RUPTURE OF THE OESOPHAGUS
BY
BETTY VIVIAN SLESSER
From the Thoracic Unit, City General Hospital and Royal Infirmary, Sheffield
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Perforation of the oesophagus still carries a high mortality, despite chemotherapy and antibiotics; and whether the rupture be in the cervical or the thoracic oesophagus, conservative management seems to be unsuccessful in most cases. Conservative measures have included the administration of chemotherapeutic drugs, intravenous fluid, gastrostomy, and in some cases drainage of the pleural cavity and of the cervical and mediastinal tissues.

Barrett (1946) has pointed out that in patients suffering from spontaneous rupture the slit in the wall of the oesophagus may be of a considerable size, whereas the communication into the pleura may be relatively small. In consequence there is inadequate drainage into the pleural cavity and severe mediastinitis results. Fluid in the pleural cavity may be secondary, a consequence of the irritation of the gastric juices, so that simple drainage of the pleura is insufficient to relieve the toxaemia of the mediastinitis. This fact may explain the generally poor results following simple drainage of the pleura. Nevertheless, this method has on occasion been successful, as shown by cases recorded by Churchill (1935), Benson and Penberthy (1938), White (1941), Graham (1944), Adams (1946), and Dorsey (1948). Moore and Murphy (1948) treated their patient at first on conservative lines by thoracotomy and drainage, and repaired the perforation three weeks later. Collis, Humphreys, and Bond (1944) recorded the first case of direct suture of a spontaneous oesophageal rupture. Unfortunately the outcome was not successful.

Barrett (1946) reviewed the literature on spontaneous rupture of the normal oesophagus, and stressed the fact that its possible occurrence must always be kept in mind in the differential diagnosis of any upper abdominal emergency, and that some success might attend prompt thoracotomy and suture when clinical signs supported by radiology had led to early diagnosis. In 1947 he was able to record the first successful suture of a spontaneous rupture of the oesophagus. This was followed by another success reported by Olsen and Clagett (1947), their repair being carried out only three hours after the onset of symptoms. Clifton (1949) has reported a successful closure, and in Scholefield's (1949) case successful suture and recovery ensued despite perforation into both pleural cavities. Lynch (1949) has three successful cases to his credit, two treated by suture and one by drainage of the pleura, the patient recovering after a stormy convalescence. The most recently recorded success is that of Dunavant and Skinner (1951).

Nevertheless, perforation due to instrumentation or by impaction of foreign bodies is commoner than spontaneous rupture, and there is a reluctance to report cases of this type. Mosher (1935) reported 19 perforations in a series of 938 oesophagectomies. Goligher (1948) recorded two cases of rupture in the post-cricoid region after the passage of a gastroscope, and urged that prompt surgical
repair should be performed as soon as the accident is recognized. He states that if this is not done cases will be lost through infection tracking down into the mediastinum. Temple (1949) sutured multiple perforations in the oesophagus caused by a swallowed razor blade. In this case diagnosis and repair were prompt. He suggested that there might be some place for expectant treatment in rupture of the cervical part of the oesophagus, especially if surgical emphysema, which he considered denoted a complete rupture, was not present; he quoted such a case. Wells, Hughes, Edwards, and Marcus (1949) have reported a successful suture of the lower end of the oesophagus, which had been ruptured by a mercurial bougie. Avery Jones, Doll, Fletcher, and Rodgers (1951) in a series of 49,000 gastroscopies found 54 oesophageal perforations, most of them in the upper part in elderly women, and in cases where a Herman Taylor instrument had been used. They concluded that "mild" cases might be treated conservatively, but that lower oesophageal ruptures and cases with a gross leak should be sutured as soon as possible. They do not agree with Goligher that it is necessary to employ routine radiographic examination of the vertebral column to exclude arthritic changes before endoscopy.

The aetiology of rupture of the apparently normal oesophagus is not clear. Mechanically the lower part is said to be the weakest, and under experimental conditions it will burst under pressures of 5–11 lb. of water (Zenker and von Ziemsens, 1878, quoted by Gott, 1933). Weiss and Mallory (1932) demonstrated fissures round the cardiac end of the gullet in cases of persistent vomiting, and Vinson and Johnson (1928) described strictures in the lower oesophagus in hyperemesis gravidarum, presumably due to fibrosis in fissured areas. Spontaneous rupture may well occur in such a fibrosed and weakened area, though many reported cases have shown no change in the muscular coats. Congenital weakness is not a likely explanation. Menne and Moore (1921) found only three cases in childhood, when the combination of vomiting and congenital defect could be expected to produce their worst effect. It is recognized that perforation of an oesophageal peptic ulcer can occur, and peptic digestion of a traumatic fissure may possibly account for some cases. Two cases of oesophageal perforation following burns are on record (Rankin, 1945; Robson, 1943). In Robson's case Curling's ulcers were present in the stomach and duodenum. Cases associated with neurogenic upset have been described (Craig and Lipscomb, 1936; Masten and Bunts, 1934). Most of the spontaneous cases have occurred in males, possibly associated with alcoholism.

