

CARDIOSPASM*

BY

G. H. WOOLER

From the Thoracic Surgical Unit at the General Infirmary, Leeds

HISTORY OF CARDIOSPASM

The first recorded case of cardiospasm is credited to Thomas Willis, who, in his *Pharmaceutica Rationalis*, published in 1672, described a patient with cardiospasm, and also foreshadowed the treatment which is carried out to-day by passing an oesophageal bougie made of whale bone. Einhorn in 1888 published a paper about a case of marked dilatation of the oesophagus. He suggested that it was due to "a lack in the reflex relaxation or opening of the cardia during the act of swallowing." This paper, however, seems to have been overlooked until 1896, when Rolleston presented a similar observation. Hurst, in 1913, propounded the same theory, and on Sir Cooper Perry's suggestion the term achalasia (absence of relaxation) was introduced by him in 1914. Hurst also pointed out that a sphincter muscle differs from other muscle in having active relaxation as a part of its normal activity. At rest its postural tone is sufficient to obliterate the lumen of the passage which it surrounds, and in activity its relaxation opens the passage and removes the obstruction previously present.

In 1922 Mosher concluded that cardiospasm was due to some deformity of the "liver tunnel." He wrote as follows: "In watching a patient with a normal oesophagus swallow, the barium is seen to come to a point momentarily at the upper border of the liver, then, after a delay of a second or two, the liver tunnel opens up and the barium streams into the stomach." In the same year Chevalier Jackson wrote that the diaphragmatic pinchcock and kinking of the oesophagus were the normal mechanisms by which the food in the stomach was prevented from regurgitation. He suggested that it was a failure of the pinchcock to relax that produced the stenosis and the so-called cardiospasm.

In 1925 Walton defined cardiospasm as a "dilatation and hypertrophy of the oesophagus in which at post-mortem examination no obstruction can be found distal to the dilatation." It appeared that the obstruction which was present during life had ceased to function after death. Walton stated that there was a spasm of the part, for which he suggested a congenital origin.

In 1926 and 1927 Rake reported on the microscopical findings of three cases of cardiospasm. Previous observers had been paying particular attention to the degree of muscular hypertrophy of the oesophagus in the vicinity of the cardia, but they had disregarded the condition of the nerve fibres and cells. Rake demonstrated an inflammatory reaction in and around the ganglia and connecting nerves of Auerbach's plexus, and he showed that in later stages this produced fibrosis of the ganglia and complete disintegration of the ganglion cells. He believed that the cause of cardiospasm was a progressive degeneration of Auerbach's plexus. These findings have recently been confirmed by Gallinaro working with Valdoni in Rome; but I believe that the degeneration of Auerbach's plexus is secondary to dilatation of the oesophagus and that it is not the cause of cardiospasm.

THE ANATOMY AND MOVEMENTS OF THE OESOPHAGUS

The oesophagus is divided into two segments. The upper segment comprises the proximal third lying above the aortic arch, and its wall is made of striped muscle supplied by the recurrent laryngeal nerves. The lower segment comprises the distal two-thirds lying below the aorta; its wall is of plain muscle supplied by the vagal plexus. The nerve fibres supplying these two segments of the oesophagus originate from different nuclei in the medulla, and control the movements of the oesophagus which are different in each of the segments.

There are three forms of oesophageal contractions: primary, secondary, and tertiary. The primary wave is initiated by the act of swallowing; it begins in the upper third of the oesophagus and proceeds in an unbroken manner down to the cardia (Plate 1a). As it passes downwards, the oesophagus above the advancing wave remains in a state of contraction. Von Brücke and Satake (1913), studying oesophageal action currents, con-

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cluded that the primary wave was an advancing tetanic contraction.

The secondary wave (Plate Ib) originates in the region of the aortic arch and is confined to the plain muscle segment. It is initiated by the presence of food in this part of oesophagus and is of a similar nature to the primary wave, consisting of a band of contraction passing down to the cardia. McLaren (1943) showed by kymography that these waves were not preceded by waves of relaxation and so were not the same as true peristalsis in other parts of the intestinal canal.

Tertiary waves also arise in the plain muscle segment. They are small worm-like contractions occurring when the oesophagus is distended, lasting only a moment, and, if there are many of them, the oesophagus has a serrated appearance. They are due to rings of contraction in the circular muscle of the oesophagus (Plate IIa).

ONSET OF CARDIOSPASM

The condition affects people of any age. The youngest of the forty-seven patients reviewed in this paper began to vomit when she was nine months old; the oldest was seventy-three when he first attended this clinic, but he had had dysphagia for forty years, during which time he had lived on a careful diet.

The usual age for the disease to manifest itself is during the third decade. In this series, it occurred twice as commonly in women as in men. It affects the highly strung rather nervous individual of the type who might develop thyrotoxicosis. The onset is initiated by psychological trauma. The cardia of such persons appears to be controlled by their emotions and goes into spasm when they are excited.

The following cases illustrate this point:

Two patients developed dysphagia after their respective mothers had died of carcinoma of the oesophagus. A farm labourer started to have dysphagia after he had been knocked down by a horse. Another patient presented herself with cardiospasm soon after her father had died. It was relieved by the Negus dilator and she was well until her mother died, when all her symptoms returned and she required further treatment.

I have mentioned these cases to show that there is nearly always a mental factor present. It seems to me to be of importance because it is so constant. It appears to precipitate cardiospasm, which, once established, becomes an organic disease incurable by psychological treatment.

CARDIOSPASM

Cardiospasm affects the whole of the lower segment of the oesophagus. It runs a continuous,

slow, progressive course which may be divided into two stages. The first is of greatly increased activity of the oesophagus: the second is the gradual cessation of all activity followed by passive dilatation.

First stage.—Patients in the first stage (Plate IIb) are "oesophagus-conscious." They can feel the oesophagus go into spasm when they are excited, and they can feel it relax after taking a hot drink. The first few mouthfuls of food may cause little discomfort, but further swallowing produces the sensation that food is sticking behind the lower third of the sternum. If they continue to eat fast, food regurgitates into the pharynx. These symptoms may fluctuate from day to day, but on the whole they gradually worsen as the disease progresses. Anything other than the normal daily routine may bring on an attack. It was interesting to notice that the patients who had not been relieved completely by treatment felt very much worse when they received my letter asking them to report again to hospital for review.

The barium swallow is of supreme importance in assisting the diagnosis and in understanding the natural course of the disease. Radiographs alone may be misleading, because important factors in the diagnosis are oesophageal movements and the time taken for barium to pass through the cardia, neither of which can be portrayed on a radiograph.

When an early case is screened the first one or two mouthfuls of barium are seen to pass readily into the stomach. The oesophagus, apart from showing increased movement in the lower two-thirds, presents no abnormality and is not dilated. As the patient continues to swallow barium, a constriction about one or two centimetres long appears at the lower end of the oesophagus. This constriction involves the cardia and the cardiac ampulla or that part of the oesophagus lying in and below the diaphragmatic hiatus. If one continues to watch for a few minutes, the spasm at the lower end becomes more noticeable. Barium is retained in the oesophagus, which begins to dilate in order to accommodate it. The retained barium stimulates oesophageal movements. Secondary waves increase in size and rapidity, as the oesophagus dilates, until it looks alive with excitement. It appears to be fighting as hard as it can to push barium through into the stomach. This activity may continue for half an hour or more until the oesophagus is empty. If the patient is given a drink of hot water it often relaxes the cardia and clears the oesophagus in a few minutes (Plate XII).

The stage of activity which I have just described lasts for months. The oesophagus then tires of its increased effort and becomes passively dilated.

This occurs in the second stage when most patients present themselves for treatment.

Second stage.—During the second stage dysphagia and sternal discomfort after swallowing grow in severity. Patients try to combat these symptoms by eating slowly, choosing their diet carefully, and washing solids down with fluids, but they lose weight rapidly because they cannot swallow sufficient food. If the cricopharyngeus is not functioning properly, they get regurgitation of food into the mouth at night. They become thin and anaemic, and may present in a severe degree of cachexia.

