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Oesophageal trauma: incidence, diagnosis, and management

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Triggiani, E. and Belsey, R. (1977). *Thorax*, **32**, 241–249. **Oesophageal trauma: incidence, diagnosis, and management.** The clinical manifestations, diagnosis, and surgical treatment of 110 cases of oesophageal trauma, admitted under the care of one surgical team between 1949 and 1973, are reviewed. The importance of early diagnosis and an aggressive surgical approach in the management of a potentially lethal situation are stressed. In our opinion, spontaneous rupture of the oesophagus, instrumental perforation, open and closed traumatic lesions, and postoperative anastomotic leaks are, as far as diagnosis and management are concerned, different aspects of the same desperate surgical problem.

Oesophageal trauma is accompanied by a high morbidity and mortality rate if diagnosis and treatment are delayed.

Perforations of the cervical oesophagus may be treated conservatively.

Intrathoracic perforations demand an aggressive surgical approach; only exteriorisation followed by reconstruction at a later date offers a reasonable chance to save the life of the patient and ultimately restore continuity.

The records of all the patients suffering from oesophageal lesions admitted to the Regional Thoracic Surgical Unit at Frenchay Hospital, Bristol, under the care of one team between 1949 and 1973 have been reviewed. Of 2950 patients submitted to various oesophageal operations, 78 developed postoperative oesophagopleural fistulae, commonly referred to as 'leaks'. Included in this series are the patients treated by all the members of the team—consultants, registrars, and surgical house officers beginning their training in oesophageal surgery. In general, the incidence of postoperative morbidity is inversely proportional to the experience of the operator but in order not to confuse the overall picture no attempt has been made to apportion responsibility for these complications. In the past, 'selection' in the reporting of clinical material has been responsible for much confusion and misleading deduction.

Additionally, during the same period 32 cases of oesophageal rupture of various aetiologies were treated (Table 1).

Table 1 *Oesophageal injuries*

Spontaneous rupture	11
Instrumental perforation	15
Foreign body perforation	3
Traumatic rupture	3
Postoperative fistula	78
Chemical burn	16
Total	126

Spontaneous rupture of the oesophagus

During the 25-year period under review 11 patients with spontaneous rupture were investigated and treated (Table 2). The age range was 28 to 75 years; there was no predominance of incidence in either sex.

The commonest early symptoms were acute pain of sudden onset in the chest or epigastrium associated with dyspnoea, usually related to an episode of vomiting or a conscious effort to control it after dietetic indiscretions. However, rupture can occur almost silently without any preliminary oesophagogastric gymnastics. A young, healthy dental surgeon ruptured the middle third of his oesophagus into the right pleural cavity while watching what to him was an amusing television programme. In one patient the vomiting was caused by acute cholecystitis associated with obstruction of the cystic duct. Suppurative media-

Table 2 *Spontaneous rupture: 6 males; 5 females*

Symptom	
Chest or epigastric pain	10
Dyspnoea	10
Dysphagia	5
Shoulder tip pain	4
Sign	
Surgical emphysema (neck)	11
Pneumothorax	10
Shock	9
Pleural effusion	9
Fever	6
Empyema	3
Cyanosis	2
Septicaemia	2
Radiology	
Surgical emphysema (neck)	11
Pneumothorax	10
Pleural effusion	10
Positive oesophagogram	9
Mediastinal emphysema	7
Pneumoperitoneum	1

stinitis was already present on admission. Total oesophagectomy, with cervical oesophagostomy and feeding jejunostomy, was performed together with drainage of the gall bladder; four months later cholecystectomy and total oesophageal reconstruction with left colon were performed successfully. This case demonstrates the necessity for a complete, accurate, preoperative diagnosis.

