beyond and symptoms urgently require treatment. It has the advantage over radiotherapy that it can be given in any hospital. Unpleasant side-effects of the treatment can be minimized by the use of pentobarbitone sodium and chlorpromazine.

I wish to thank Professor L. J. Davis for his helpful advice in the preparation of this paper. Part of the expenses for this work was borne by the Rankin Fund of the University of Glasgow.

REFERENCES

- Brown, A., and Davis, L. J. (1949). *Glasg. med. J.*, 30, 197.
 Davidson, M., Smithers, D. W., and Tubbs, O. S. (1951). *The Diagnosis and Treatment of Intrathoracic New Growths*, p. 127. Oxford Med. Publ., London.
 Oswald, N. C. (1956). *British Medical Journal*, 1, 761.
 Smithers, D. W. (1951). In Davidson, Smithers, and Tubbs's *Diagnosis and Treatment of Intrathoracic New Growths*, p. 188. Oxford Med. Publ., London.
 Wintrobe, M. M., Cartwright, G. E., Fessas, P., Haut, A., and Altman, S. J. (1954). *Ann. intern. Med.*, 41, 447.

PERFORATION OF PEPTIC ULCER OF THE OESOPHAGUS INTO THE PERICARDIAL CAVITY

REPORT OF A CASE

BY

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Chronic peptic ulcer of the oesophagus is a well-recognized complication of those types of diaphragmatic herniae which allow regurgitation of acid gastric contents into the oesophagus (Jackson, 1929; Dick and Hurst, 1942). Like peptic ulcers elsewhere, if untreated they may perforate. Because of the usual anatomical situation (on the postero-lateral aspect) perforation occurs most commonly into the mediastinum or into one or other pleural cavity (Friedenwald *et al.*, 1929; Coffey and Dravin, 1951; Gent, 1951). Very rarely the aorta (Christopherson, 1917) or the pulmonary veins (Barrett, 1950) are involved. We describe here a case in which the ulcer was situated on the anterior wall and perforation took place into the pericardial cavity.

Case Report

A sales representative first came under the care of one of us (K.S.) in 1943, when he had a duodenal ulcer, radiologically proved. In 1948, at the age of 58, he was found to have a "sliding" diaphragmatic hernia. In September, 1949, a small pocket of peptic ulceration was seen radiologically on the posterior wall of the lower end of the oesophagus. Oesophagoscopy confirmed the presence of the ulcer. In May, 1951, he was operated upon. An oesophageal ulcer and short oesophagus were found in association with a diaphragmatic hernia. The hernia was repaired and the stomach replaced in the abdomen. The diaphragm was fixed to the oesophagus at a higher level. Left phrenic crush and double vagotomy were also carried out.

After this the patient felt well and regained weight until, in October, 1951, he began to get more dyspepsia, associated with pain in the left shoulder. A further barium-meal examination showed that reflux was still occurring from stomach to oesophagus, although there had been no recurrence of the hernia.

In January, 1953, one of us (K.S.) was called to see him because of symptoms of general malaise and pain in the left shoulder. His temperature was 99.2° F. (37.3° C.), pulse

80 a minute and regular. He was thought to be suffering from a mild influenzal infection and was advised to remain in bed. At 2 a.m. the following day he woke up suddenly with severe retrosternal pain. When seen at 4.30 a.m. he presented the clinical picture of coronary thrombosis. An injection of $\frac{1}{2}$ gr. (22 mg.) of "omnupon" was given. Three hours later he was still deeply shocked and in great pain, and an unusual murmur was then heard throughout the precordial area. It could not be separated from the heart beat. One had the impression that liquid was rushing through a narrow opening, the sound being comparable to that of water passing through a narrow gorge. His blood pressure, previously about 170/110, was now 140/80.