In rupture caused by instrumentation, the pathological lesion calling for investigation is obviously a factor of considerable importance in deciding the nature and site of the perforation, e.g., dilatation of a stricture, taking specimens for biopsy. It is sometimes possible to see during the manoeuvre that perforation has occurred. More often, however, the signs appear some hours or days later, and the perforation may then be due to an ischaemic area slowly undergoing necrosis. Ruptures have been found in the post-cricoid region remote from the lesion under review. Goligher (1948) stated that it was unlikely that spasm of the cricopharyngeus muscle was a factor, because in so many cases there was no difficulty in instrumentation. He drew attention to the fact that these upper oesophageal perforations occur on the posterior wall where the mucosa is supported only by circular fibres, the longitudinal muscle coat being defective in this area. Furthermore, in the extended position of the head and neck during endoscopy, the pharyngo-oesophageal wall is compressed backwards
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against the bony resistance of the cervical vertebrae. This compression may well be excessive if, due to spondylitis, the vertebral bodies are irregular. He therefore recommends routine radiographic examination of the cervical vertebrae to determine whether such osteophytic outgrowths are present.

CASE HISTORIES

Case No. 1.—M. H., a woman aged 73, was admitted to the Royal Infirmary, Sheffield, on May 5, 1950, complaining of severe dysphagia for all forms of food, liquid or solid. For the previous four months there had been pain over the lower ribs on the left side, and in the epigastrium immediately after meals, together with increasing anorexia and dysphagia. Loss of weight was considerable, but she had never actually vomited. She had a feeling that food never seemed to enter the stomach properly.

On admission she was noted to be a small, rather wasted woman, but physical examination was otherwise negative, and there were no abnormal neurological findings.

A barium swallow and barium meal showed no abnormality in the oesophagus, stomach, or duodenum.

She was kept under observation for 12 days, during which time it was noted that she did not appear to have any difficulty in taking semi-solid food, nor did she vomit. She still complained of food sticking behind the xiphisternum.

On May 18 endoscopy was performed under intratracheal anaesthesia. No difficulty was experienced in passing the oesophagoscope, but on approaching the lower end of the oesophagus a longitudinal tear appeared on the left antero-lateral wall through which the diaphragm and lung could be seen. It was felt that the instrument had been passed down to but not through the tear, and that it had not directly produced the trauma. The patient was immediately turned on her right side and a left thoracotomy was performed. A longitudinal tear was found in the pleura covering the oesophagus about one inch above the diaphragm, and, on incising the pleura further, a similar though longer tear was found in the left antero-lateral wall of the oesophagus. The edges were clean-cut, and there was no sign of malignancy or of any pre-existing inflammatory lesion. The tear was sutured with two layers of interrupted silk sutures, and the pleural cavity drained for 48 hours.

Recovery was uninterrupted. The patient went on to solid diet on the tenth day. There was no further dysphagia, and she was discharged from hospital on June 3, 15 days after operation. There was at no time any suggestion that the case might have been one of cardiospasm.

The patient remained well until September, when the dysphagia recurred, and she could only swallow liquids. She was readmitted on September 13, and the following day endoscopy was again performed under general anaesthesia. In view of her history this was undertaken after some hesitation and with considerable anxiety. It was noted that the lower end of the oesophagus appeared to be narrowed by a spasm. The oesophagoscope was not advanced further, but bougies were passed quite easily up to size 16, and there was no hint of any damage to the oesophagus. Eight hours after endoscopy she complained of severe abdominal pain, and the physical signs resembled those of a perforated gastric ulcer.

In view of the previous history, rupture of the oesophagus was considered to be the most likely diagnosis. Radiological examination showed the presence of air below but not above the diaphragm. Under general anaesthesia the abdomen was opened by a left paramedian incision. Air escaped on incising the peritoneum, but there was no leakage of gastric contents into the peritoneal cavity. On examining the stomach and lower oesophagus, a longitudinal tear was found in the left lateral wall of the abdominal
oesophagus. The edges were neither ragged nor bruised, and were sutured in two layers with fine interrupted silk sutures, with an omental graft. The abdomen was closed without drainage. The post-operative course was uneventful, and up to the present time she has not had any more dysphagia.