Radiography of the chest may show a dilated oesophagus appearing as a smooth convex opacity encroaching on the right lung field from the side of the sternum, and sometimes it projects beyond the right border of the heart. The lateral film frequently shows anterior bowing of the trachea due to the dilated oesophagus pressing it forwards (Plate IIIa). On screening, the oesophagus still looks very active as in the first stage, but the waves of contraction, passing down the plain muscle segment, fade out before they reach the constriction. The part of the oesophagus just proximal to the constriction appears to be immobile and dilated. Cessation of movement followed by dilatation commences here. It gradually spreads up the plain muscle segment until this shows no normal activity, but remains passively dilated. Primary waves may continue in the upper segment, but they do not pass along the plain muscle segment in a normal manner. Here in the lower segment small worm-like contractions are seen which appear to be an effort of the circular muscle fibres alone to convey the food along. Finally after many years these tertiary contractions also disappear. All movements of the oesophagus then cease and it becomes a large patulous, tortuous bag (Plate IIIb).

COMPLICATIONS

Cardiospasm sometimes causes certain complications which may be the presenting symptoms.

In this series of forty-seven patients, three presented with lung complications, and two with rheumatism.

Lung complications

1.—A girl aged 16 gave a two-months history of cough with sputum, dyspnoea, and two haemoptyses. Radiography showed collapse of the right lung, caused by pressure on the right main bronchus (Plate IVa). It was only after very close questioning that a history of dysphagia could be obtained. This had developed at the age of ten, when she was too young to appreciate her condition. Her mother thought that she had just gone off her food. During

the subsequent five years the oesophagus gradually dilated, eventually compressing the right main bronchus. Enlargement of the oesophagus to the left was prevented by the aortic arch and the descending aorta (Plate IVb).

2.—Another girl of 18 presented as a typical case of bronchiectasis. She stated that she had had pneumonia five years previously, and as a result she developed a cough which had persisted. Ever since she had had bronchitis each winter, and one week before her first attendance she had a small haemoptysis. She gave no history of dysphagia; and there was nothing in her symptoms to suggest that she had cardiospasm. Her chest radiographs, however, showed cardiospasm and bilateral bronchiectasis, which was probably due to a chronic inhalation pneumonia, the result of food which had been regurgitated from the oesophagus (Plate Va).

3.—The third lung complication occurred in a woman aged 31, who had been in the habit of taking liquid paraffin after every meal because she believed that it "lubricated her alimentary tract," allowing food to glide into the stomach. The paraffin being of low density floated on the top of the liquid content of her oesophagus. She also found eating such a nervous strain that she had to lie down after every meal. Small amounts of liquid paraffin then regurgitated into her pharynx which she inhaled, causing a paraffin pneumonia (Plate Vb).

Rheumatic complications

1.—A woman, aged 42, stated that her joints had been swollen for two months—her interphalangeal, knee- and elbow-joints being mostly affected. When questioned she gave a fifteen-year history of dysphagia which commenced after her first pregnancy. She had an enormously dilated oesophagus, the contents of which smelt as bad as a pelvic abscess. The lower third of the oesophagus was found to be grossly inflamed and ulcerated.

The arthritic condition was most probably due to toxic absorption from the ulcerated oesophagus, for, after the oesophagus had been washed out and the cardiospasm relieved by dilatation her joint symptoms dramatically cleared up overnight. The relief, however, was only temporary, and when the spasm returned her joints swelled up again. The patient in fact was able to tell the condition of her cardia merely by inspecting her joints.

2.—Another woman, aged 32, presented in a similar way, as a case of rheumatoid arthritis—with swelling of her hands, knees, and elbows. The condition of her joints cleared up just as effectively as in the previous patient after the cardiospasm had been relieved.

OESOPHAGOSCOPY

If the barium swallow shows the oesophagus to be dilated, oesophagoscopy is more safely performed with local anaesthesia. Under general anaesthesia, when the cough reflex is abolished,

food regurgitating into the pharynx may quickly be inhaled. It is absolutely essential to avoid this. If the oesophagus is greatly dilated, it may contain foul-smelling fluid due to bacterial decomposition. The oesophageal mucosa may be inflamed and show gross ulceration, which is in part due to stagnation and decomposition of its contents, and in part due to pressure necrosis caused by the weight of retained food in the lumen. It is the lower horizontal part of the oesophagus running behind the heart to the hiatus which bears the full weight of the contents, and is consequently the first to ulcerate. In this series of forty-seven cases, three showed gross ulceration.

After the oesophagus has been cleansed, the cardia may be difficult to find, for the adult Negus oesophagoscope will not reach to the cardia of a greatly lengthened and kinked oesophagus. It lies around a bend to the left (Plate VI). When, however, the cardia has been found it appears rather like the top of a purse with folds of mucosa radiating from the centre of it. On passing a fine bougie through, it is gripped sufficiently to cause a slight resistance. I have only felt five myself, but in none of these would I describe it as a tight constriction.

After dilatation of the cardia, stomach contents regurgitate back into the oesophagus. Frequently the junction of oesophageal with gastric mucosa may be seen. Allison, using his clip method, applied a Cushing clip to the junction of the two mucosae. It was interesting to discover afterwards, in a few cases where this method had been adopted, that the clip was lying at the proximal end of the oesophageal constriction. This proves that, in some people, the part actually in spasm is lined by gastric mucosa.

DIFFERENTIAL DIAGNOSIS OF CARDIOSPASM

In children there is no other condition which produces radiological appearances similar to those of cardiospasm. For completeness I should like to mention two other causes of dysphagia occurring at an early age.

1.—The so-called "congenital stenosis" is most probably a short oesophagus with part of the stomach lying in the chest, the stenosis being due to peptic ulceration of the oesophagus. It may appear as a sharply defined narrow stricture with smooth margins, or it may be irregular (Plate VIIa).

2.—Simple stricture following the accidental drinking of a corrosive substance occurs in children. The site at which this usually develops is two or three inches above the diaphragm. This is probably due to the fluid

being held up here momentarily by muscular contraction (Plate VIIb).

Late in life carcinoma is the most important lesion to consider in the differential diagnosis, especially when the history is not typical of cardiospasm and the symptoms are of recent onset (Plate VIIIa). It may be impossible to decide radiologically the true nature of the condition—for a high carcinoma of the stomach can spread submucously around the cardia, producing obstruction and the appearance of cardiospasm. Oesophagoscopy reveals no ulceration, for the growth is deep to the mucosa—but on passing a bougie the cardia feels hard and fixed. The constriction is tighter than a cardiospasm.

None of these investigations is necessarily conclusive, and, in such circumstances, the only course is to explore the area of the stenosis.

Simple spasm of the oesophagus does occur, but is extremely rare. Only one case has been recorded in this clinic. This was a man, aged 42, who gave a twelve-years history of attacks of dysphagia coming on when he worked overtime. The attacks lasted two or three days (Plate VIIIb). Oesophagoscopy showed that the whole of the oesophagus below the bifurcation of the trachea was in spasm, the mucous membrane being quite normal. He was relieved of his symptoms by the passing of the oesophagoscope which dilated the lower segment. He has not required further treatment.

TREATMENT OF CARDIOSPASM

The aetiology of cardiospasm still remains unknown, and no satisfactory operation affecting the nervous control of the cardia has been devised to overcome the spasm. The frequent passing of oesophageal bougies or swallowing a mercury tube is distressing to the patient and is unnecessary. Nevertheless these methods have kept many people alive since they were first used in the seventeenth century.

More satisfactory forms of treatment are rupture of the muscle fibres by stretching, surgical division of the muscle in the constricted area, and oesophago-gastrostomy.

In 1904 von Mikulicz described his operation of digital dilatation of the cardia through the stomach. Walton acclaims this method; but I believe that as good results are obtained with the Negus hydrostatic dilator. It causes far less trouble to the patient; it can be performed on out-patients, and can easily be repeated if necessary.

