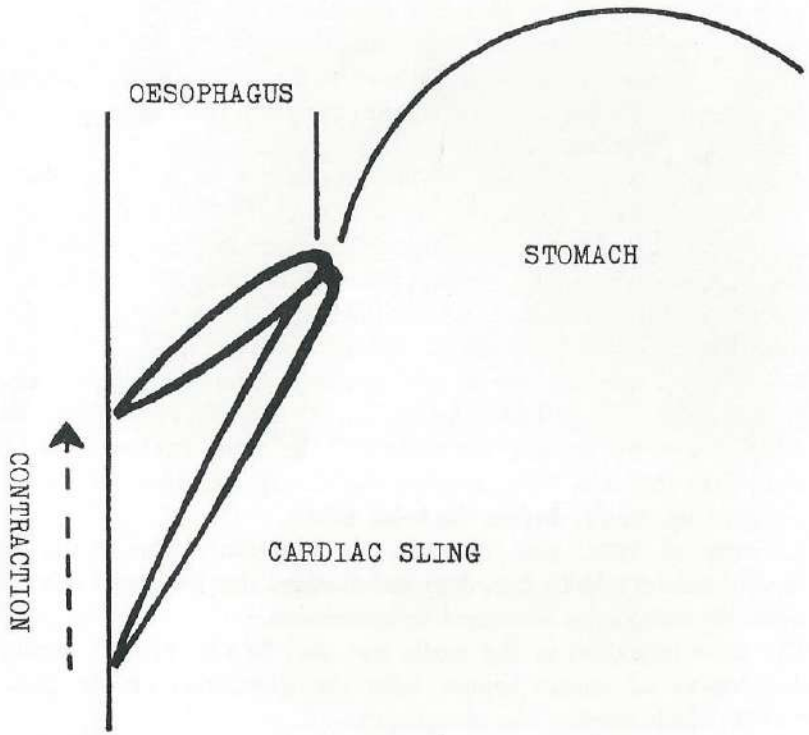
The same relaxation of the cardia was seen by KAY (1951) during thoracotomy on human beings, following stimulation of the vagal nerves, which shortens the oesophagus.

It ensues that lengthwise contraction of the oesophagus coincides with relaxation of the cardia.

O'MULLANE (1954) observed (during general anaesthesia) that when the cardia was retracted orally by means of a small intragastric balloon, connected to double-lumen catheter, reflux occurred, implying relaxation of the cardia.

By roentgencinematographic procedures, it was established by NAUTA (1955) that after swallowing, propulsion of the bolus is mainly handled by lengthwise contraction of the oesophagus. The oesophagus is well adapted to lengthwise contraction by its strong longitudinal muscular coat. In the lower gullet the inner circular muscle coat, moreover, takes a spiral course and thus reinforces the action of the longitudinal muscles (288, 310).

The longitudinal muscular coat of the oesophagus fans out along the lesser curvature. Moreover the fundic region of the stomach is attached to the diaphragm by the gastrophrenic ligament. Considering these anatomic features it follows that, when the lower oesophagus contracts, the cardia is elevated more on the side of the lesser curvature than on the lateral side and a slight rotation ensues (See the schematic drawing no. XVII). The angle of entry of the oesophagus into the stomach will thus become enlarged. The sling of collar

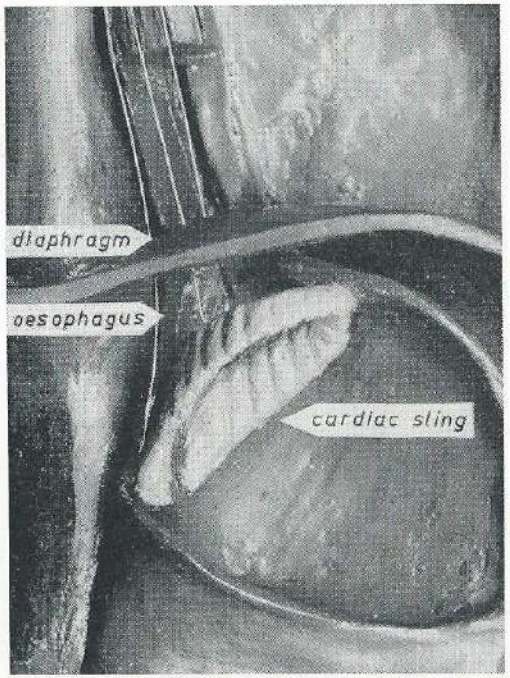


XVII

The effect of contraction of the lower gullet on the length of the cardiac sling

fibres forming the cardiac sphincter, becomes foreshortened by this different angulation and consequently ineffective. On illustration no. XVIII the effect of this elevation of the lesser curvature is evident. To illustrate this point, we marked the cardia of two dogs and four patients with metal pellets and clips. During laparotomy we sutured two metal pellets onto the lesser curvature and one in the cardiac notch without interfering with the various ligaments. During thoracotomy two pellets were sutured to the oesophagus. In addition we marked the hiatus of one dog with three clips. Two weeks after the operation we commenced with the experiments. The dog was

fed minced meat, mixed with some barium paste, without anaesthesia or any praemedication. Some seconds after swallowing, X-ray plates were exposed at an even rate with an interval of ¼ of a second, by means of the SCHÖNANDER AOT film changer. In all, 8 experiments were done. Of these, we got only three times satisfactory exposures of the bolus passing through the cardia as it proved unexpectedly difficult to capture the moment of passage (345, 346). All X-rays exposed on the right moment showed similar results as is evident on

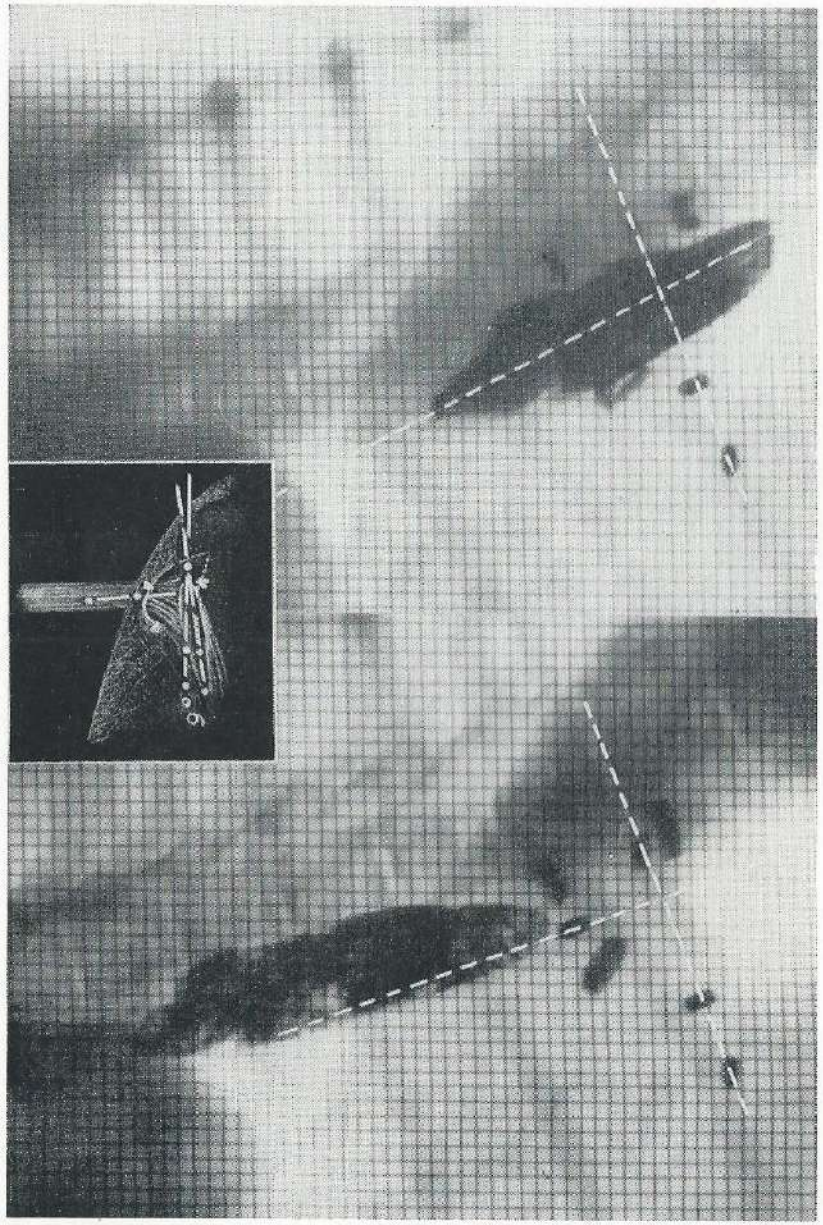


XVIII

Contraction of the lower oesophagus elevates the lesser curvature of the stomach and consequently foreshortens and relaxes the cardiac sling

illustration no. XIX and XX. These X-rays were exposed ¼ of a second after each other. It is clear that during passage of the cardioesphageal junction, the angle of entry of the oesophagus into the stomach enlarges.