The important diagnostic sign is surgical emphysema in the neck, secondary to mediastinal emphysema. This sign was present in every patient at the time of admission to hospital. Unfortunately, it is frequently missed, especially after the 'silent' or less dramatic onset of symptoms when the patient has usually been admitted with an erroneous diagnosis of myocardial infarction. In these circumstances there may be a delay of several days before the correct diagnosis is reached, by which time suppurative mediastinitis and empyema will have occurred.

A chest radiograph will reveal a pleural effusion and probably a pneumothorax on the side into which the oesophagus has ruptured. In six patients the rupture was in the lower third into the left pleural cavity, but in three patients the rupture was in the middle third and involved the right pleural cavity. In one of these, an over confident diagnosis had been made, the chest radiograph had been omitted, and an abortive left thoracotomy was performed.

An oesophagogram with contrast medium, preferably iodised oil, is obligatory to confirm the diagnosis and locate the site of the rupture. In nine of 10 patients thus examined the leak was clearly demonstrated.

Early diagnosis is essential to the success of conservative surgical treatment. In six patients the correct diagnosis was made within 24 hours and in four of these immediate thoracotomy and primary repair of the oesophagus were carried out. In two patients the rupture was so extensive that no repair was possible; in one an oesophagogastric resection was followed by an intrathoracic oesophagogastrostomy, and in the other patient exteriorisation and secondary reconstruction with left colon at a later date was necessary. Three of the four patients treated by primary suture of the rupture developed postoperative complications—recurrence of the leak with subsequent chronic empyema in two patients and a broncho-oesophageal fistula in one. Of the five patients whose diagnosis had been delayed for 24 hours or longer, three were treated by total resection and exteriorisation followed by a secondary interposition procedure with left colon two months later with excellent results. 'Conservative' procedures, pleural drainage, and feeding jejunostomy were carried out in two patients with unsatisfactory results; one patient died of sepsis a week later and the other developed a chronic empyema (Table 3).

Our experience suggests that if diagnosis is delayed, if the rupture is extensive or the oesophageal tissues are infected, oedematous, and friable, or if suppurative mediastinitis is already present, then the only treatment likely to save the patient's life is exteriorisation followed by secondary reconstruction at a later date. By 'exteriorisation' is indicated total resection of the intrathoracic oesophagus, leaving the patient with a cervical oesophagostomy to prevent the aspiration of saliva, and a feeding gastrostomy, the cardia having been closed. The results have fully justified this extended or staged surgical pro-

Table 3 *Treatment of spontaneous rupture*

Treatment	*Diagnosis*[1]		*Result*[2]		
	A	*B*	*Recovered*	*Comp.*	*Died*
Primary repair by suture	4		1	3 (recurrent leaks)	
Resection, oesophagogastrostomy	1		1		
Exteriorisation, staged reconstruction	1	3	4		
'Conservative treatment'		2		1	1

[1] Diagnosis: A (early < 24 hours); B (late > 24 hours).
[2] Recovered: Uneventful convalescence.
Complicated: Ultimate recovery after convalescence with complications.
Site of rupture: Thoracic—Middle third 3 (right side)
—Lower third 6 (left side)
Abdominal 1
Not localised 1

gramme for a condition that until recently had been regarded as invariably fatal.

Instrumental perforation of the oesophagus

The common cause of instrumental perforation of the oesophagus is the passage of some form of oesophagoscope or dilator; less commonly the organ may be damaged during an operation on the mediastinum or hilum of the lung when difficulty is encountered during the dissection. It is impossible to ascertain the global incidence of instrumental perforation as few of these catastrophies are reported, for obvious reasons.