Briefly, the correct method of using this dilator is as follows. The oesophagoscope is passed under local anaesthesia. After cleansing the oesophagus,

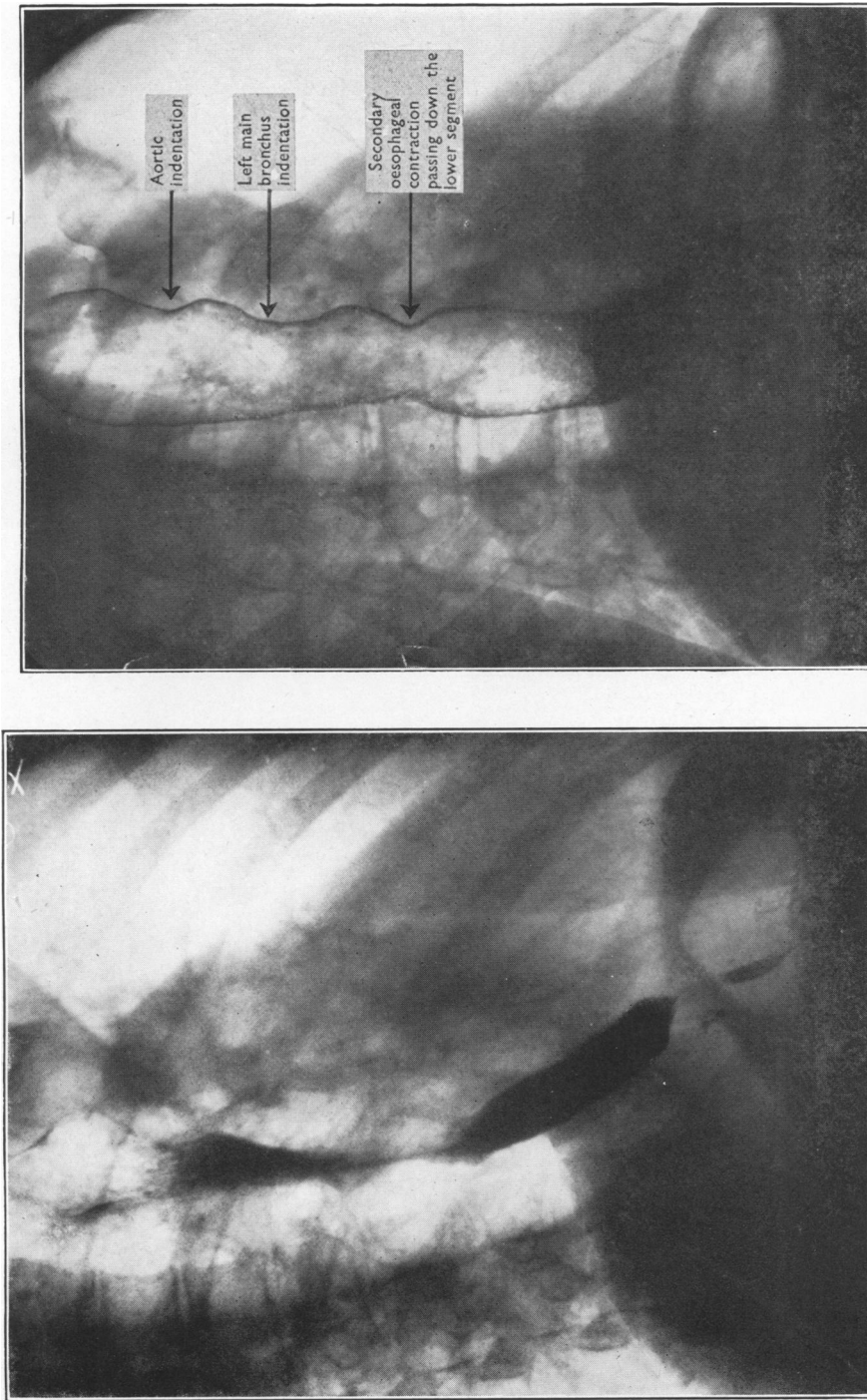


PLATE I.—(a) Primary oesophageal contraction has just left the upper segment and is shown passing down the lower segment. (b) Secondary oesophageal contraction passing down the lower segment.

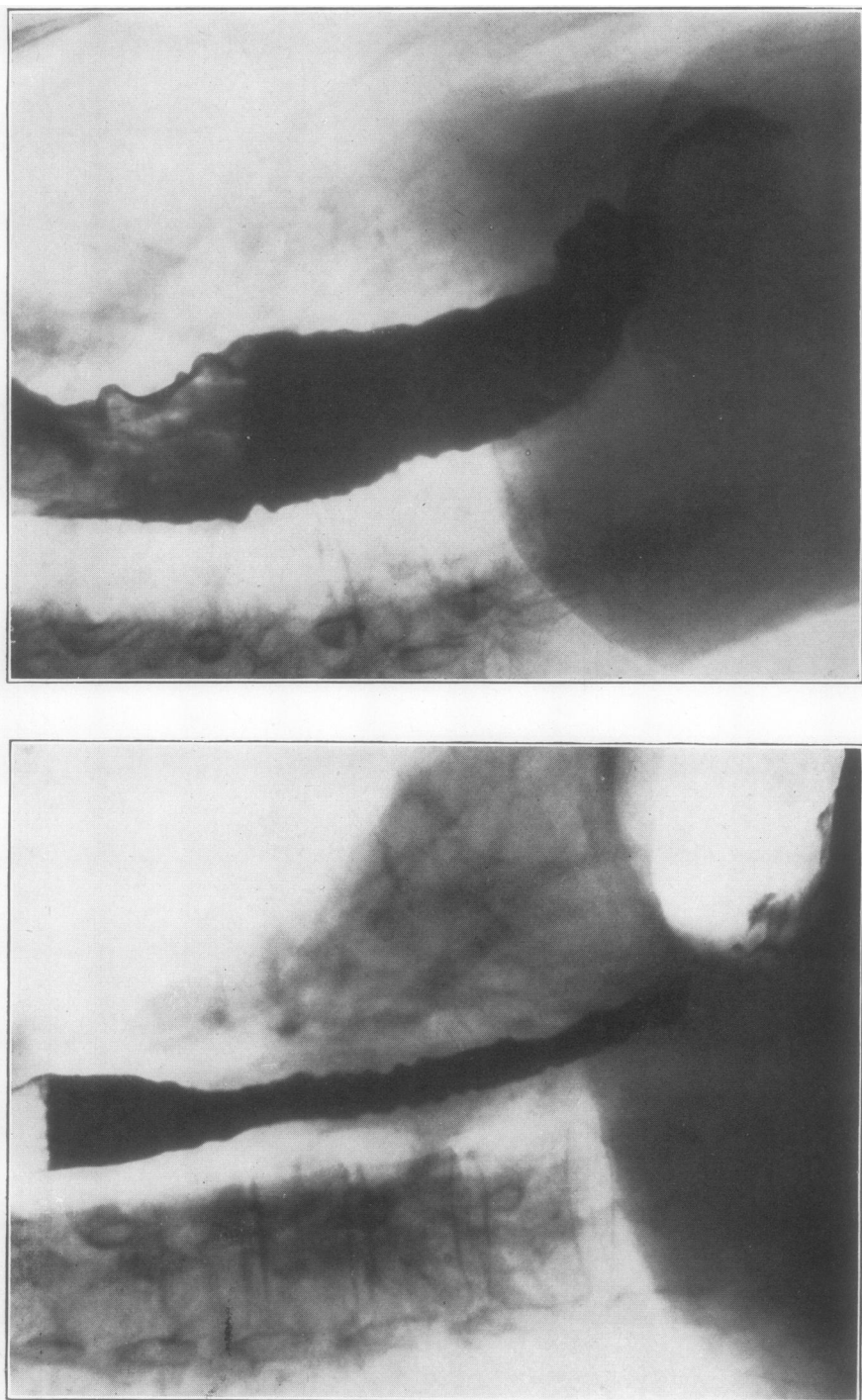
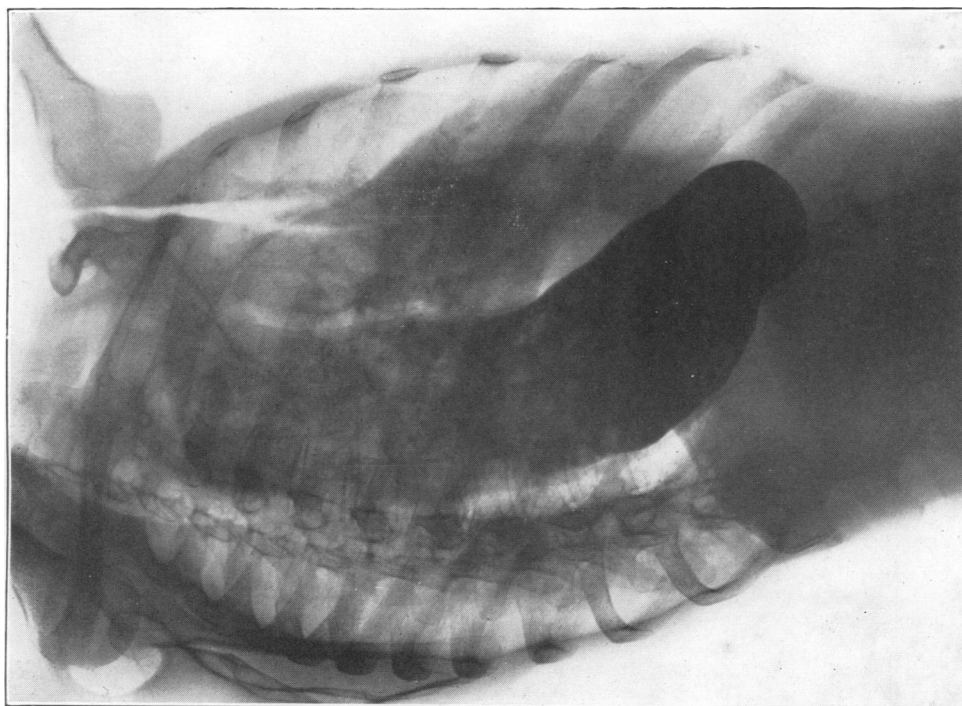
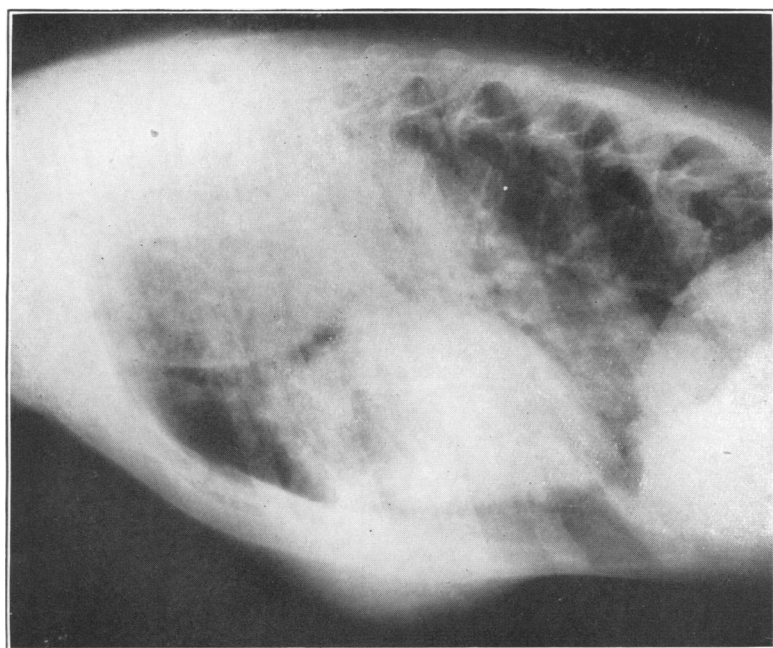