Following these experiments, we marked the human cardia in four instances with CUSHING clips, during laparotomy, which was done for cancer of the distal part of the stomach. As these cases were inoperable one clip was sutured near the cardiac notch and one onto the lesser curvature, close to the oesophagus. Care was taken not to interfere with the various ligaments. Two weeks postoperatively one series of



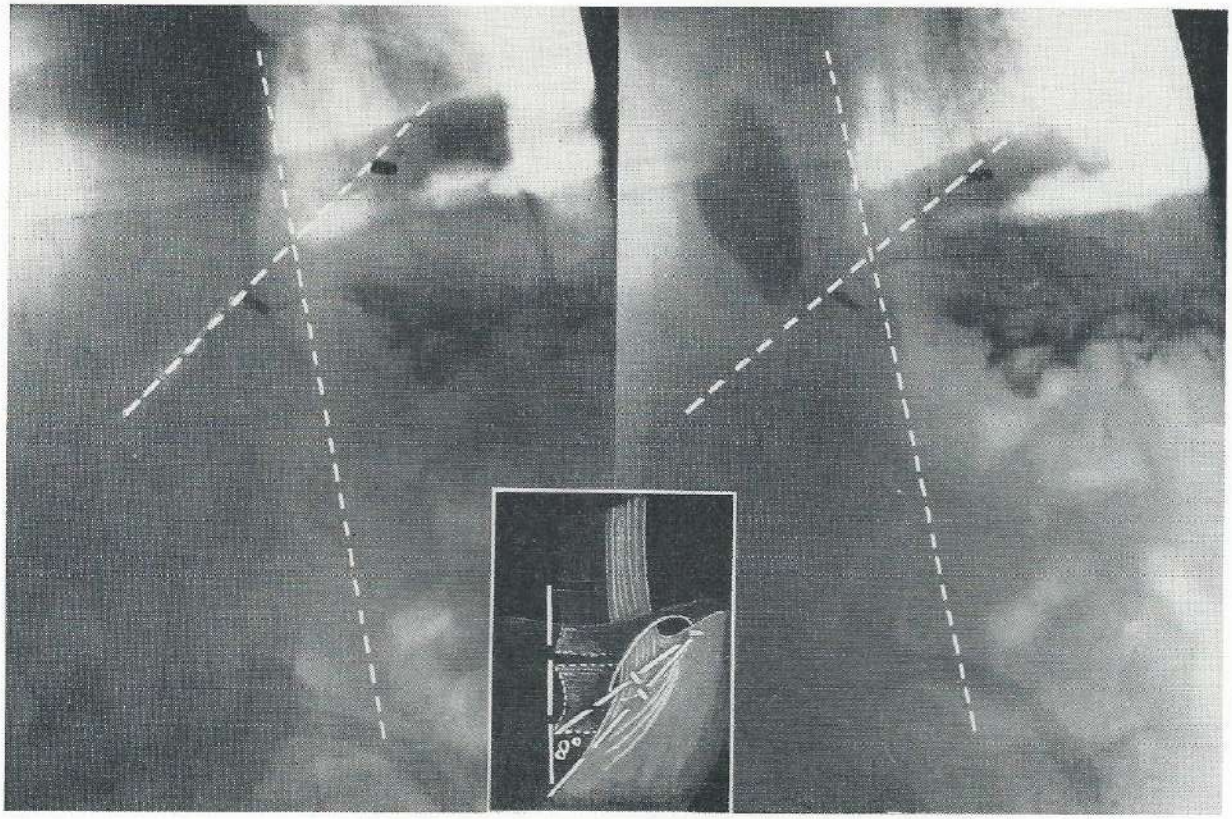
XIX and XX

The angle of entry of the oesophagus into the stomach enlarges 9° during passage of the gastroesophageal junction. Insert shows the position of the metal pellets on the oesophagus and the stomach and the clips on the hiatus

X-rays of each of three patients was obtained by means of the SCHÖNANDER roentgen apparatus. As these patients had been exposed to extensive X-ray examinations prior to operation we confined ourselves to one series alone. Two of them were given a spoonful of a watery barium suspension and the other a piece of bread, soaked in barium suspension. The moment of cardiac relaxation was established in each patient beforehand, during repeated fluoroscopy. It transpired that it was easier to capture this moment when solids were swallowed. In the final experiment, a full series of 30 X-rays was exposed with an interval of ¼ of a second, shortly after swallowing. Illustrations no. XXI and XXII were thus obtained from a 63-year-old male (H.M. 14684). On these it is evident that the angle of entry of the oesophagus is enlarged the moment the bolus passes the cardioesophageal junction. In two other patients we failed to obtain satisfactory results, due to the same difficulty as experienced in the experiments on dogs, namely that of capturing the final stage of deglutition. From the fourth patient, a 66-year-old female (H.M. 13430), we fortunately obtained, during fluoroscopy, an exposure shortly before the bolus arrived at the cardia. (Illustration no. XXIII and XXIV). Here it can be seen that the cardiac angle increases before the bolus has reached the cardioesophageal junction. We took minute care in every instance that the patient did not move between exposures.

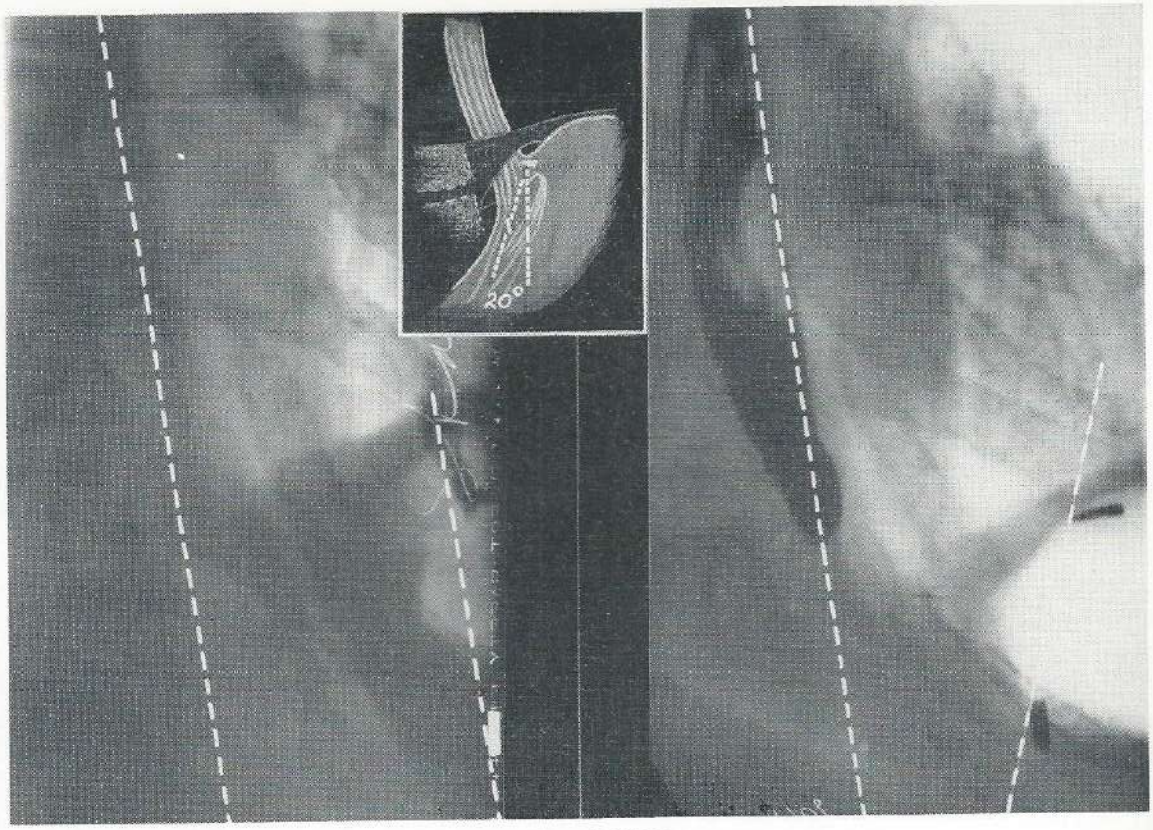
Increasing angulation of the cardia was only seen in the final state of deglutition. Respiratory movements were in no way responsible, as could be checked from the same series of plates. During respiration the cardia only moves up and down.