This report concerns 15 cases of instrumental perforation. In the Regional Thoracic Surgical Unit at Frenchay Hospital, between 1949 and 1973, there were 12 cases of instrumental perforation in a consecutive series of 5900 oesophagoscopies, an incidence of 0·2%. Three patients were referred from other hospitals with an established diagnosis of instrumental perforation. In all cases the rigid, open-ended oesophagoscope (Belsey type) was used. Many of the operations were performed by junior surgical staff in training. Although local anaesthesia was employed in the great majority of these 5900 examinations, it may be significant that, in nine of the 12 cases perforated, general anaesthesia had been used, possibly reflecting the greater need for gentleness in the unconscious patient. In eight patients the perforation occurred during a diagnostic examination and in seven cases during the dilatation of a stricture (Table 4). The stricture was malignant in four patients and benign in three, including one case of fibrous stenosis secondary to epidermolysis bullosa and another secondary to scleroderma. In three patients the perforation occurred at the site of a pharyngeal diverticulum. Since it became apparent as a result of this experience that oesophagoscopic examination carries an additional risk in this situation, the procedure is now contraindicated when a known diverticulum is present or when the barium swallow examination fails to reveal the state of the cervical oesophagus.

Table 4 *Instrumental perforations*

Total cases 15	Frenchay Hospital 12 (oesophagoscopies 5900; incidence 0·2%) Other hospitals 3						
Anaesthesia: Local	6	Procedure:		Diagnostic	8		
General	9			Therapeutic	7		
Oesophageal lesion:		Oesophagogram:		Site:			
Neoplasm	4	Positive	9	Cervical	6		
Hiatus hernia	3	Negative	2	Mid third	3		
Diverticulum	3	Not performed	4	Lower third	6		
Benign stricture	3						
Other	2						
Treatment:			Recovered		Complicated result		Died
Cervical-conservative			4		2		
Thoracic							
Repair by suture			2				
Emergency resection and reconstruction			2				2
Exteriorisation, staged reconstruction			3				

During diagnostic oesophagoscopy the common sites for the perforation were the cervical oesophagus (40%) or the lower end in the region of the inferior sphincter (40%). Early diagnosis is both necessary and possible, even during the course of the examination. If the operator becomes 'lost' and is confronted by unfamiliar appearances, then he is almost certainly outside the oesophagus. The instrument is withdrawn immediately and the diagnosis is confirmed by an oesophagogram with iodised oil. The rapid appearance of pain in the chest, tachycardia, surgical emphysema in the neck, pneumothorax, dyspnoea, and upper abdominal rigidity secondary to mediastinal irritation give the typical clinical picture of perforation. The oesophagogram was positive in nine of 11 cases; this investigation was not performed in four other patients in whom the site of the perforation was obvious at the time of the examination.

Treatment of instrumental perforations is dictated by three factors—the site of the perforation, the underlying pathology calling for oesophagoscopy, and the speed of diagnosis. Perforation of the cervical oesophagus occurred during diagnostic examinations. All six patients were treated by 'conservative' measures—nil by mouth, intravenous fluids, and broad-spectrum antibiotics. In four patients spontaneous healing occurred uneventfully; in two, a local upper mediastinal abscess required drainage through the neck and was followed by a transient salivary fistula which closed within a few days.

Intrathoracic perforations call for emergency thoracotomy. Immediately the diagnosis has been confirmed the patient is prepared for thoracotomy. Ideally, a one-stage repair of the perforation, with interrupted inverting sutures of stainless steel wire, is combined with the originally planned operation for the underlying lesion. In two cases, repair of lower-third perforations was combined with repairs of the hiatal hernias. In four patients with neoplasms of the middle third, the perforation resulted from attempted bouginage; an emergency

oesophagectomy and one-stage reconstruction with intrathoracic oesophagogastric anastomosis was performed in all four cases. Two patients recovered (one is alive, well, and free from recurrence four years later) but two succumbed, one from coronary thrombosis and the other from anastomotic problems. In three patients with benign strictures in whom one-stage reconstruction was contraindicated by gross mediastinitis despite early diagnosis, exteriorisation of the oesophagus was followed by left colon interposition at a later date, with satisfactory results.

In retrospect, all the cases of intrathoracic perforation with gross underlying oesophageal pathology causing stenosis would have been treated more satisfactorily and more safely by exteriorisation and staged reconstruction with left colon. The prognosis following conservative management of intrathoracic perforations, by pleural drainage and feeding jejunostomy, has been observed to be very poor in terms of survival.