PLATE II.—(a) Tertiary oesophageal contractions. (b) The oesophagus in the first stage of achalasia, showing increased secondary and tertiary waves. When screened it looked alive with excitement.



(b)



(a)

PLATE III.—(a) Anterior bowing of the trachea caused by dilatation of the oesophagus and occurring in a case of achalasia. (b) After all movements have ceased the oesophagus becomes a large patulous bag. A woman aged 56 who had had dysphagia for over twenty years.

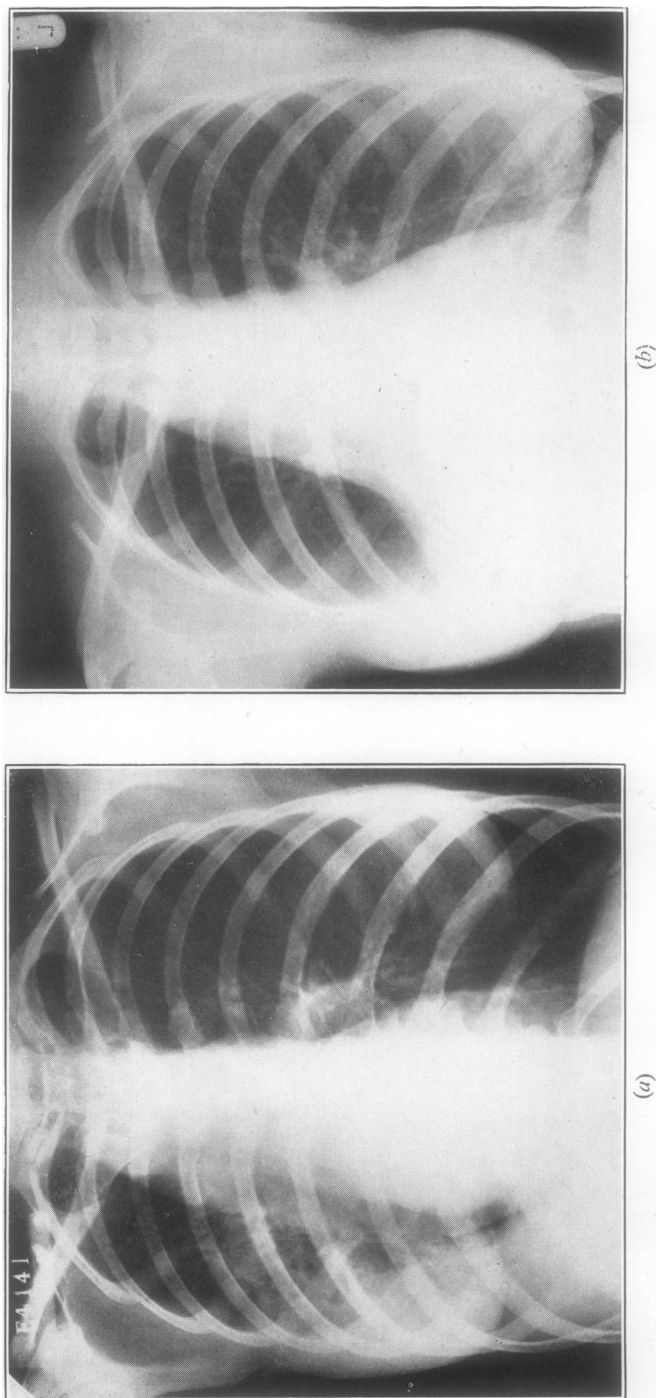


PLATE IV—(a) *Complications* (I). A girl aged 16 years, with a six-year history of dysphagia. The radiograph shows widening of the superior mediastinum due to a dilated oesophagus. The heart and mediastinum are displaced to the right. An inflammatory opacity occupies the lower two-thirds of the right lung. (b) The same patient as in (a) ten months later on admission to this hospital. The radiograph shows collapse of the right lower lobe and widening of the superior mediastinum. The right lower lobe re-expanded within twenty-four hours of the cardia's being dilated with the Negus hydrostatic dilator.