These experiments confirm our hypothesis, that foreshortening of the cardiac sling by oesophageal contraction is responsible for the relaxation of the cardiac closing mechanism.



XXI and XXII

The angle of entry of the oesophagus into the stomach enlarges 8° during passage of the gastroesophageal junction. Insert shows the position of the CUSHING *clips on the stomach*



XXIII and XXIV

The angle of entry of the oesophagus into the stomach enlarges 20° before the bolus arrives at the cardia. Insert shows the position of the CUSHING *clips on the stomach*

Chapter X

MECHANISM OF REFLUX

In chapter VIII it was seen that reflux is the main cause of the disease. Once a free communication between stomach and oesophagus has been established, gastric contents reach the oesophageal mucosa in the horizontal or head-down position. In addition to gravity, gastric contents is sucked into the gullet by the negative intrathoracic pressure and this pressure gradient rises when straining, lifting weights etc. FRY (1952) showed that pressure changes in the gullet are quantitive reflections of intrathoracic pressure changes. During normal respiration the pressure change in the oesophagus is approximately 15 cm of water, as CODE (1958) recently accurately determined. Similar findings were reported by DORNHORST (1954), CREAMER (1955), FYKE (1956), ATKINSON (1957) and CROSS (1958).

When gravity and sucking co-operate as for instance in the supine position, a larger segment of the gullet should become involved. But the curious fact is that usually only the terminal end of the gullet is inflamed. The proximal part of the gullet, however, is not less susceptible than the terminal end, as has been proved in experiments on dogs and cats (Chapter VIII). A difference in susceptibility of the human gullet, has never been observed either.

The actual reason why the terminal part is only diseased, we consider to be twofold. Firstly: in the supine position the proximal part of the oesophagus slopes upwards. This is increased by the elevation of head and shoulders which is the common sleeping position. In the upright position, of course, the proximal part is rarely bathed due to the pressure gradient involved. Secondly as soon as the lower part of the oesophagus is flooded, a wave of peristalsis commences high up in the chest in response to the distension of the lower gullet and sweeps the contents downwards into the stomach (118, 142). The oesophagus is thought by some to be incapable of antiperistalsis (117), the gastric contents consequently rarely reaches the upper limits of the oesophagus.

Oesophagitis, which particularly complicates oesophagogastrostomy done for achalasia, may well be due to the absence of this latter mechanism (Chapter VII).

This second factor holds good provided that the oesophageal muscular wall is not inflamed. When inflamed these muscles do not function properly and, in this way, inflammation may spread orally. Probably in this manner, the so-called ascending fibrosis originates. The muscular wall of the oesophagus, however, has a strong resistance to the spreading of the inflammation from the mucosa and submucosa (373).

In addition to the above, a further factor is spasm, which is ". . . . nature's way of protecting the proximal oesophagus from further excoriation" (BARRETT 1955).

In the preceding chapter it has been expounded that the cardiac sphincter relaxes when the oesophageal muscles contract and the oesophagus consequently shortens. This may explain why the open cardia, made evident by reflux, is so common in the sliding diaphragmatic hernia with a short oesophagus. This condition is nearly always acquired (8, 42, 95, 237, 240, 356, 373, 447). It may be deduced from the fact that the condition is generally reversible (361, 374, 399, 511) by simple traction on the cardiac end during operation (11, 171), and that cases are known where spontaneous reduction has occurred (36, 398, 506). Remarkable in this respect is the report of FISHER (1957) concerning 9 cases of short oesophagus and oesophagitis. Following treatment of the peptic ulcerations, the hernia disappeared altogether.

A congenitally short oesophagus, so-called brachy-oesophagus, is rare, contrary to former belief. In extensive series of diaphragmatic hernias, HARRINGTON (1955) encountered the entity in less than 5%, SWEET (1953) in 4%. In our series we encountered none. It should be pointed out that in some cases where reduction of the cardia into the abdominal cavity is impossible, this may be due to inflammatory fibrosis of the oesophageal wall and the surrounding tissues (8, 237, 373, 447).

This shortening is thought by some authors to be due to elastic recoil of the oesophagus (11). This recoil is believed to become effective when the phreno-oesophageal and gastro-phrenical ligaments are weakened, as a result of old age for instance and a wide hiatus is formed in some way (19, 402). If this were true, why is reflux not found in every case of hiatus hernia? In large hernias a wide hiatus usually exists and the ligaments mentioned are undoubtedly slack, but in these cases the cardia is usually competent. HARRINGTON and other authors (296, 349, 377) consider the hiatal hernia to be essentially a congenital condition. This makes the concept of passive elastic recoil improbable as then the condition should always be found at birth or shortly afterwards.

It must be deduced that shortening of the oesophagus is caused

by contraction of the muscular wall (80, 167, 188, 210, 237). Contraction of the muscles may be induced firstly: by stimulation of the vagal nerves or their corresponding cerebral centra (16, 348), secondly: by stimulation of the muscles.

Stimulation of the vagal nerves is a common occurrence in hiatal hernias, according to HARRINGTON. The syndrome of retrosternal pain, heartburn, referred pain in the neck and the ears and salivation can indeed, in the absence of oesophagitis, be best explained by vagal irritation (51, 276, 308, 413). Vagal irritation, in this instance, might be due firstly: to the pinching of the hiatal muscular sling (51, 210), secondly: to distension of the herniated pouch, which is caused by the negative intrathoracic pressure and obstructed communication between the herniated pouch and the rest of the stomach. This obstruction was convincingly demonstrated by AYLWIN (1953). A wide hiatus in the case of a hernia may thus be considered a blessing and may partly explain the absence of symptons in large hernias. In this case stimuli, received from pressoreceptors (242), stimulate the efferent fibres of the vagal nerves by reflex.

Irritant foci in the upper abdomen may in the same way cause shortening of the oesophagus by reflex (WILSON 1950, BENEDICT 1954). Thus can be explained the coincidence of a gastric or duodenal ulcer or cholecystopathia with oesophagitis. This was confirmed by HILLEMAND (1951) and others (185), who frequently observed spastic contractions of the oesophagus in the presence of peptic ulcer or cholecystopathia.

Before HILLEMAND, it was observed by JOHNSTONE in 1946 that an irritable focus caused contraction of the oesophagus and that regurgitation resulted.

It appears that laparotomy may fall into the same catagory. SMITHERS (1950) remarked that "oesophagitis, which sometimes follows abdominal operations may result from reflex contractions of the oesophagus". REDISH (1951) confirmed that stimulation of the peritoneum is followed by oesophageal contraction.

In 1946 GILBERT and DEY proved in dogs that by stimulation of the vagus, the oesophagus shortens; the same shortening occurred when scratching the peritoneum, manipulating the liver, distending the gallbladder or cystic duct and manual stretching of the stomach wall. This reflex was abolished by vagal section.

Apart from this vago-vagal reflex, oesophageal shortening results after direct stimulation of the vagal cerebral nuclei. Higher centra are also involved and for instance stimulation of the fissura SYLVII and insula REILLII are known to cause oesophageal shortening (275). It was demonstrated by FERGUSON (1950) that following insertion of an inflated balloon in the temporal region of a cat, oesophagitis

resulted. Oesophagitis, which originates shortly after endocranial operations or during acute affections of the brain or its covering membranes, may be hereby explained.

Once the oesophageal mucosa is excoriated and inflammation spreads, the muscular wall of the oesophagus subsequently contracts (6, 95, 151, 171, 326, 506). Thus evolves a vicious circle (32, 424). Oesophagitis shortens the oesophagus, which in its turn causes reflux, which again maintains the inflammation.