Postoperative anastomotic fistulae

Anastomotic complications are the commonest cause of death after major oesophageal surgery and must be included in any survey of oesophageal trauma. Prevention is dependent upon an awareness of the causes of anastomotic leaks but even then is not always possible owing to the inherent vulnerability of the organ to trauma. Some of these factors are recognised and accepted:

1 Anatomical factors: the fragility of the oesophageal tissues and the absence of a serosal layer.
2 Infection of the suture line resulting from severe oral sepsis.
3 The use of irritant suture material.
4 Multilayer suture techniques involving the strangulation and devitalisation of too much tissue; only living tissue can heal.
5 The considerable intraluminal pressures that can develop in the oesophagus during normal peristaltic activity.
6 Inadequate surgical techniques resulting in failure to obtain meticulous mucosal inversion and apposition.
7 Vascular trauma from the use of intestinal clamps.
8 Inadequate mediastinal and pleural drainage.
9 In the very old and very young patients the oesophageal tissue may be too fragile to tolerate any form of suture technique.
10 The design of the reconstruction after oesophageal resection is probably the single most important factor influencing the incidence of anastomotic leaks. In a series of over 500 oesophageal reconstructions performed in a 10-year period by one surgical team using the same anastomotic technique in all cases, the incidence of leaks was:
(a) after intrathoracic oesophagogastrostomy: 12%
(b) after cervical oesophagogastrostomy: 6%
(c) after left colon interposition: 1%.

The intrathoracic oesophagogastrostomy is notoriously the most dangerous form of reconstruction in terms of postoperative mortality and morbidity; moreover, it is responsible for the least satisfactory long-term functional results. Autodigestion at the suture line by peptic secretion is probably the major factor initiating the catastrophic train of events culminating in the death of the patient in most cases. By contrast, the patient who develops a leak after a cervical oesophagogastric anastomosis will probably survive if there is adequate drainage of the local infection.

In spite of the recognition of these causative factors and attempts at prevention by their eradication or control there will unfortunately remain a significant number of cases where the survival of the patient will depend upon early diagnosis and aggressive surgical intervention.

During the period under review 2950 oesophageal operations were performed by one team employing standardised techniques with continuity of clinical control (Table 5). The incidence of oesophageal fistulae was 2·6%. In a series of 921 oesophageal reconstructions involving some form of anastomosis the incidence was 7·7%.

The clinical features observed in this series of cases are presented in Table 6. Pain in the chest of sudden onset 48 hours or longer after the operation associated with tachycardia, rapid deterioration in the patient's general condition, and a massive pleural effusion were the cardinal signs suggesting a leak. Confirmation emerged from the nature of the fluid—thin, brown, sanious,

Table 5 *Postoperative fistulae*

Operation	*No. of patients*	*No. of fistulae*	*Incidence %*
Hiatal hernia repair	1825	7	0·38
Oesophageal myotomy	235	—	—
Reconstruction			
with stomach or jejunum	486	49	10
with left colon	267	4	1·5
Primary anastomosis for congenital atresia	129	17	13
Local resection and anastomosis	8	1	12·5
Total	2950	78	2·6

Table 6 *Postoperative fistulae*

	No. of cases
Clinical signs	
Fever	68
Chest pain	60
Persistent tachycardia	58
Shortness of breath	57
Pleural infection	28
Upper abdominal pain	12
Acute respiratory failure	10
Pericarditis	7
Dysphagia	7
Cervical abscess	6
Salivary fistula	4
Acute shock	3
No clinical evidence (occult leak)	7
Chest radiograph	
Atelectasis	55
Pleural effusion	53
Pneumothorax	31
Mediastinal emphysema	18
Mediastinal widening, pericardial effusion	24
Oesophagogram	
Positive	44
Negative	16
Not performed (fistulae revealed at routine postoperative barium swallow)	7

foul-smelling—and the radiographic demonstration of a fistula by an oesophagogram with iodised oil. This examination is obligatory whenever a leak is suspected, even when the signs are insidious in onset and less dramatic in intensity. The lipiodol swallow was positive in 44 of the 60 patients in whom it was used. Rarely, a leak may remain silent or 'occult' and be discovered in a routine postoperative barium examination. This manifestation is of clinical interest but little importance as spontaneous healing without further treatment is usual (Table 7).