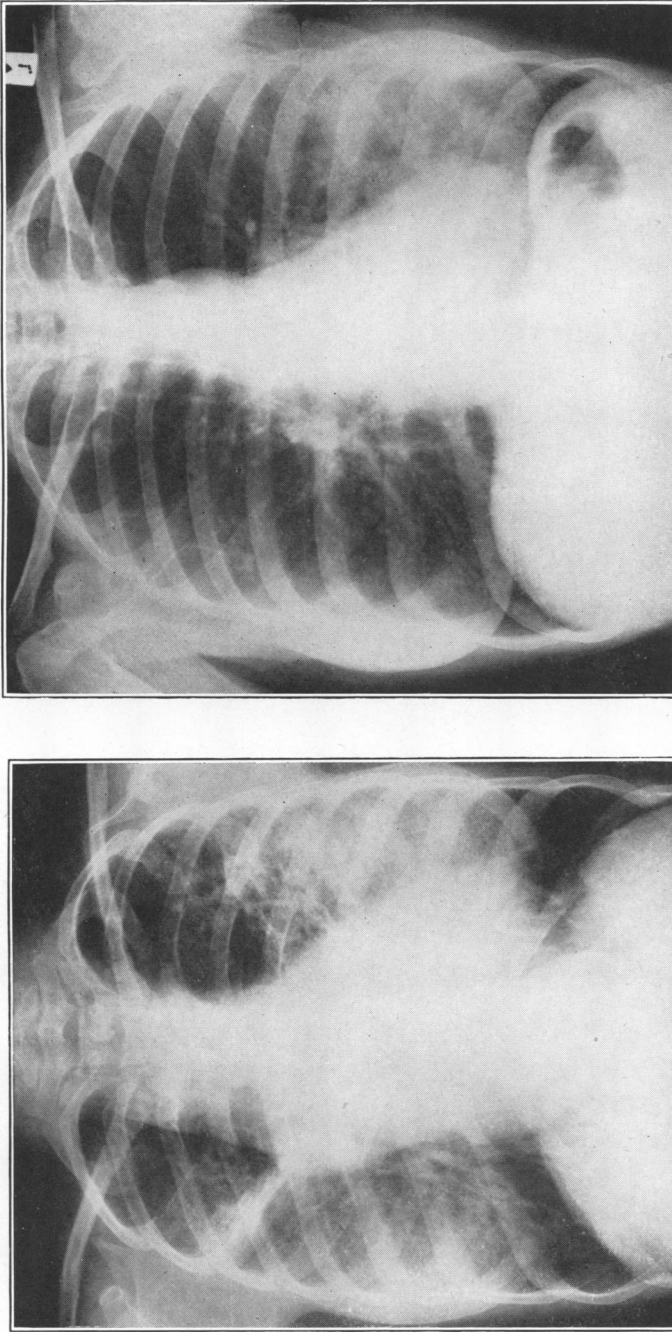


PLATE V—(a) *Complications* (2). Chronic inhalation pneumonia causing bilateral fibrosis and bronchiectasis. Widening of the superior mediastinum due to the dilated oesophagus. (b) *Complications* (3). Inhalation paraffin pneumonia occurring in a patient suffering from achalasia.



PLATE VI.—The oesophagus is elongated and kinked. On oesophagoscopy the cardia may be difficult to find when it lies around a right-angle bend to the left.

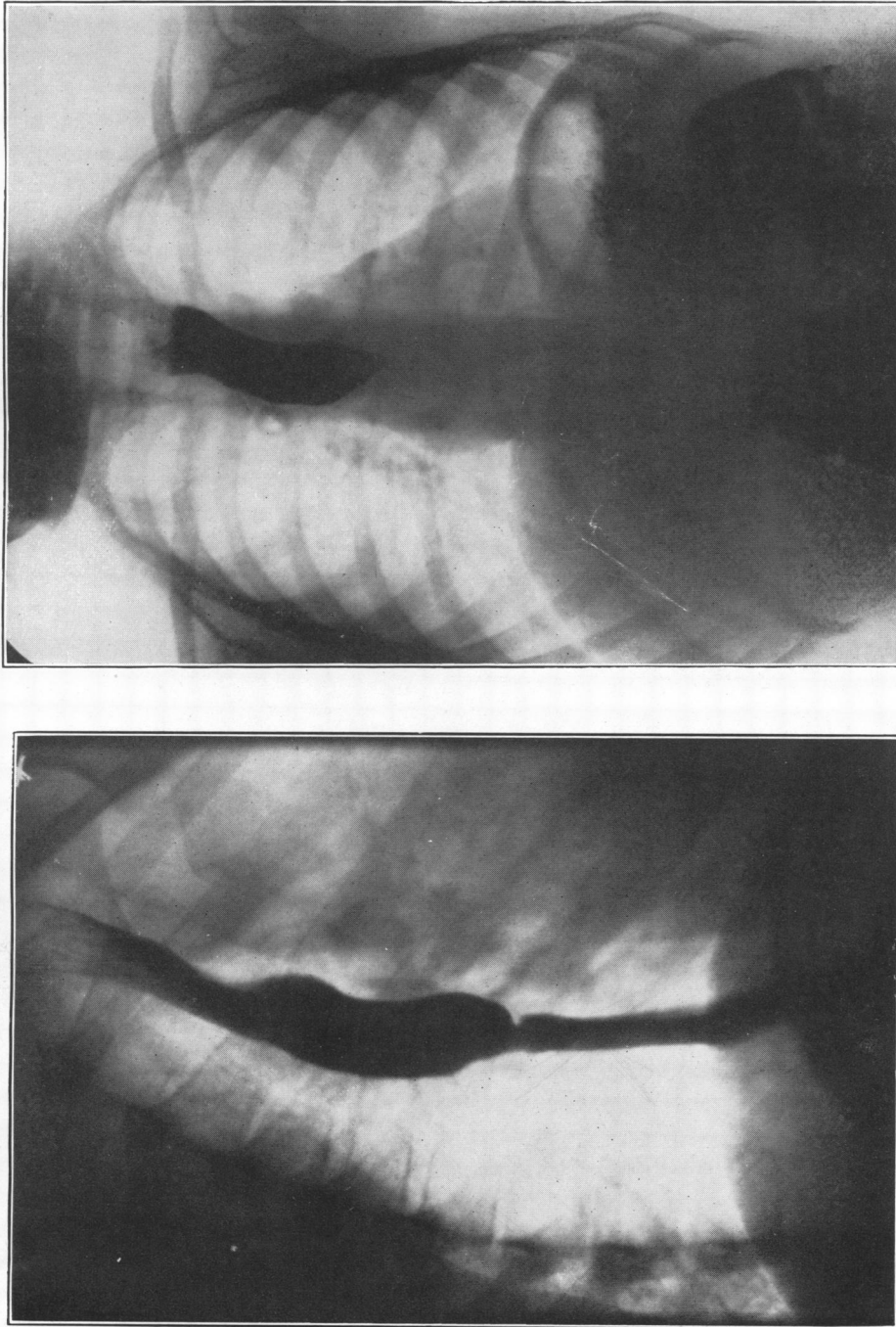
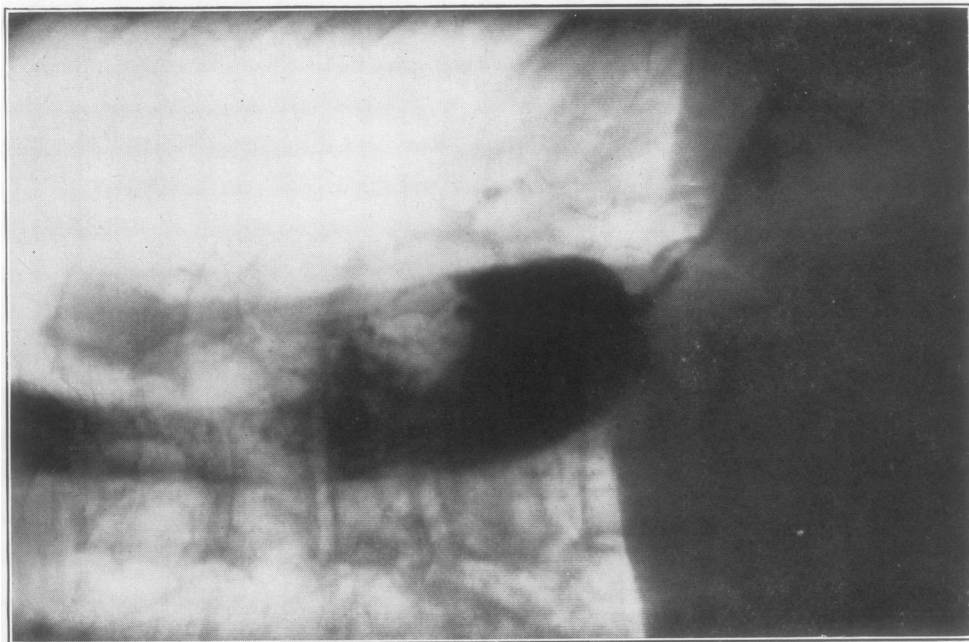
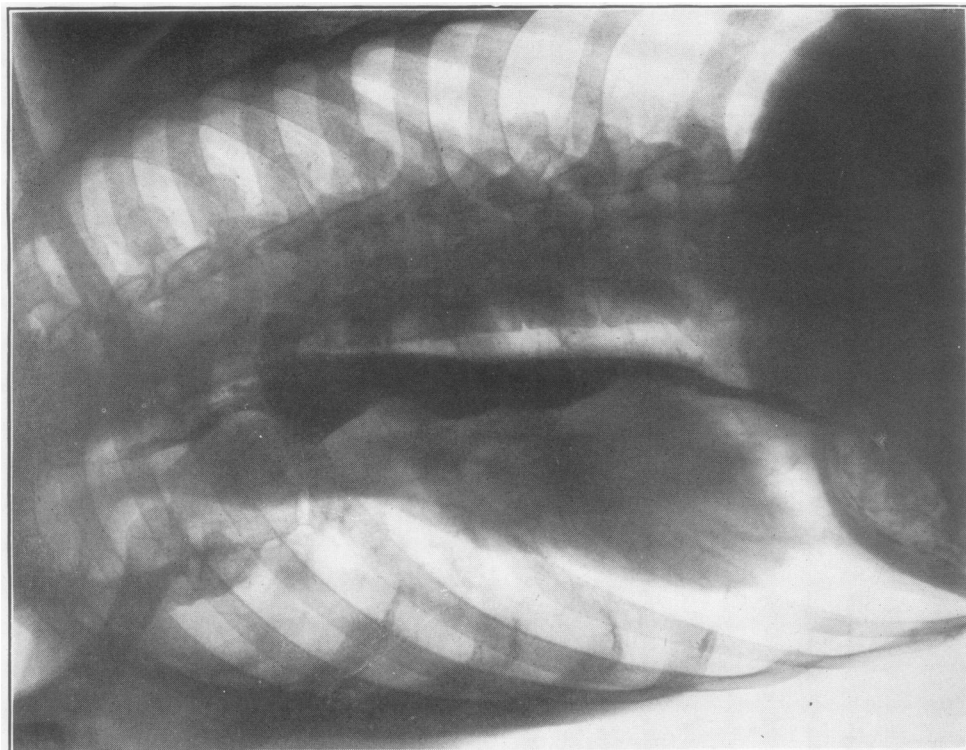