The oesophagus has a great tendency to recovery, but once inflammation has settled in the lower part, a chronic course may develop due to this vicious circle. By this vicious circle inflammation, originally due to another cause, may persist. For instance prolonged vomiting may induce this. Laceration of the oesophagus due to prolonged vomiting may easily occur. It may even lead to the MALLORY WEISS syndrome (131, 302, 430, 499) and rupture of the organ.

In due course when prolonged inflammation has produced fibrosis, a more or less permanent condition is established. The cardia has then become permanently incontinent (37, 95, 240).

It was seen in chapter VIII that not in all cases, where reflux is present, inflammation subsequently results. One additional and predisposing factor is a diminished buffering capacity of the oesophageal secretions following vagal irritation. When the two coincide the chances of inflammation are increased.

From these observations it may be concluded that stimulation of the vagal nerves or its cerebral centra plays an important role in the development of the disease.

In our series we considered the cause of the disease to be due to

oesophageal hiatal hernia in	40 cases.
following cardioplasty	6
following proximal gastric resection	4
following total gastrectomy	2

In 3 cases oesophagitis developed following subtotal gastrectomy (BILLROTH).

In 4 cases the disease developed during pregnancy. In 2 the aetiology could not be determined.

BOOK III

Chapter XI

MEDICAL TREATMENT

Medical treatment rarely effects a permanent cure, but is a valued adjuvant to surgery.

Medical treatment of oesophagitis has not been found very effective in the long run (28, 53, 87, 121, 149, 152, 156, 240, 417). If oesophagitis is the complication of some other disease, its cure may be effected by treating this disease. For example, medical regimen for duodenal ulcer was found to be beneficial for oesophagitis as well (26, 487, 505, 506). Because of this success, it has also been applied to patients without duodenal ulcers (7, 40, 45, 95, 102, 121, 219). Usually they benefit at first, but the disease is not cured in the majority of the cases.

Antacids, which cover the mucosa with a thin film, may be used to advantage (26). Tablets of, for instance, aluminum hydroxyde gel and magnesium trisilicate, which should be sucked the greater part of the day, cover the mucosa with an alkaline film and this protects it from gastric juice.

When the oesophagitis is severe, a bland, liquid diet is usually preferred and is often the only one tolerated (45, 95, 102). Spices and spirits are better avoided (45). To minimize reflux, the last meal of the day should be taken more than 4 hours before the patient retires. The patient should be advised to elevate the head-end of the bed and sleep propped up in a semi-sitting position (7, 26, 40, 95, 102, 121, 171, 219, 447). In the case of babies and small children, who spent the greater part of the day in bed, it becomes imperative to keep them in a sitting position. To effect this, it is advisable to construct a harness by which they can be kept in this position (49, 94).

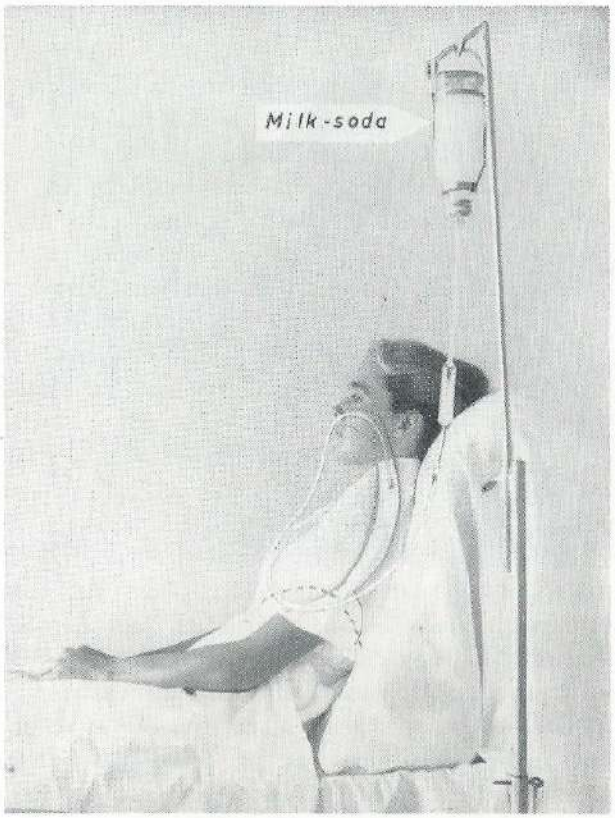
The common antispasmodics are usually of no avail in relieving spasm. Although amyl-nitrite and amphetamine have some effect, they are only short acting (463). In view of the aetiology (chapter X), one could suppose that belladonna, atropine and similar agents would relieve the disease and indeed, with its use, some symptoms disappear (5, 7, 28, 95, 102, 506). But a drawback is its retarding

action on the motility of the stomach, which increases the residual contents, by which again reflux into the oesophagus is intensified. Atropine also causes decreasing tonus of the gastric muscles (21), theoretically thereby diminishing the sphincteric action of the collar fibres. In cases, which originate shortly after subtotal or total gastrectomy, however, the use of atropine and similar agents may be considered.

When a patient's condition becomes critical, due to either malnutrition, dehydration or haemorrhage, foreclosing an immediate operation, imminent death can be prevented by a continuous, intra-oesophageal drip of milk-soda (114). By this procedure, favoured by WINKELSTEIN (1954), a tube is introduced into the proximal part of the gullet and through this a mixture composed of equal parts of milk and a solution of 0.15% $Ca(OH)_2$ flows at the rate of 4 to 6 litres per day, for adults. The gullet is thus continuously bathed by this strongly alkaline solution. Naturally the patient should be nursed in a sitting position in bed and no oral intake, other than alkalies, should be allowed as this tends to interfere with the protective film of milk-soda. This therapy can be continued until the oesophageal ulcerations have healed, which, depending of course on the severity may last from 1 to 2 weeks. Dramatic improvement has been achieved in this manner in only a few days time. Illustrative is the following case. It concerns a 74-year-old woman (H.M. 13172) with severe oesophagitis. Repeated haemorrhage caused her condition to deteriorate rapidly. Daily blood transfusions proved to be of no avail. The severity of her condition rendered all operative procedures including oesophagoscopy, impossible. We applied the above described therapy. In addition she was given a spoonful of "ALUMINOX", a gel of aluminum hydroxyde 6 times per day. She improved rapidly and after two days the haemorrhage ceased. She was up in a week's time and was discharged in good condition 4 weeks later. Her condition is excellent after more than a year, although recent fluoroscopy has revealed abundant reflux. She still sleeps in a semisitting position.

Another case, suffering from obstruction in addition, a child, aged 18 months (H.M. 13501), was admitted in a deplorable condition. Her condition precluded any operation. The same therapy was instituted in smaller doses. To keep her in a sitting position, a harness was constructed. In view of the existing total obstruction, overflow of sodamilk was feared. To prevent this, a tube was introduced into the gullet directly above the stenosis, to which continuous suction was applied. After 48 hours it became apparent that some of the fluid had passed the stenosis. This was indicated by the difference between the amount of fluid introduced through the drip and that

recovered by suction. Freer passage soon became evident and after 4 days we discontinued suction. The child could soon be fed by mouth. She improved rapidly and was fit for radical operation in 30 days. These striking results have led us to employ this procedure preoperatively in every severe case. On illustrations nos XXV and



XXV

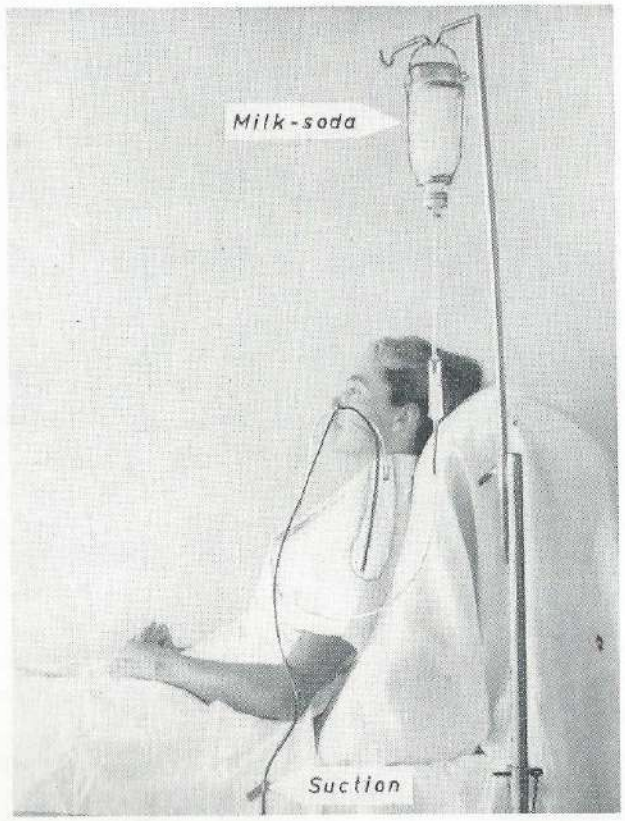
Semisitting position of the patient and continuous intra-oesophageal drip of sodamilk. Note the location of the nasal tube in the proximal part of the gullet

XXVI, the position of the patient and the location of the nasal tubes can be seen.