Treatment of postoperative cervical 'leaks'

There were eight instances of anastomotic problems after cervical oesophagogastrostomy; all were treated conservatively with antibiotics and temporary intravenous feeding. In six of the eight patients spontaneous healing occurred uneventfully except for mild local infection. One patient developed a temporary salivary fistula; another succumbed to acute mediastinitis.

Treatment of intrathoracic 'leaks'

By contrast, the 'conservative' management of leaks after intrathoracic oesophagogastrostomy proved disastrous. In 40 patients treated by pleural drainage, by either catheter or rib resection, feeding jejunostomy or gastrostomy, and wide-spectrum antibiotic therapy, the mortality rate was 50%. In 15% a chronic empyema developed, and in only 35% was healing ultimately obtained after long periods in hospital and numerous revisions of the drainage.

In five patients in whom the diagnosis had been made early, before gross mediastinitis had occurred, an emergency thoracotomy was carried out with the object of closing the leak. The results were equally disappointing and in only one of the five was satisfactory repair achieved. In four patients the leak recurred within a few days.

In view of these recurring failures a more aggressive policy was adopted, and in six patients, where early diagnosis of the complication appeared to justify the procedure, the entire anastomosis was resected, all devitalised tissue was removed, and the anastomosis was reconstructed. In only one patient was a satisfactory result obtained; in the remainder the leak recurred with a fatal outcome in 50%. The pathology of this catastrophic complication remains baffling. In most cases the size of the pleural effusion and the fulminating nature of the mediastinitis were out of all proportion to the size of the anastomotic dehiscence, usually a minute defect due apparently to the failure of a single suture. The use of multilayer anastomotic techniques does not reduce the risk of this complication. In view of the findings at thoracotomy and the apparent technical simplicity of the problem, the unacceptable results and high mortality and morbidity are doubly disappointing.

In a further series of 12 cases where late diagnosis and the presence of severe suppurative medi-

Table 7 *Treatment of postoperative fistulae*

Type and site of leak		*No. of cases*	*Treatment*	*Recovered*	*Complicated*	*Died*
'Occult':	Cervical	1	None	1		
	Thoracic	5	None	5		
	Abdominal	1	None	1 (100%)		
Obvious:	Cervical	8	Conservative	6	1	1
	Thoracic	5	Repair of fistula	1	1	3
		6	Resection of anastomosis and reanastomosis	1	2	3
		12	Exteriorisation and staged reconstruction	7	3	2
		40	Drainage and feeding gastrostomy or jejunostomy	14	6	20

astinitis appeared to contraindicate any attempt at repair or reconstruction of the anastomosis, exteriorisation of the remainder of the oesophagus, cervical oesophagostomy, closure of the gastric remnant, and feeding gastrostomy were performed, followed by staged reconstruction with left colon interposition two to three months later. Surprisingly, the results in this group were the best obtained with a mortality rate reduced to 17%.

In retrospect the probable answer to this problem is to avoid an intrathoracic oesophagogastric anastomosis whenever an alternative form of reconstruction is technically possible. If a leak occurs, then our experience suggests that the only way of saving the patient's life is by exteriorisation and staged reconstruction.