PLATE VII.—(a) *Differential diagnosis* (1). A peptic ulcer of the oesophagus occurring in a patient who has a short oesophagus. A boy aged 13 years who had had dysphagia since birth. (b) *Differential diagnosis* (2). A child aged 1 year 6 months with a corrosive stricture. Radiograph taken three months after swallowing caustic soda.



(a)



(b)

PLATE VIII.—(a) *Differential diagnosis* (3). A man aged 62 years, with one year's history of sternal discomfort after food. Three months before admission he had been oesophagoscoped elsewhere and was thought to have cardiospasm; Hurst's bougies were passed regularly. At operation he was found to have a high carcinoma of the stomach completely surrounding the cardia and producing radiographic appearances which simulate achalasia. (b) *Differential diagnosis* (4). True spasm of the oesophagus.

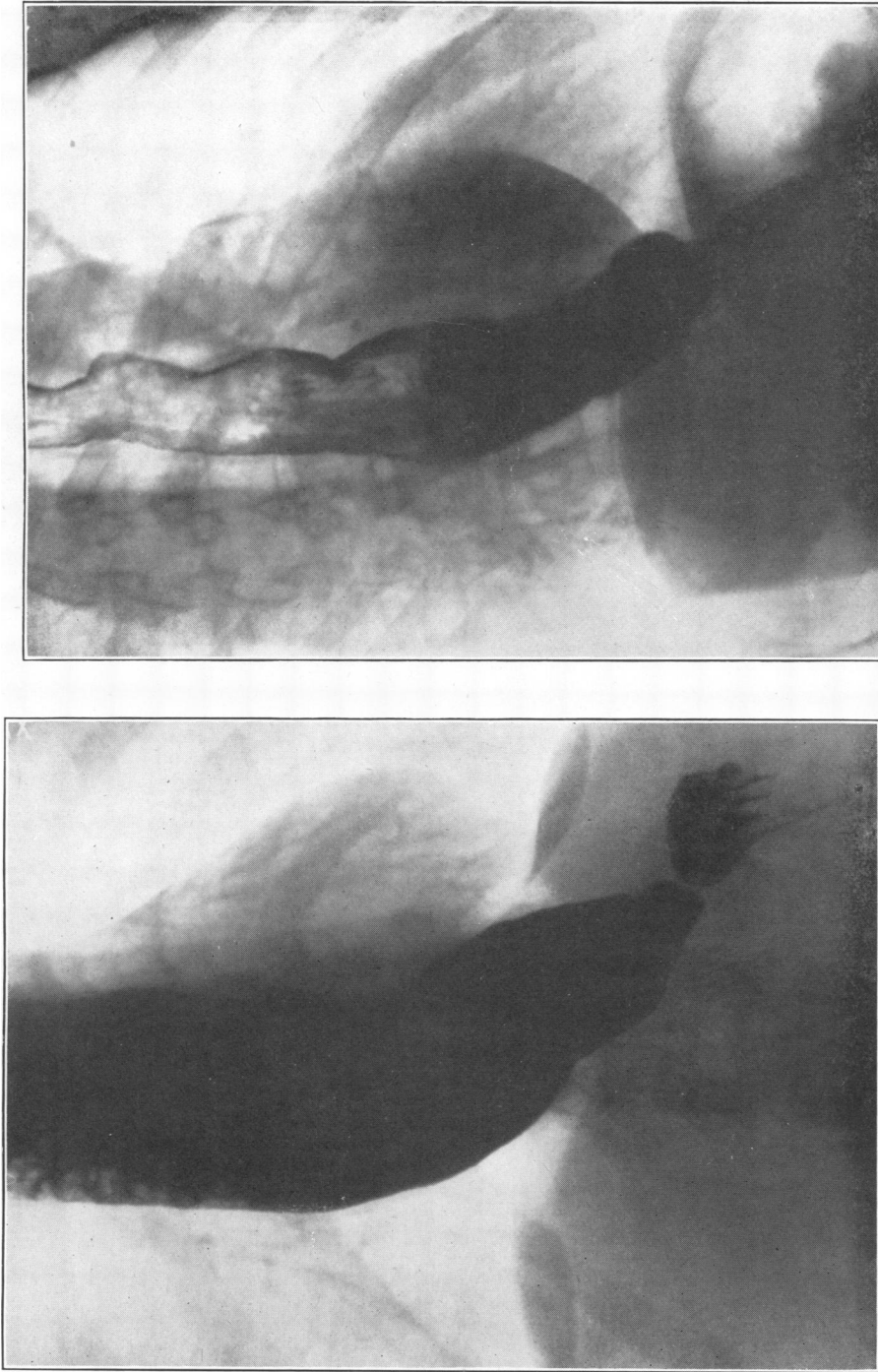


PLATE IX.—*Return of oesophageal movements.* (a) Large dilated oesophagus showing three tertiary contractions only. (b) Eight months later after treatment with Negus hydrostatic dilator. There are good secondary waves passing down the middle third of the oesophagus, but they do not yet reach the lower part, where there are now tertiary contractions.