In January, 1951, she was admitted for the removal of a small scirrhous carcinoma of the breast, and it is of interest to note that both her mother and sister died from this disease.

The pathological cause of this patient's dysphagia has never been determined. Her initial symptoms suggested a peptic ulcer of the oesophagus, but at operation no such condition was present. The cause of the rupture on both occasions was instrumentation. On the first occasion dilatation of the proximal oesophagus may have been sufficient to initiate the rupture. On the second occasion the passage of a bougie caused an incomplete tear of the mucosa, which subsequently became complete, possibly as the result of muscular contraction.

This case illustrates the fairly good prognosis to be expected where prompt suture of a perforation of an apparently normal oesophagus is performed, since no great technical difficulty was encountered, and healing was rapid. Where stenosis distal to the rupture exists, the prognosis is probably different.

Case No. 2.—G. C., a man aged 18, was admitted in March, 1950, to the City General Hospital, Sheffield, complaining of a rabbit bone sticking in the throat in the region of the hyoid bone. There was marked spasm of the paravertebral muscles and the left sternomastoid with tenderness on pressure, but no surgical emphysema.

A laryngoscope was passed under general anaesthesia, and a piece of rabbit bone about 1 cm. long was seen projecting from the left lateral wall of the hypopharynx. It was removed by forceps and measured 2 cm. Some 1.5 cm. of bone had therefore perforated the wall of the pharynx. A tear in the mucosa could be seen but was not sutured.

The patient was put on a fluid diet and large doses of penicillin. The pain and muscular spasm passed off in two days, and he made an uninterrupted recovery.

This case suggests that there is some place for conservative treatment in perforation of the upper oesophagus or hypopharynx, where signs of leakage and contamination are absent.

Case No. 3.—F. L., a woman aged 73, was admitted to the City General Hospital, Sheffield, on August 3 complaining of vague dysphagia and a sensation of food sticking at the root of the neck over a period of three years.

A barium swallow and barium meal gave normal results, as did blood counts. Laryngoscopic appearances were normal.

On August 10 oesophagoscopy was performed under general anaesthesia. No difficulty was found in passing the instrument down to the cardia, but at this point there was pronounced spasm, and the instrument would not go any further. Bougies also failed to pass. No sign of growth or ulceration was visible. The investigation took some 30 minutes.

Two days later she complained of considerable dysphagia, with pain and tenderness in the neck, most marked on the right side, and the temperature rose to 102° F. The larynx was reddened and oedematous, but there was no surgical emphysema. By August 14, the signs and symptoms were even more marked, and dysphagia was extreme. There was still no emphysema, and radiographs showed no air leak into the pleural cavity. Gastrostomy was performed, and she was put on intravenous salines and large doses of penicillin.
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On August 18 drainage of a retro-pharyngeal abscess was carried out by an incision along the posterior border of the right sterno-mastoid muscle. Much foul-smelling pus was evacuated, but thereafter, although there was relief from the dysphagia, the patient's general condition deteriorated and she died on August 23.

Necropsy showed a large mediastinal abscess caused by pus tracking down from the retro-pharyngeal space. No obvious tear was seen in the pharynx.

This case raises some interesting points, which must remain speculative, since no other pathology was found at necropsy. Although no obvious mechanical difficulties were encountered until the cardia was reached, hyperextension of the head for endoscopy maintained for 30 minutes may have made it possible for pressure necrosis to occur in the hypopharyngeal region.

In any case simple drainage was the only possible procedure, as there was no tear which could have been sutured. However, due consideration must be given to the extent of the surgical drainage required in these cases, as it is obvious that drainage of the cervical tissues was too late or inadequate, and that by the time it was carried out it should have been supplemented by mediastinotomy.

Case No. 4.—A man aged 50 was admitted to the Horton Hospital, Epsom, as an acute abdominal emergency. He had had a large midday meal and three and a half hours later had vomited copiously and complained of severe upper abdominal pain. It was not certain whether the vomiting or the pain occurred first. He only vomited once, and the vomitus contained streaks of blood and had a sour taste. He had a previous history of heartburn, but this had never been sufficiently severe to warrant investigation in hospital. A diagnosis of either acute pancreatitis or perforated peptic ulcer was made, but signs and symptoms were not felt to be quite typical of either condition.