An electrocardiogram taken sixteen hours after the onset of the pain showed no evidence of myocardial infarction. Nevertheless, the differential diagnosis was thought to lie between: (1) myocardial infarction, (2) dissecting aneurysm, and (3) perforation of a peptic ulcer of the oesophagus or perforation of an ulcer in a reformed hiatus hernia. The patient's general condition was such that open exploration or any other investigation could not be considered. The murmur persisted unchanged throughout the day until death occurred, about 21 hours after the onset of the severe pain.

Necropsy was performed sixty hours after death (D.D.G.). On opening the chest the pericardial sac was found to be distended, and 100 ml. of greenish-black liquid, containing semi-digested food particles, was removed. Two small perforations were found in the posterior part of the sac just to the left of the inferior vena cava (Fig. 1), through which more of the greenish-black liquid was seeping. The flow of this liquid (which was later seen to be identical in appearance with the stomach contents) could be increased by pressure on the stomach. At the lower end of the

oesophagus, on its anterior aspect, was found a pear-shaped ulcer (5 by 5 by 4.5 cm.) 2 cm. deep, four-fifths of which lay about the level of the attachment of the diaphragm (Fig. 2). The edges of the ulcer were thickened and overhanging. The central part of the floor was extremely thin and two perforations were seen (0.8 by 0.5 and 0.2 by 0.2 cm.). A probe passed through these emerged in the pericardial sac.

FIG. 1.—The heart has been lifted forward to show the perforations, through which a probe is lying.

FIG. 2.—The posterior wall of the oesophagus has been cut to show relationship of ulcer to cardia. A probe lies through one of the perforations. A piece of suture material appears in the inferior edge of the ulcer.

Around the edge of the ulcer were found three sutures attaching diaphragm to oesophagus. Both pleural cavities contained 250 ml. of fibrinous liquid but no food particles. A well-developed Meckel's diverticulum was present in the ileum. No other significant abnormalities were found.

Histological examination of sections from the edges of the ulcer was made difficult by the extent of post-mortem change which had taken place. The appearances were compatible with peptic ulceration; no evidence of malignancy was found. The ulcer floor consisted of vascular fibrous tissue. In a section from the lower edge gastric mucous membrane was seen. In sections from the upper edge no epithelium was recognizable: they included two thick, well-defined layers of smooth muscle.

Comment

A search of the literature has yielded very few reported instances of oesophageal lesions forming communications with the pericardial cavity. Parkes (1849) described the first case, due to trauma, in a sword swallower aged 19, fit and athletic. His patient also developed, some hours before death, a "friction sound both diastolic and systolic . . . mixed with . . . a peculiar kind of metallic rhonchus carried up to the top of the sternum." At post-mortem examination it was found that the sword had penetrated the oesophagus and passed into the pericardium at the angle formed by the reflection of the sac from the right auricle.

The second case was described by Trotter (1852), the cause being "simple ulceration." Rose (1883) reported a case in a boy of 13, the oesophageal lesion being a "silent ulcer." Fairen (1927) claimed to have collected seven cases from the literature, but he gave no references, and we have been able to find only the above three cases reported prior to his paper. He himself added two more—one following the ingestion of lye, and one due to a malignant ulcer.

The next case was that of Imperatori (1928), who found a fistula between oesophagus and pericardium in a child of 13 months who had swallowed an open safety-pin. Barrett and Franklin (1949) described the perforation into the pericardium of an ulcer occurring in the oesophagus after oesophago-jejunostomy.

Finally, Viollet (1954) has described a case in a patient very similar to ours who had had dyspepsia for years and at necropsy was found to have oesophagitis and a benign ulcer just above the cardia. All these cases were fatal. In only four was the perforation due to peptic ulceration.

The diagnostic clue in our patient, and in Parkes's (1849), was the peculiar murmur heard over the praecordium, the significance of which was not appreciated at the time. If it had been, the question of surgical intervention and pericardial toilet would have had to be considered: certainly conservative measures would appear to have no part to play in the treatment of the condition.