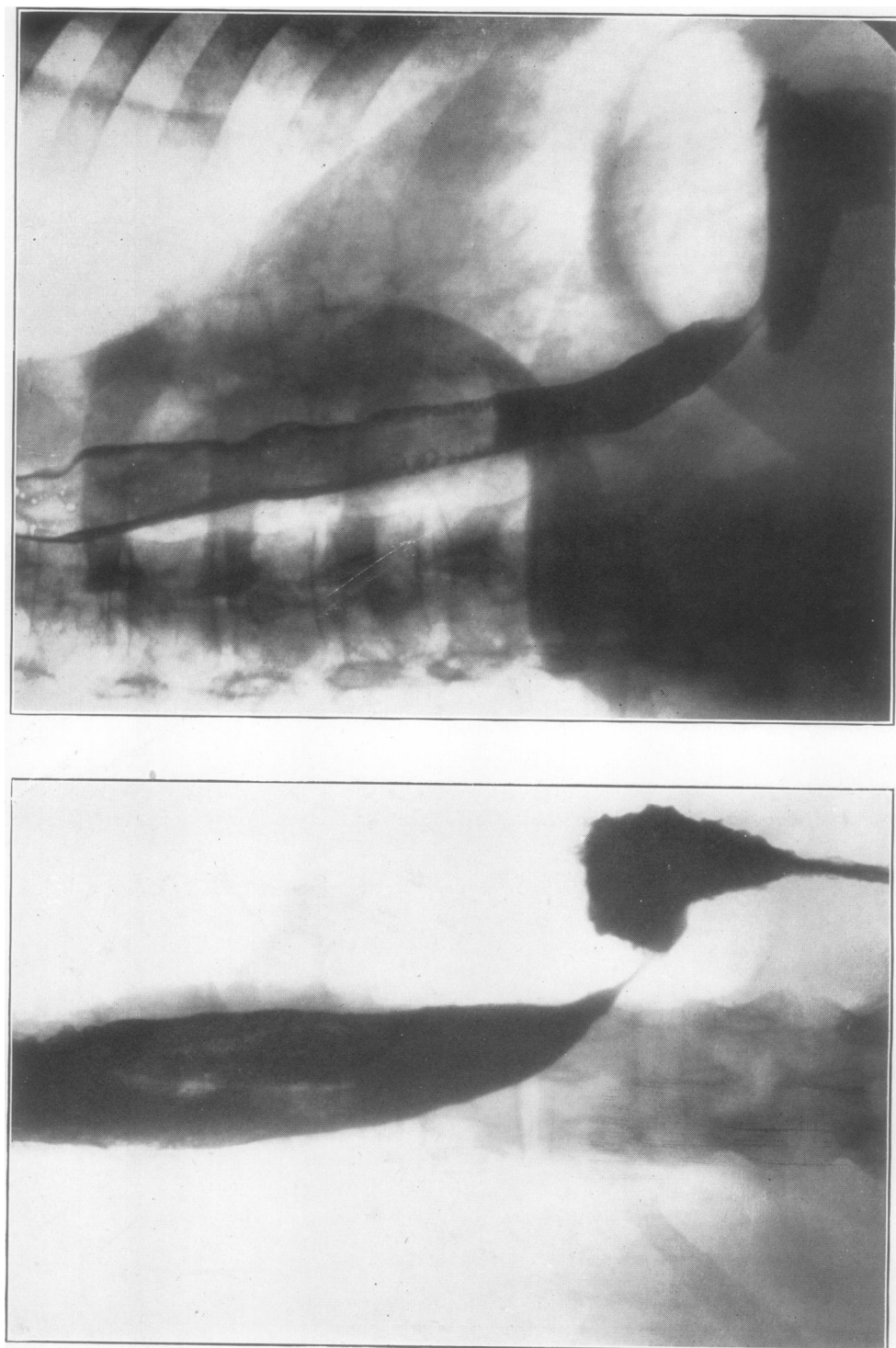


PLATE X.—*Return of oesophageal movements.* (a) Moderately dilated oesophagus showing a few weak tertiary contractions in the middle third. (b) Six months later, after treatment with the Negus dilator. The oesophagus is not dilated, good secondary waves passing down the lower segment almost to the cardia. Just proximal to the cardia weak tertiary contractions can be seen.

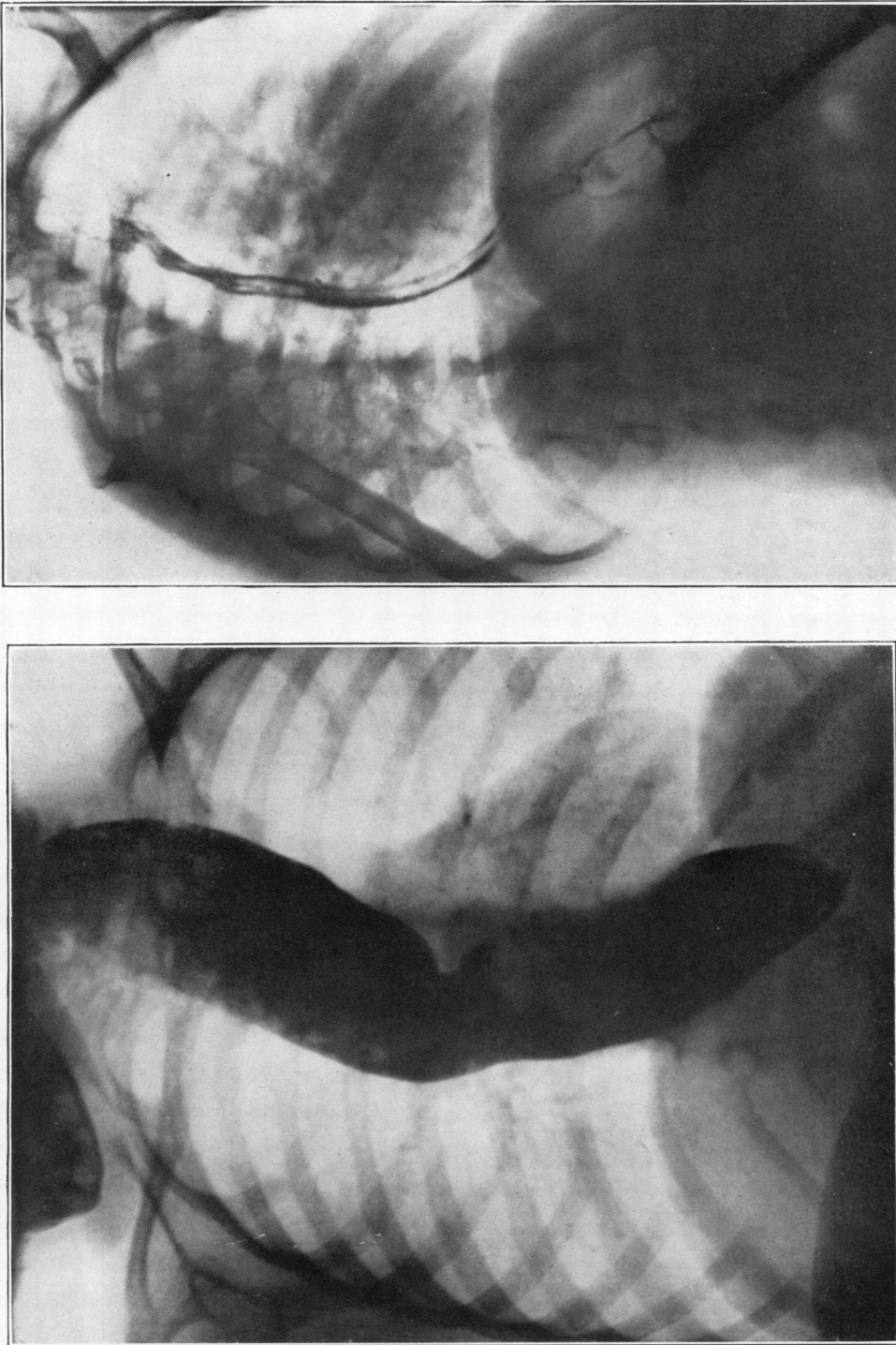
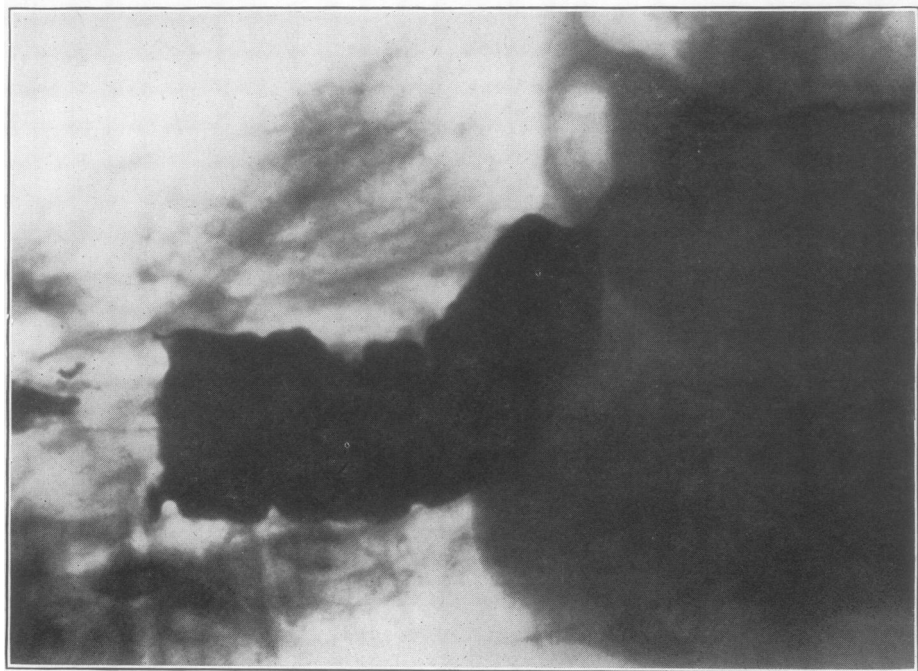
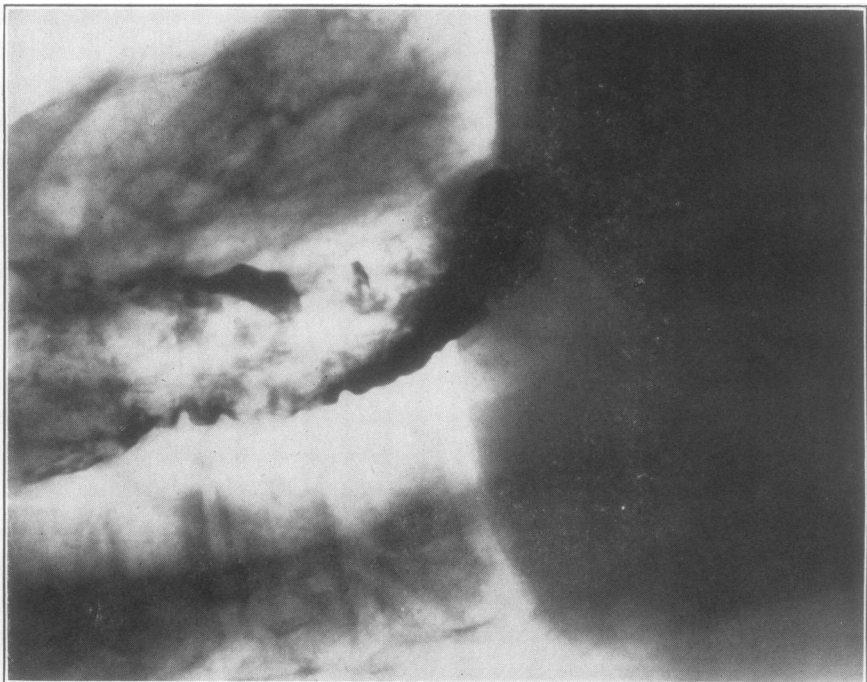