Operation on a highly inflamed oesophagus may prove a hazardous undertaking. For instance inflammation may prevent the repair of hiatal hernia by its adherence to the surrounding tissues. When attempting reduction of the hernia, the oesophagus may tear due to the friability of the tissues. It is an obvious advantage when the inflammation can be cleared preoperatively.

As seen in chapter X, inflammation of the lower gullet causes

contraction of the oesophagus, by which the cardia is raised into the thorax. In our opinion recurrence after repair of hiatal hernia, which coincides with oesophagitis, may be largely due to this vicious circle. Therefore if oesophagitis is cured temporarily before hiatal hernia repair, the possibility of recurrence is greatly diminished.



XXVI

In case of total obstruction, overflow of sodamilk is prevented by suction on an additional tube (black), directly above the stenosis

Chapter XII

SURGICAL TREATMENT

Surgical treatment should be instituted at an early stage.
Restoration of the cardiac function should be the ultimate goal.
Dilatation of an inflammatory stricture is inefficient treatment when reflux persists.
A stricture should be resected, whenever possible.

A wide diversity of operations have been advocated in the treatment of peptic oesophagitis. We do not intend to discuss them all fully. Some of these operations are major procedures, which are disproportionate to the benign nature of the disease. Others are minor operations, often inefficient. Therefore surgical treatment is usually deferred until all other therapies have failed.

We feel, however, that surgical treatment should be commenced at an early stage of the disease, before complications render it difficult (96, 374). Only then can we hope to obtain restitutio ad integrim (204).

Surgical treatment for uncomplicated oesophagitis falls into two groups. The one aims at diminishing production of acid gastric juice and the other at counteracting reflux.

SUBTOTAL GASTRECTOMY

Local treatment of an oesophageal ulcer by means of endoscopy with silver nitrate, chromic acid or cautery, has proved a failure (6, 7). WANGENSTEEN (1949) and CORNELL (1950) were the first to realize the practical value of subtotal gastrectomy. By this procedure the production of acid gastric juice is greatly diminished and the stomach contents rapidly evacuated; the latter being of obvious importance (87, 121, 295, 304, 352, 501). The effectiveness of this procedure was pointed out by MACLEAN and WANGENSTEEN (1956) in a follow-up study of 19 patients. (It should be remarked, however, that 11 of these cases were not ordinary gastric resections in the Billroth manner.)

There is no unanimous agreement about its effectiveness however, and failures are not uncommon. This is not surprising as after subtotal gastrectomy, gastric remnants are known to produce gastric juice in quantity. Besides, oesophagitis has been known to commence directly after this procedure thus disproving it as an ultimate therapy

(3, 48, 272, 399, 452). We feel that the operation has its place in those cases where oesophagitis coincides with gastric or duodenal ulcer.

WELLS (1955), who is convinced that oesophagitis following subtotal gastrectomy is due to reflux of bile and pancreatic juice, hopes to prevent this by the following modification. Instead of the end-to-side anastomosis of stomach and jejunum, as performed in the Billroth II procedure, the anastomosis is carried out by means of a ROUX in Y loop. Vagotomy is performed in addition.

TOTAL GASTRECTOMY

After total gastrectomy the oesophagus is spared further contact with acid gastric juice (28, 294). We have performed total gastrectomy in 3 cases; one case with exsanguinating haemorrhage, another with ascending fibrosis of the oesophagus following subtotal gastrectomy and a third with oesophageal ulcers following a GRÖNDAHL procedure for achalasia. The first of these cases is still in good health, 4 years postoperatively. The latter two developed strictures of the anastomoses subsequent to the operation. The first of these required repeated dilatations and the other resection of the stricture. The former is now in good health 3½ years after the last operation. The latter's secondary operation was only recently performed.

Total gastrectomy is rarely indicated as the magnitude of the operation is usually disproportionate to the nature of the disease. Moreover oesophagitis has been known to originate shortly after total gastrectomy (90, 95, 294). See chapter VIII.

VAGOTOMY (57)

Vagotomy, performed either transabdominally or transthoracically was advocated by some authors (115, 295, 505). It is definitely known that the amount and the acidity of the gastric juice decreases following vagotomy (57, 145, 146, 217). Simultaneously, however, the gastric motility decreases (21, 77, 217), thereby increasing the residual contents of the stomach, which again intensifies transcardial reflux (161, 184, 241, 265, 441, 473). This effect of vagotomy may be counteracted by additional pyloroplasty or gastro-enterostomy, which provide better drainage of the stomach (295). In the long run results have been disappointing, however (31, 175, 325, 512). This may be due to the fact that, in the majority of cases, the quality and quantity of gastric juice return after 1—3 years to the approximate level, prior to vagotomy (354).

PYLOROPLASTY

Pyloroplasty alone has been advocated by BURFORD (1956) in cases where oesophagitis is due to an irreducible hiatal hernia with a short oesophagus. This simple procedure (FINNEY, 170) provides rapid drainage of the gastric contents into the duodenum. The little remaining gastric juice is of course sucked into the thorax. During normal respiration the pressure changes do not exceed 15 cm of water, therefore when the stomach is raised into the thorax for more

than 15 cm the oesophagus which lies above this keeps clear of gastric juice as long as the patient remains in the upright position.

REPAIR CARDIAC MECHANISM

Operative procedures, designed to counteract reflux, comprise those which aim at repairing the cardiac closing mechanism and those which aim at substituting the mechanism when the cardia is destroyed. BARRETT, who is convinced that the cardia is solely a valve, restored the mechanism by insuring a deep cardiac notch. This is done during laparotomy by suturing the lateral wall of the oesophagus to the medial wall of the fundus of the stomach.

This simple procedure has been employed when no hiatal hernia exists, but the cardia is nevertheless incompetent, as for instance in the condition known as "chalasia" (BERHENBERG 1950, ALLEN 1950, BERK 1945) or "Malposition cardio-tubérositaire" (LORTAT-JACOB 1957, DUHAMEL 1953). Such a case was reported by STENSRUD (1957), where all evidence of reflux disappeared after this procedure.