Oesophageal perforations associated with retained foreign bodies

In three patients a retained intraluminal foreign body was directly or indirectly responsible for a perforation (Table 8). In one patient a middle third perforation, diagnosed within four hours of its occurrence, was treated successfully by primary suture. A second patient in whom spasm of the lower sphincter secondary to gastro-oesophageal reflux had caused impaction of a piece of mutton bone and subsequently perforation in the region of the cardia, was subjected to early thoracotomy, suture of the perforation, and synchronous repair of the hiatal hernia; convalescence was uneventful. In the third patient the perforation had occurred during attempts to remove a piece of bone from the lower oesophagus at another hospital 48 hours previously. The patient, aged 64, also had a known hiatal hernia; mediastinitis was present on admission. The oesophagus was exteriorised and a reconstruction with left colon was performed two months later with a satisfactory final result. This patient would probably have succumbed to a more 'conservative' surgical approach.

Oesophageal rupture due to trauma

Of the three patients included in this group (Table 9), two were transferred to the unit with perforations resulting from intraoperative accidents, a cervical laceration occurring during subtotal thyroidectomy, and an abdominal perforation complicating vagotomy. The first patient developed a cervical abscess and salivary fistula but spontaneous healing followed conservative management. The second patient, a man of 59 years, developed acute abdominal pain soon after the operation; a radiological examination after an iodised oil swallow revealed a leak into the mediastinum and peritoneal cavity. An emergency left thoracotomy and oesophagogastrectomy with intrathoracic anastomosis was followed by an uneventful convalescence and recovery.

The third patient, a young man aged 21 years, sustained a steering wheel injury during a car accident. The extent of the intrathoracic trauma did not become apparent for 48 hours when an iodised oil swallow revealed an oesophagotracheal fistula. Emergency right thoracotomy disclosed an 8 cm rent in the membranous part of the trachea and virtual destruction of the upper third of the oesophagus. The trachea was repaired and the oesophagus resected with cervical oesophagostomy and feeding gastrostomy. One month later

Table 8 *Foreign body perforations*

Sex	*Age*	*Foreign body*	*Other lesions*	*Level*	*Delay in diagnosis*	*Treatment*	*Result*
F	56	Denture		Mid third	4 h	Primary suture	Recovered
M	36	Bone	Hiatus hernia	Lower third	6 h	Primary suture and hiatus hernia repair	Recovered
F	64	Bone	Hiatus hernia	Lower third	48 h	Exteriorisation and staged reconstruction	Recovered

Table 9 *Oesophageal rupture due to trauma*

Sex	*Age*	*Cause*	*Clinical signs*	*Oesophagogram*	*Treatment*	*Result*
M	21	Car accident	Surgical emphysema; dysphagia; aspiration	Tracheo-oesophageal fistula	Tracheal repair; exteriorisation; staged reconstruction	Recovered
F	51	Thyroid operation	Cervical abscess; salivary fistula	Leak from cervical oesophagus	Conservative	Recovered
M	59	Vagotomy	Acute abdomen	Leak in mediastinum and peritoneum	Oesophagogastrectomy	Recovered

the oesophagus was reconstructed by a long isoperistaltic transplant of left colon. The final result was satisfactory.

Corrosive burns of the oesophagus

During the period under review 16 cases of severe stenosis resulting from chemical burns of the oesophagus were admitted for radical surgical treatment. Six of the children and three of the adults had swallowed caustic soda accidentally; in the remaining seven adults the trauma resulted from abortive attempts to commit suicide. In every case repeated attempts had been made to dilate the stricture without success so these cases must be regarded as a selected group; obstruction was severe or complete in all. The treatment adopted was resection of the stenosed segment with synchronous reconstruction of the oesophagus by an isoperistaltic transplant of left colon; in most cases the entire oesophagus had to be replaced owing to the extent of the stenosis. There was one operative death due to anoxia resulting from an anaesthetic error; in the remaining 15 patients convalescence was uneventful, and normal swallowing was restored.