PLATE XI.—*Treatment by modified Heller's operations.* (a) A girl aged 2 years 9 months commenced to vomit when 9 months old. She did not respond to treatment with the Negus dilator. (b) The oesophagus fourteen weeks after extra-mucous cardiomyotomy.



(a)

PLATE XII.—(a) Oesophagus in the active stage, showing secondary and tertiary contractions. (b) A radiograph taken three minutes later shows the oesophagus emptying rapidly after a drink of hot water.



(b)

the cardia is sought. This is dilated with gum elastic bougies up to size 30. The stilette of the dilator is then passed through the cardia, and the Negus hydrostatic dilator is inserted over the stilette. It is important that the constricted part of the oesophagus should be in the centre of the Negus bag when the latter is inflated, otherwise the bag slips either up or down, producing no dilatation of the constriction. To avoid this it is advisable to inflate the bag with 20 to 40 ml. of water at three or more different levels, each time making sure that the bag is gripped by the constriction after inflation. The dilatation is repeated twice at fortnightly intervals.

RESULTS OF TREATMENT WITH THE NEGUS HYDROSTATIC DILATOR

Out of a total of forty-seven cases, thirty-eight received treatment with the Negus hydrostatic dilator. Five of these were not successful and later required Heller's operation. The remaining thirty-three have been seen again up to a period of four years following treatment. Twenty-seven can eat anything and state they have been cured; whilst the remaining six have been relieved to a certain extent. They are under observation because they are still having discomfort after food.

Radiologically the results may not appear quite so dramatic as the relief of symptoms (Plates IX and X). Normal oesophageal activity returns in the reverse order to that in which it was lost in the natural course of the disease, which I have previously described. Treatment in stage one is always successful. Early in the second stage, when activity is ceasing and the oesophagus is dilated, treatment causes reversion to stage one and increased peristaltic activity. After further dilatation the oesophagus generally regains normal movement. Late in the second stage, when the oesophagus is greatly distended and all movements have stopped, it may never regain normal activity after treatment. In such cases delay at the cardia is overcome by dilatation, and the oesophagus may return to a more normal size, but it usually remains inactive, showing no evidence of contraction. Failure of response to the Negus hydrostatic dilator is due to failure to disrupt the muscle fibres.

When this method of dilatation has been given a fair trial and has proved unsuccessful, surgery must be tried. I believe that the best operation is an extra-mucous oesophago-cardiomyotomy, first performed by Heller on April 14, 1913, in Leipzig. On that day he intended doing an oesophago-gastrostomy for a patient with cardiospasm; but found it impossible because the oesophageal wall

was so friable and degenerate and because he was struck by the similarity of the constriction ring to that of hypertrophic pyloric stenosis of infants. Therefore, using an abdominal approach, he incised all the muscle fibres longitudinally down to the mucosa.

In 1942 Barlow recommended Heller's operation through the abdomen, his reason being that, when the oesophagus is lengthened, he can follow von Hacker's method of pulling the redundant part through the hiatus into the abdomen and suturing it there. It is otherwise left to sag in the right cardio-phrenic angle of the chest. I believe that the operation is more easily and safely performed through the chest, because the cardia is more accessible from above. The pathology can be assessed and easily seen, and the operation is less dangerous than when the surgeon is burrowing blindly from below. I divide patients suitable for Heller's operation into three classes: those who have failed to respond to treatment with the Negus hydrostatic dilator; young children frequently do not respond to dilatation alone and often require further operative treatment; and all cases where the diagnosis is in doubt, and exploration is necessary to make sure that cardiospasm alone is present.

RESULTS OF HELLER'S OPERATION

Seven patients have had Heller's operation performed through the chest. All have done well and now have no symptoms except one who at operation was found to have a small hiatal hernia of the stomach as well as cardiospasm. After Heller's operation had been performed the pouch of stomach was returned to the abdomen and the hiatus closed to prevent recurrence of the hernia. Unfortunately it was closed rather tightly so that she is still having dysphagia: but this is improving daily, for the operation was performed only a month ago.

The other six are extremely well and able to eat anything. They have also made a dramatic improvement radiologically, each oesophagus returning to a normal size with no delay at the cardia (Plate XI).

OE SOPHAGO-GASTROSTOMY

Lambert in 1913 was the first to perform an oesophago-gastrostomy for cardiospasm. He did this by pulling the redundant part of the oesophagus through into the abdomen, and, after opening the stomach, he applied a crushing clamp to the fundus and oesophageal wall—treating it in the same way as a colostomy spur: he believed that this was safer than performing a direct anastomosis.

Ochsner and de Bakey (1940) consider oesophagogastrostomy to be the operation of choice for cardiospasm. It relieves the obstruction and has much to commend it, but, as Maingot (1944) points out, many of the patients are left with distressing regurgitation of gastric contents, particularly when they are lying down. Maingot has now abandoned this operation for Heller's, which he considers is safer and worthy of more general adoption. I have no experience of this method and have never seen the operation performed, so it is hardly fair for me to comment. I do, however, believe that any procedure which is going to produce regurgitation of gastric contents into the oesophagus should be abandoned if possible because there is always the risk that at some future date this will cause peptic ulceration of the oesophagus, which is a more distressing condition.

Thus I recommend, first, dilatation with the Negus hydrostatic dilator, and, if this fails, Heller's operation.

TABLE
SUMMARY OF TREATMENT

Total number of cases	47	
1. Negus dilator ..	38	
	27	Good result
	5	Required Heller's operation
2. Heller's operation ..	6	Still under observation
	7	
	6	Good result
	1	Under observation
3. Mikulicz's operation	5	
	3	Good result
	2	Symptoms returned and were relieved with the Negus dilator
4. Refused treatment ..	2	

SUMMARY

The history, pathology, and mechanism of cardiospasm are discussed.

The physical signs and the symptoms of cardiospasm, in its various stages, are described.

The treatment is discussed and the results of treatment are given (see Table).

My thanks are due to Mr. Philip Allison for his great help in preparing the paper and also for his permission to use his notes of the patients. My thanks are also due to Dr. A. S. Johnstone for allowing me to reproduce his radiographs and to read his reports, which are most inspiring. My only contribution has been to review their cases and to collect salient facts which I hope are of interest.

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