Radiographs of the chest showed a small effusion at the left base.

He was seen by Mr. Barrett (personal communication, 1951) six and a half hours after the onset of symptoms, and a diagnosis of spontaneous rupture of the oesophagus was made. Two and a half hours later a left thoracotomy was carried out. The typical appearances were seen in the pleural cavity, with gross oedema of the mediastinal pleura. Behind the pulmonary ligament a tear was found in the mediastinal pleura, communicating with a large collection of blood-stained oedematous material in the mediastinum. A hole was found in the lower oesophagus, and this was sutured with two simple through-and-through sutures of thread. The tear in the mediastinal pleura was left open and the pleural cavity was drained with an under-water seal.

Convalescence was stormy. The patient developed a total empyema and a small oesophageal leak into the pleural cavity. Eventually, with continued drainage, the lung expanded to close the empyema cavity, and the leak closed. He has had no further trouble. Subsequent oesophagoscopy showed a partial stricture at the approximate site of the perforation. There was no evidence of "reflux oesophagitis."

It cannot be certain whether this was a true spontaneous rupture of a previously normal oesophagus, or whether it was a perforation of a peptic ulcer of the oesophagus.

Case No. 5.—W. K., a man aged 50, had had mild dyspepsia from time to time, but was otherwise apparently healthy. On December 2, 1950, after some heavy manual work in cold weather he had had a cup of tea containing a small quantity of whisky, rested afterwards, and had a light meal. Later in the evening he complained of a feeling of abdominal distension, which was relieved by brandy, and he went to bed about 10.30 p.m. At 3 a.m. he awoke with a feeling of great nausea and almost immediately afterwards he vomited profusely, and the vomitus contained streaks of blood. The act of
vomiting was associated with violent pain in the left chest, and he felt "as though he was going to die." At 3.30 a.m. he was seen by a consultant physician and was found to be lying on his back afraid to move and extremely apprehensive. He was complaining of severe pain in the upper abdomen and the left side of the chest, and felt that "if he could bring up wind it would ease him," but he could not do so. The abdomen was distended and rigid. The left side of the chest was hyper-resonant, with diminished breath sounds. The heart sounds had a peculiar quality, similar to the "bruit d'airain." Surgical emphysema was present, confined to a small area above each clavicle. A diagnosis of spontaneous rupture of the oesophagus was made, and he was transferred to a general hospital. By the time he was admitted, surgical emphysema had reached the eyebrows. Radiographs of the chest confirmed the presence of hydro-pneumothorax.

Six hours after the onset of symptoms, a left thoracotomy was carried out. About one pint of stomach contents was removed from the pleural cavity. A tear was found in the oesophagus, just above the diaphragm, running vertically upwards for one inch. This was sutured with interrupted silk sutures and the chest was closed with intercostal drainage.

Four days later, fluid, suggestive of milk, began to drain from the tube, and within the next day or two it became obvious that there was an oesophageal leak. The patient was transferred to the Thoracic Surgical Unit at the Sheffield Royal Infirmary on December 13.

His general condition was good, and it was decided to reopen the chest and suture the fistula. The chest was opened through the original incision. The lung was found to be fully expanded and adherent over a wide area. The mediastinal pleura was gangrenous and sloughing. The tear in the oesophagus had completely reopened. The edges were freshened and re-sutured, dead and foreign material was removed, and the chest closed without drainage. For four days he did well, but then fluid began to drain through the chest wound, and indigo-carmine, given by mouth, appeared in the fluid. A rib-resection and drainage was done, followed by a jejunostomy, but he died on December 26, 12 days after the second operation.

At post-mortem examination, there was no sign of peptic ulceration of the oesophagus or of new growth. There was a little scarring over the duodenal cap, but no ulcer crater and no stenosis. The jejunostomy had leaked and there was peritonitis.

Case No. 6.—D. H., a woman aged 21, was admitted to the Thoracic Unit at the City General Hospital, Sheffield, with unilateral right lower lobe bronchiectasis, associated with rather severe recurrent haemoptysis.