Discussion

The successful management of spontaneous rupture of the oesophagus depends upon two factors—early diagnosis and complete diagnosis, not only of the location and extent of the rupture but also of the cause of the vomiting that precipitated the rupture. Regrettably, the common mistaken diagnosis of myocardial infarction stems from failure to examine the patient for surgical emphysema in the neck and for a pleural effusion, failure to request an immediate radiograph of the chest, and failure to perform an iodised oil swallow examination of the oesophagus. Suppurative mediastinitis will be present within 12 hours of the accident, more especially in the presence of dental sepsis, which plays an important role in prognosis as far as survival is concerned. The ideal treatment consists of early diagnosis and location of the rupture, and emergency thoracotomy with primary repair of the rupture within 12 hours of the event. In the presence of established mediastinitis, severe dental sepsis, and severe inflammatory oedema in the wall of the oesophagus adjacent to the rupture, primary suture will fail and exteriorisation followed by staged reconstruction offers the only opportunity for saving the patient's life. So-called 'conservative' treatment by catheter drainage of the pleura and antibiotic therapy carries a 50% mortality rate in our experience, with the alternative of prolonged hospital treatment for pleural suppuration and feeding by jejunostomy. There have been no fatalities after staged reconstruction.

The treatment of instrumental perforations is determined largely by their position. The incidence of cervical perforations can be reduced by refraining from performing oesophagoscopies on patients with pharyngeal diverticula, and by preoperative barium examination of the cervical oesophagus to exclude the presence of 'silent' pouches. The mortality rate from perforations can be reduced by the routine elimination of all dental sepsis before the examination.

Cervical perforations can be treated conservatively—no oral feeding, intravenous alimentation, and antibiotics. Eighty per cent will heal spontaneously; in 20% a localised cervical abscess will develop and require open drainage. Perforations of the intrathoracic oesophagus demand immediate thoracotomy, repair of the perforation, and synchronous correction of the lesion for which the examination was being performed. For example, if the perforation occurred at the cardia in a patient with an hiatal hernia, then an immediate definitive repair should follow closure of the defect, the fundoplication buttressing the suture line. When the perforation occurs during the dilatation of a stricture, an emergency resection of the stricture and immediate reconstruction by the method of choice according to the pathology of the stricture has been found to provide the most satisfactory results.

The prevention of postoperative anastomotic fistulae depends to a greater extent on the design of the reconstruction than on the anastomotic technique employed. The incidence of fistulae after the three forms of reconstruction mainly employed in this series was 12% after intrathoracic oesophagogastrostomy; 6% after cervical oesophagogastrostomy; and 1% after interposition of left colon. In all three techniques a similar single-layer anastomosis with non-absorbable suture material, in this case monofilament stainless steel wire, was used. The high risk of 'leaks' and postoperative mortality after intrathoracic oesophagogastrostomy has prompted the abandoning of this technique in favour of the other two techniques whenever possible. The probable cause of the leak is autodigestion of oesophageal tissue at the suture line when gastric secretion seeps along a suture under the influence of the negative intrathoracic pressure. The low incidence after the interposition of left colon reflects the benign, non-erosive nature

of the mucus secreted by the colon.

The established leak when occurring within the chest presents a desperate clinical situation to which the patient commonly succumbs. Emergency thoracotomy, suture of the fistula, or refashioning of the anastomosis is rarely successful in salvaging the patient's life. In the authors' experience the only hope for the recovery of the patient from this complication rests in an aggressive approach to the problem—exteriorisation of the remaining oesophagus, closure of the stomach and its return to the abdomen, cervical oesophagostomy, feeding gastrostomy, and staged reconstruction at a later date with left colon. This policy is justified only when the prognosis for survival from the original pathology of the obstructing lesion is hopeful.

In view of the lower operative mortality and morbidity, and in spite of its greater complexity, the left colon interposition procedure should now replace intrathoracic oesophagogastrostomy as the reconstruction of choice even after resections for malignant strictures where the long-term prognosis is always in doubt. The cervical oesophagogastrostomy can be reserved for those patients in whom the use of colon is contraindicated by intrinsic colonic disease or mesenteric endarteritis associated with systemic hypertension. Fistulae from a cervical anastomosis frequently heal spontaneously after drainage of the local infection.