Summary

A case is described of perforation of a peptic ulcer of the oesophagus into the pericardium. Only four such cases have been reported previously.

We wish to thank Dr. A. M. Barrett, of the Department of Pathology, University of Cambridge, for his help with the post-mortem examination, and Dr. L. B. Cole for reading the manuscript.

REFERENCES

- Barrett, N. R. (1950). *Brit. J. Surg.*, **38**, 175.
 — and Franklin, R. H. (1949). *Ibid.*, **37**, 194.
 Christopherson, J. B. (1917). *Lancet*, **1**, 369.
 Coffey, J. H., and Dravin, I. (1951). *Amer. J. dig. Dis.*, **18**, 285.
 Dick, R. C. S., and Hurst, A. (1942). *Quart. J. Med.*, **11**, 105.
 Fairen, V. (1927). *Progr. Clin.*, **35**, 164.
 Friedenwald, J., Feldman, M., and Zinn, W. F. (1929). *Amer. J. med. Sci.*, **177**, 1.
 Gent, J. C. (1951). *J. roy. nav. med. Serv.*, **37**, 44.
 Imperatori, C. J. (1928). *Laryngoscope (St. Louis)*, **38**, 268.
 Jackson, C. (1929). *J. Amer. med. Ass.*, **92**, 369.
 Parkes (1849). *Trans. path. Soc. Lond.*, **2**, 40.
 Rose (1883). *Berl. klin. Wschr.*, **20**, 826.
 Trotter, J. W. (1852). *Trans. path. Soc. Lond.*, **3**, 316.
 Viollet, G. (1954). *Mém. Acad. Chir. (Paris)*, **80**, 593.

FOETAL EXSANGUINATION ASSOCIATED WITH SURGICAL INDUCTION OF LABOUR

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In our experience it is not unusual for a blood-stained tap to be obtained when labour is induced by surgical rupture of the hind-waters. That this accident may be associated with damage to foetal vessels in the placenta and consequent serious foetal blood loss has been brought home to us by the deaths of two babies within the past few months.

Surgical induction of labour is generally regarded as a minor obstetric procedure, and, provided it is carried out with care, accidents to mother or baby are not expected. We record here the clinical details of our two cases because the complication that we describe carries a very real threat to the life of the child.

Case 1

The patient, a 5-gravida aged 29, was admitted to hospital when 37 weeks pregnant because of albuminuria and hypertension. One week after admission it was decided to induce labour by rupturing the hind-waters. The membranes were swept and an attempt was made to rupture the hind-waters with a Drew Smythe catheter. A few millilitres of blood was obtained and the procedure was abandoned. The foetal heart sounds were checked at once and afterwards at regular intervals, but no distress was noted. Once or twice during the next 24 hours there was a small ooze of blood from the vagina. Twenty-eight hours after the attempted induction of labour the foetal heart suddenly became irregular and a lower-segment caesarean section was performed at once. A live child, weighing 7 lb. 8 oz. (3.4 kg.), was delivered. The baby looked pale and the haemoglobin level at 20 minutes was 70% (10.3 g.). The maternal aspect of the placenta was quite normal, but on the foetal side there was a blood clot of 2 to 3 oz. (60 to 85 ml.) lying under the membranes. When the blood clot was gently removed it was found to surround a puncture wound in one of the large foetal vessels. A portion of the blood clot was examined by a modification of the method described by Singer, Chernoff, and Singer (1951) and proved to be foetal blood.

The baby, at the age of 1 hour, was given a blood transfusion, and although the child's condition did initially improve it became very oedematous and died at the age of 52 hours. No urine had been passed, nor had any been obtained on catheterization. Post-mortem examination revealed extensive tubular necrosis in both kidneys, and death was considered to have been due to shock following upon haemorrhage.

Case 2

The patient, a 6-gravida aged 34, was admitted to hospital at term on account of mild pre-eclamptic toxæmia. Six days after admission surgical induction of labour was attempted.