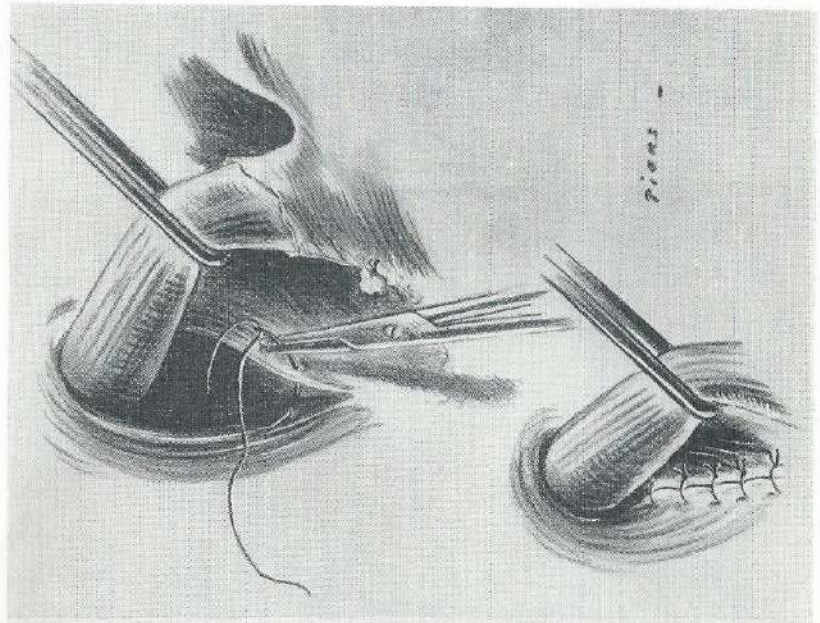
When reflux is due to a hiatal hernia, general opinion holds that the cardiac function is restored by the hernia repair. If some degree of oesophagitis is present, the recurrence rate is high, however (64). Various operations, transthoracic and transabdominal, have been devised to prevent this. It does not lie in the scope of this treatise to comment on all of them. In most procedures the main purpose is to reduce the size of the hiatus. In many cases of sliding hernia, the hiatus is hardly enlarged, however, and in these cases reduction in plainly not the solution. Realizing this, many surgeons aim at repairing or reinforcing the ligaments around the hiatus. It can be imagined, however, that the rather weak structures involved, which moreover are always moving, cannot resist this continuous traction. When oesophagitis is present, spastic contraction of the oesophagus, due to the inflammation, is probably responsible for the recurrence. In chapter X it was pointed out that in the case of oesophagitis a vicious circle is established and the upwards traction thus persists. In the same chapter it was demonstrated that the upwards traction of the oesophagus causes incompetence of the cardia. Therefore although the anatomy is temporarily restored by the operation, the cardiac function is not (64, 511).

For these reasons BOEREMA (1955) developed his „gastropexia geniculata anterior". Except for being an efficient method in hernia repair the favourable results obtained by this procedure are due to the fact that the cardiac sphincter is restored to its normal action. As this constitutes the main point in the surgical treatment of oesophagitis, a description of the different stages of this operation seems justified. It is an abdominal operation, which has the advantage that irritative foci, for instance gastric duodenal ulcers and cholecystopathia, can be eliminated at the same time. An upper abdominal incision is

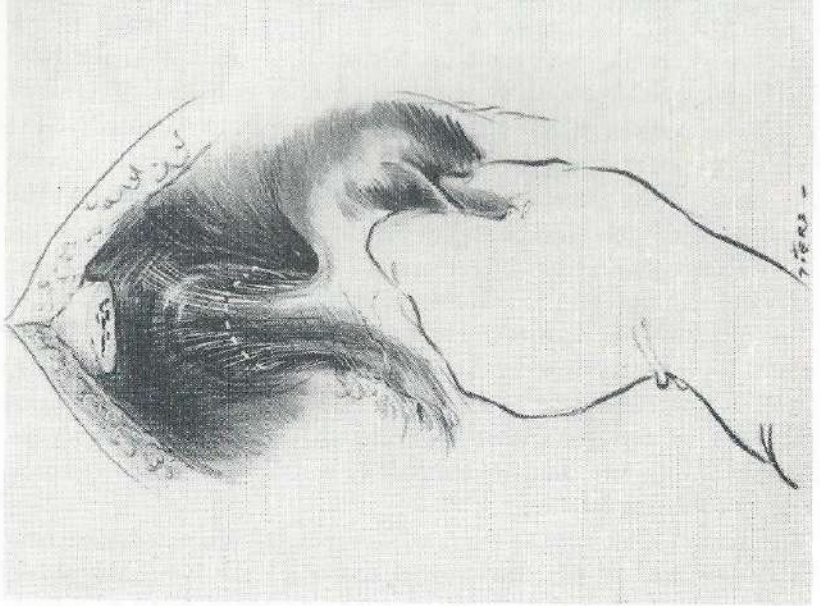
made from the navel to the sternum. The xiphoid process is removed. The diagnosis may now be verified and the abdominal cavity explored for additional pathology. The coronary ligament of the left lobe of the liver is severed and the lobe is folded back onto the right lobe. The hernia can be reduced by traction on the stomach. By maintaining this traction the now taut peritoneum is incised transversally on the gullet. (Illustration XXVII). Usually the left gastric artery must be ligated and severed to obtain access to the hiatal orifice. The enlarged hiatus is then reduced by approximating the crura by silk sutures behind the oesophagus (Illustration XXVIII). Care should be taken to leave sufficient room for the bolus to pass. No reduction is attempted when the hiatus is not markedly enlarged or when access to it is difficult, as we feel that it is not essential in the case of sliding hernias. Hereafter the left lobe of the liver is replaced. The essential part of the operation now follows. By traction on the lesser curvature of the stomach a small part of the oesophagus is pulled into the abdomen behind the left lobe of the liver. In this position the lesser curvature is sutured to the anterior abdominal wall (Illustration XXIX).

In our opinion the favourable result of this operation is due to this fixation of the lesser curvature to the anterior abdominal wall. This prevents the cardia from sliding back into the thorax and more important still, the cardiac sling is restored to its original length by traction on the lesser curvature and thus may resume its function. Following operation a slight dysphagia may be expected, which may be due to oedema of the hiatal muscle sling. When localized at the cardia, the obstruction may be the consequence of the fixation of the cardia to the anterior abdominal wall. The cardia can only be slightly raised, thus hampering the final stage of deglutition. When dysphagia is due to the former cause it usually disappears in a few days time. In the latter case it may occasionally last for a few months. This operation has been performed in 17 cases in our series with one death. It proved a failure in three instances. These cases (H.M. 10181, H.M. 14028, H.M. 13172) suffered from severe oesophagitis and from the onset we had grave doubts whether the cardia would resume its function. They were long-standing cases where inflammatory fibrosis was probably responsible.

In one case (H.M. 12513) the cardio-oesophageal junction had become so friable that on the first attempt to reduce the herniated gastric pouch, during laparotomy, the oesophagus was completely torn, necessitating another operation. In all other cases, who have been followed-up at regular intervals, there has been no recurrence from minimally 3 months to maximally 4 years postoperatively.

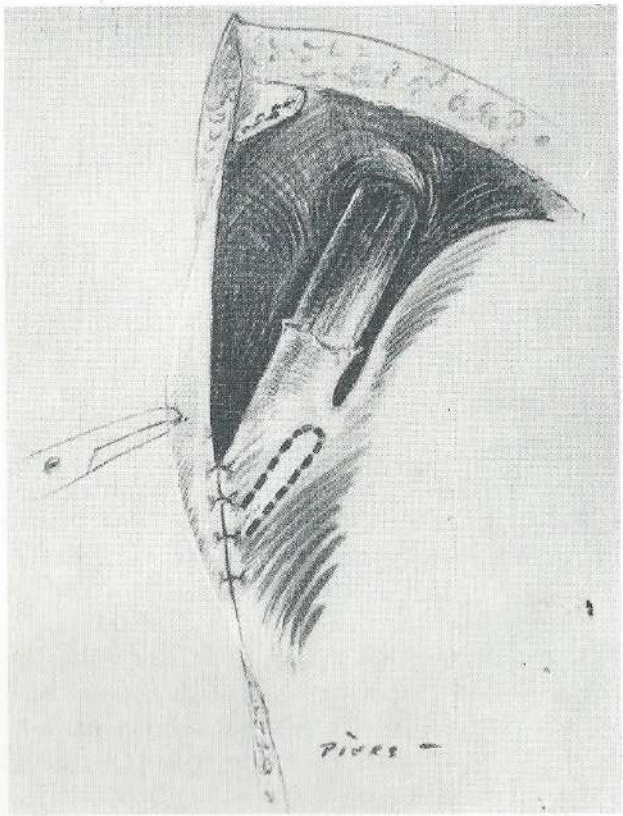


XXVIII
The crura of the diaphragm are approximated by silk sutures behind the oesophagus



XXVII
The stomach is retracted downwards and the taut peritoneum on the oesophagus is incised transversally

When an oesophageal stricture, due to chronic inflammation, causes malnutrition, something has to be done. These strictures can be dilated by blind bouginage, which is a hazardous procedure, however (7, 44, 400). Perforation, which is usually lethal, frequently occurs. By JACKSON, PLUMMER'S method (1, 45), a bougie or dilator is introduced over a previously swallowed string (121) which leads the



XXIX
The lesser curvature of the stomach is sutured to the anterior abdominal wall. The broken line indicates the cardiac sling

way for the bougie. It has been advocated by PORTMANN (1931) and others (487) to exteriorate the string through a gastrostomy. When the ends are tied, this endless string can be used indefinitely. Dilatation of the stricture through an endoscope, which is undoubtedly the safest procedure, has the disadvantage that endoscopy is an unpleasant experience, endured unwillingly at regular intervals.