In the case of neglected or undiagnosed fistulae where the patient's condition is critical, nothing can be done other than pleural drainage and feeding jejunostomy. Few patients will survive this situation.

Oesophageal perforations associated with retained foreign bodies result either from failure to detect non-radio-opaque foreign bodies or attempts at endoscopic removal of very irregular articles. These may be partial dentures or, in the case of children, the sharp-edged metal obturators torn from the tops of beer cans, which now litter the countryside. If endoscopic removal cannot be effected safely and without undue manipulation, then emergency thoracotomy and removal by oesophagotomy before infection has supervened is the safer course of action.

Accidental injuries to the oesophagus during the course of any operation on the mediastinum should be identified and repaired immediately, and a feeding jejunostomy added if doubt exists regarding the efficiency of the repair. Owing to the frequency of incompetence of the lower oesophageal sphincter in apparently normal people, feeding gastrostomies are contraindicated in any situation where it is deemed advisable to 'rest' a damaged oesophagus while healing occurs. Unsuspected oesophageal perforations will be quickly revealed by the early appearance of mediastinal emphysema or pneumothorax in the immediate postoperative period.

Extensive ruptures of the oesophagus can complicate closed chest injuries as a result of sudden compression between the sternum and thoracic spine. Early diagnosis may be obscured by the severity of the associated injuries, but if the possibility is considered the diagnosis can be quickly and easily confirmed by chest radiography after the swallowing of 10 to 20 ml of iodised oil. Unlike barium, residual oil in the mediastinum causes no complications. Staged reconstruction may be indicated by the severity of the oesophageal trauma if the patient survives the other injuries. In the case of penetrating wounds of the thorax a perforation may be suspected from the trajectory of the bullet or knife. As with all oesophageal injuries, early diagnosis, before the inevitable mediastinitis, and early repair are the key to the survival of the patient. The risk of fibrous strictures secondary to wounds of the oesophagus is proportional to the number of surgical assaults on the mediastinum in attempts to restore continuity; the first, if correctly performed, is that most likely to succeed.

Corrosive burns of the oesophagus result from the accidental or suicidal ingestion of caustic soda, ammonia, or concentrated acid solutions; in both situations, prevention is difficult and beyond the scope of this report. The prognosis is determined within seconds of the injury by the depth of the burn, as in the case of thermal injuries to the skin. No first-aid treatment is likely to influence the course of the inflammatory response; the outcome varies from minor, commonly multiple strictures amenable to eventual dilatation to complete oesophageal stenosis. There is no conclusive evidence that steroid therapy influences the subsequent fibrosis. Two practical problems ensue—the value, if any, of early dilatation, and the choice of resection and reconstruction or bypass for the severe, undilatable stricture. Too early attempts to pass dilators through an acutely inflamed, ulcerated oesophagus are merely adding further trauma to existing trauma and are illogical. The full extent of the damage will not become apparent for two to three weeks; this is the time to oesophagoscope the patient and decide whether dilatation is possible and likely to relieve the dysphagia, or whether radical surgical treatment is indicated after preliminary gastrostomy. Contemporary at-

tempts to prevent stenosis by the early use of plastic stents still await evaluation.

The outstanding problem remains the choice between resection and bypass in the management of severe, undilatable, corrosive strictures. The disturbing factor is the increase in reports of malignant degeneration in these strictures, mostly emanating from Eastern Europe. The available evidence to date favours one-stage synchronous resection and reconstruction in preference to bypass. Contrary to previously held views, resection of the corrosive stricture is technically easier than the resection of the chronic peptic stricture as the fibrosis is confined to the mucosa and submucosa; perioesophagitis and mediastinitis are present only in those patients in whom ill-advised and over-aggressive attempts to dilate the stricture have resulted in rupture of the oesophagus.

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