At operation the lower lobe was extremely adherent, and the fissure ill-defined. The lobe was removed by the two-tourniquet method, and the chest closed with intercostal drainage. The remaining lobes expanded well post-operatively, and she appeared to be making good progress. Six days later a little fluid appeared in the right pleural cavity. This was aspirated and thought to be an ordinary effusion. Ten days later the patient's condition was deteriorating, and she felt that fluid taken by the mouth was trickling into the chest. Methylene blue was given by mouth, and appeared in the pleural fluid. Bronchoscopy showed a large fistula in the stump of the lower lobe bronchus, with food debris lying in it. A rib-resection and a gastrostomy were carried out, but the gastrostomy feeds had a tendency to leak back into the pleural cavity. A jejunostomy was then carried out, and gastrostomy feeds were discontinued. Thereafter she started to improve. Her morale was excellent and she co-operated in every way. The lung gradually expanded leaving only a small empyema cavity, but the bronchial and oesophageal fistulae persisted. On January 6, 1941, the right chest was reopened. The lung was found to be densely adherent to the diaphragm and pericardium. The oesophageal fistula
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was found in the right lateral wall lying just behind the stump of the bronchus. All signs of acute inflammation had subsided, and the edges of the fistula were freshened and closed without difficulty. The stump of the right lower lobe bronchus had completely re-expanded, and there was an associated collapse of the right middle lobe. This lobe was removed, and the bronchus closed with interrupted nylon sutures; the chest was closed with drainage. At no time post-operatively was there any suggestion of an oesophageal leak. The empyema cavity closed slowly over a period of months. The jejunostomy tube was removed on the eighteenth post-operative day.

The patient is now married, and has no symptoms referable to the chest. On further questioning she admits that ever since she can remember she has had occasional attacks of choking and coughing associated with the taking of fluids by mouth, which suggests that the oesophageal fistula was not entirely traumatic as was thought at first, but that there might have been a congenital oesophageal bronchial communication present, and that the bronchiectasis was secondary to this.

DISCUSSION

It has been suggested that, following prompt suture of a tear due to instrumentation of the apparently normal oesophagus, healing by primary intention is to be expected. With delay of a few hours, however, the tissues become oedematous, friable, and begin to slough. This is especially the case when the tear of the oesophagus is associated with regurgitation of gastric contents, which especially occurs in a spontaneous rupture of the oesophagus. Despite prompt diagnosis some delay in suture of the oesophagus is inevitable. By the time the necessary medical advice has been obtained, the patient transferred to hospital, and shock and toxaemia treated, several hours have passed. Healing by primary intention is now less certain. There arises the problem of the management of the oesophageal fistula. It seems that some cases will close spontaneously, where good drainage of the pleural cavity is maintained, as shown in Case 4. Presumably a large fistula in which all the stitches have cut out will not do so, and it then becomes a matter of discussion as to whether or not one ought to make the attempt to do a secondary suture. Patients are in a poor condition to withstand a second major operation, and by the time the leak is diagnosed the tissues, not having recovered from the initial onslaught of infection, are in no state to hold sutures. In Case 5 the attempt to resuture the fistula was probably made too early. Free and continued drainage might have been better and, had the patient survived, secondary suture carried out at a later date, when the acute inflammation had subsided. That this does occur, and re-suture is then relatively easy, is illustrated by Case 6. Provided that the lung expands and walls off the infection from the general pleural cavity, and that there is free drainage from the mediastinum, patients should be able to survive the acute stage of the inflammation, as long as arrangements are made to feed them other than by the oral route. Intravenous fluids do not keep the patient in the necessary "balance" for more than three or four days, despite the infusion of amino-acid preparations, proteins, and vitamins. In cases where the leak is in the lower oesophagus, gastrostomy feeds are apt to leak back through the fistula, perpetuating the infection and starving the patient. Feeding by jejunostomy is probably the best method, although in patients with malnutrition the tissues do not heal well, and there is a risk of leak and of peritonitis. Probably a Roux type of jejunostomy is the safest if it is to be maintained for some weeks.

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BETTY VIVIAN SLESSER

SUMMARY

The literature on perforation of the oesophagus has been briefly reviewed. This includes cases of spontaneous rupture and those due to foreign bodies and to instrumentation.

Five cases of rupture of the oesophagus and one of rupture of the hypopharynx have been recorded.

In all cases of perforation of the thoracic oesophagus, it would appear that the best prognosis is offered by early suture of the actual tear.

In cases of perforation of the cervical oesophagus and hypopharynx it would appear that there is some place for conservative treatment. If there is no sustained response to this, operation should be undertaken. In some cases no actual tear will be seen, and drainage alone is possible. In such cases the danger of mediastinal abscess must be kept in mind, and the extent of surgical drainage must be planned accordingly.

The management of cases of oesophageal fistula is discussed and illustrated by three cases.

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Betty Vivian Slessor

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