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[1]) 1, 3, 4, 28, 29, 42, 44, 47, 48, 151, 152, 176.

Dilatation has only met with temporary success, however, and is only possible in the case of a short stricture. When the disease is allowed to persist, reflux following dilatation is again established and recurrence of the stricture is sure to occur. According to BROWN-KELLY (1953): "When age, general condition or economic factors preclude surgery, the bougie has its place."

WANGENSTEEN (1949, 1956) discovered that following subtotal gastrectomy dilatation was seldom necessary more than a few times. This procedure diminishes the amount and acidity of gastric juice and provides rapid drainage of the gastric contents, by which little or no reflux occurs.

RESECTION

Resection of the oesophageal stricture is usually preferable. EGGERS (1935) performed this for the first time. With resection of the stricture, which usually includes the cardia, a procedure should be employed which limits reflux at the same time, except when submucous resection of the stricture is possible as in the procedure of ALLISON (1956). In that case it can be hoped that the cardia will resume its function. Oesophagoplasty and cardioplasty as advocated by SWEET (1954) and others (357, 358, 510), are lacking in this respect and provide only temporary relief. Every procedure which lacks the essential feature of limiting reflux should be condemned.

Solution has been sought in the construction of an artificial valve. (WATKINS (1954) and DILLARD (1954)).

Hitherto we do not consider these attempts to be successful as these valves are partly composed of oesophageal elements, not immune to gastric juice.

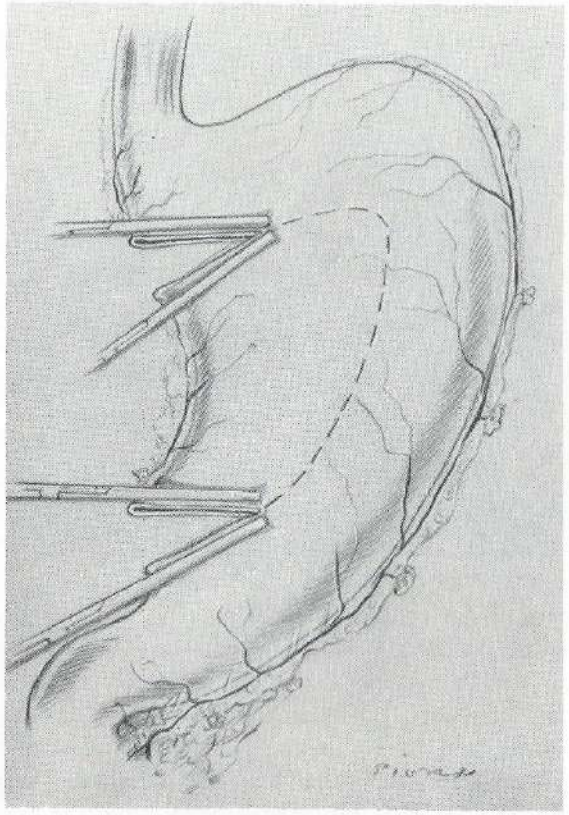
ALLISON (1949) originally sought to circumvent this problem by closing the cardia and connecting the oesophagus to the jejunum, by the procedure of ROUX-Y. He discontinued the practice because these patients developed anaemia and malnutrition (15, 29, 296). Lately ALLISON (1957) propagates oesophago-jejuno-gastrostomy. In this procedure a segment of jejunum is interposed between the oesophagus and the lower back surface of the stomach. This jejunal loop is anastomosed end-to-end to the oesophagus and obliquely to the posterior side of the stomach. The cardia is closed and pyloromyotomy is performed in addition. The vagal nerves are preserved if possible. According to the author the operation is tedious and caution should be taken because mistakes are easily made. Interposition of the jejunal segment was previously advised by other authors (121, 211, 438). It has been proved in experiments on dogs that it indeed prevents reflux of gastric juice into the oesophagus (438, 439).

The interposed jejunal loop is a substitute cardia on the theory that its peristalsis prevents the gastric juice from reaching the oesophagus (29, 121, 322). The acidity is somewhat neutralized at the same time

(124, 267). This practice is based on the fact that the small gut is more resistant to the action of the gastric juice than the oesophagus although it is not immune however (124, 126, 296, 317). In experiments on dogs ulcers indeed developed in these jejunal loops (397, 439). WOOLLER (1957) claimed that no jejunal ulcers developed in his cases, nor in the cases of ALLISON. These authors consider the entrance of the jejunal loop into the back of the stomach to have a valve-like action (15, 513). A great drawback of the procedure is a high incidence of necrosis of the proximal part of the transplanted jejunal segment, causing a high mortality rate (12, 29, 436). Therefore interposition of a segment of colon has been attempted which has not proved successful however (181, 249, 332, 422, 438). Although less chance of interference of the bloodsupply assures a safer anastomosis, the interposed segment does not adequately drain its contents into the stomach. Transplantation of the valvula BAUHINI has not met with success either (NISSEN 1955).

ELLIS (1954, 1956) advocates subtotal gastrectomy following resection of the stricture which may be more effective than WANGENSTEEN'S practice of subtotal gastrectomy, combined with dilatation. It has however a similar shortcoming. In the published data of the experiments it transpires that some dogs still developed oesophagitis (156, 352). This is not surprising as reflux is still possible. A small quantity of gastric juice as well as duodenal contents still damages the oesophageal lining. Considering the mechanism of reflux, one of the few operations which successfully prevents oesophagitis once the cardia has been resected, is that of BOEREMA (1951). A description of the various stages of the procedure seems therefore justified. It is a thoraco-abdominal procedure with separate incisions in the abdomen and thorax. In the supine position an incision is made in the upper abdomen and the xiphoid process is removed. The coronary ligament of the left lobe of the liver is severed and the lobe is folded back onto the right lobe. The left gastric artery is ligated and severed and the cardia and abdominal oesophagus are dissected free. The stomach is then freely mobilized by cutting through the lesser omentum and greater omentum with preservation of respectively the right gastric artery and the vascular epiploic arch up to the fundic region. Clamps are now applied parallel to the greater curvature and others at an angle to these, approximately parallel to the fundus. See illustration XXX. The triangular gastric segment embraced by these clamps, comprising the lesser curvature is excised. The cut margin is stitched with interrupted through-and-through sutures of catgut. A second layer consisting of interrupted seromuscular silk sutures invaginates the former layer. In this way a long tube is constructed. Finally pyloroplasty is performed and the incision is

closed. The patient is then turned onto his right side. Left thoracotomy through the periostal bed of the 6th rib follows. Both lobes of the lung are retracted forwards. The mediastinal pleura is incised, well forwards to the oesophagus to insure a pleural flap which is needed in the final stage to cover the anastomosis. The oesophagus

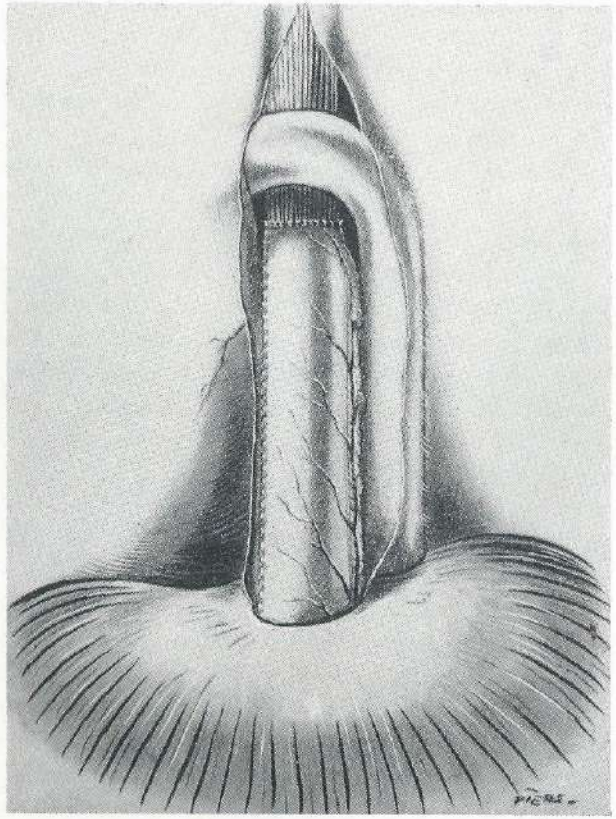


XXX

Clamps applied parallel to the greater curvature and fundus of the stomach. The triangular gastric segment embraced by these clamps, comprising the lesser curvature, is resected

is dissected free from the hiatal orifice to the aertic arch. The previously constructed tubular stomach is drawn up into the thorax through the hiatus. The oesophagus is then transsected close to the aortic arch. End-to-end anastomosis in two layers is then performed. No special technique is nesessary (381). The distal oesophageal segment and the excess of the tubular stomach are resected. Shortly before the anastomosis is completed a RYLE's tube is drawn through and introduced into the stomach. The stomach may be anchored

to the hiatus by a few silk sutures. The mediastinum is closed by approximating the cut margins of the pleura with nylon sutures. Antibiotics may be locally applied to the site of the anastomosis. The incision is finally closed after suction drainage of the pleural cavity is provided through a separate stabwound.



XXXI

End-to-end anastomosis of the oesophagus and tubular stomach close to the aortic arch

This operation, although a major one, is a comparatively safe procedure as the stomach has an abundant bloodsupply (206) According to LAYNE (1943) and EMERSON (1953) one of the four arteries is sufficient to supply the entire stomach.

Pyloroplasty is an essential part of the procedure (352). It ensures rapid drainage of the stomach and is especially important because the vagal nerves are severed. Another essential feature is the site of the anastomosis high up in the chest. Favourable results of this were noticed by BELSEY (1953) and others (32, 163, 476, 504).

As long as the stomach is empty, juices are derived from below the diaphragm. They are sucked into the thorax by the negative intrathoracic pressure. If the lower part of this viscus consists of stomach, the oesophagus is not affected as the pressure changes involved do not exceed 15 cm of water usually (Chapter X). After the operation the patient should be advised to elevate the head-end of the bed thus preventing these juices from reaching the oesophagus at night. In addition the patient should be discouraged from eating shortly before retiring. This operation has been performed in 6 cases in our series. The follow-up is from minimally 6 months to maximally 6 years. All patients are in good health. There is one recurrence. It concerned a case where the anastomosis was low in the chest. At the time (1953) the importance of anastomoses high in the chest was not fully realized.

HAEMORRHAGE

Bleeding, unless exsanguinating, can be usually controlled by blood transfusion and confining the patient to bed. In profound cases a continuous intra-oesophageal drip of milk-soda (see chapter XI) may be employed in addition. If this method fails, tamponade may be considered in severe cases. CASTLETON (1948) reported a case where haemorrhage was controlled by local application of oxycel by means of endoscopy. Primary resection of the lower oesophagus should rarely be performed as disastrous results may be expected.

PERFORATION

Perforation of an oesophageal ulcer is generally lethal and surgical treatment is the only hope (444). Drainage of the cavity in which the ulcer has perforated, is usually indicated (234). In addition intraluminal continuous suction drainage of the oesophagus was advocated by FISCHER (1957). Attempts at closing the defect in the oesophagus usually fail, due to the extensive inflammatory reaction of the surrounding area, in contrast to spontaneous rupture of the oesophagus. In these cases a rubber T-tube may be inserted into the oesophagus through the defect as described by THOREK (1951, 1958). The long end of the tube is extracted through an extra stabwound in the back. The drain is kept in situ until the patient has completely recovered. One patient of THOREK retained this drain for 9 weeks without ill defects.

Of course antibiotics should be freely used. Their effect must not be underestimated. CANTOR (1953) reported a case where recovery was due to treatment by antibiotics.

Chapter XIII

PREVENTION

Oesophageal hiatal hernia is the condition most commonly complicated by oesophagitis. Especially in the case of the sliding type with a short oesophagus, oesophagitis may be expected to develop at any time.

Hiatal hernia repair in the presence of oesophagitis, consisting of reducing the hiatus and reinforcing the ligaments is an insufficient surgical therapy and may prove a hazardous undertaking as well. Therefore repair of every oesophageal hiatal hernia, once its symptoms have become manifest, should be considered before oesophagitis develops. Once inflammation has become established in the lower gullet, a vicious circle exists and recurrence of the hernia is inevitable.

In surgery of the oesophagus and cardia of the stomach, only procedures which efficiently prevent reflux oesophagitis, should be employed. Particularly in the case of achalasia, much harm has been done by oesophago-gastrostomy in the fashion of GRÖNDAHL and HEYROVSKY. These procedures should be condemned. Efficient surgical treatment is profferred by HELLER'S extramucous cardiomyotomy, which is usually performed on the anterior surface of the cardia alone (14, 36, 73, 134, 234, 256, 261, 362, 393, 416, 491). Transcardial reflux rarely results from this procedure. In experiments on dogs by GEEVER (1953) it was seen that no reflux was initiated following a short longitudinal extramucous incision in the cardia. However, long incisions (exceeding 10 cm) invariably made the cardia incompetent. Considering the anatomy of the cardiac sphincter this is not surprising. A long incision transsects the sphincter completely and incompetence results.

The treatment of achalasia according to the procedure of WANGENSTEEN (1951) and a similar one proposed by DELOYERS (1951), seems to be unsuitable. It consists of resection of the cardia and proximal part of the stomach and anastomosis of the antrum to the proximal oesophagus. WANGENSTEEN himself has discontinued the practice as some patients developed anaemia. (Discussion HAWTHORNE 1956).

Following resection of the cardia and lower gullet, either for cancer,

oesophageal varices, benign tumours or strictures, one of two procedures should always be employed. One consists of substituting a physiological cardiac sphincter, for instance by interposition of a segment of jejunum, the other of oesophago-gastrostomy with anastomosis high in the thorax. Bypassing the stomach in ROUX-Y fashion as planned by ALLISON and others, has not proved a satisfactory procedure. Most patients develop anaemia and suffer from malnutrition due to loss of appetite and the inability to eat enough (15, 29, 296).

SUMMARY

Reflux oesophagitis, usually a benign disease, occasionally takes a pernicious course. It then becomes imperative to reverse this course by efficient medical or surgical treatment as it ultimately leads to inanition and death. Although it may occur at any time in life, the onset is usually in the years past fifty. The affection is slightly prevalent in males. The duration of symptoms is usually many years. The disease is commonly associated with oesophageal hiatal hernia of the sliding type. The disease may complicate any laparotomy, but has become notorious in surgery of the cardia of the stomach. An oesophageal stricture due to chronic inflammation usually takes some years to develop. It may progress rapidly, however, as in the case of ascending fibrosis of the oesophagus.

Bleeding is invariably present but severe haemorrhage is unusual. Chronic loss of blood ultimately causes anaemia.

Perforation of an oesophageal ulcer is extremely uncommon and is generally lethal.

In the second part of the treatise, the aetiology and the mechanism of reflux are discussed. The aetiology is reflux of mainly gastric juice and occasionally other digestive juices. Incompetence of the cardia, which allows this reflux, is due to oesophageal contraction. This is due to either vago-vagal reflex or direct stimulation of the muscle coat itself. The function of the cardia is explained by the action of the collar fibres described by HELVETIUS. These fibres form an efficient sphincter which derives its strength from its valve-like structure. Incompetence is due to rotation of this sling of muscle fibres as a result of oesophageal contraction.

In the third part of this treatise the medical and surgical treatment are discussed. Medical treatment, although not always effective in the long run, may be a valuable adjuvant to surgery. Temporary relief may be obtained by a continuous intra-oesophageal drip of milk-soda. Severe haemorrhage can thus be controlled and spasm of the oesophagus relieved. In surgical treatment, restoration of the cardiac mechanism, when possible, is the ultimate goal. When the cardiac sphincter is destroyed, either one of two procedures may be employed. Firstly construction of a substitute cardiac mechanism and secondly performing anastomosis high up in the chest. The logic of this latter

procedure lies in the pressure gradient involved. Hereby the proximal part of the oesophagus is spared reflux of stomach contents if sufficient peripheral drainage of the stomach is provided.
Prevention lies in the preservation of the cardia when possible and restoration of the normal anatomical conditions in the case of oesophageal hiatal hernia.

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