CASE REPORT

The patient, a 3-gravida 2-para aged 25, was admitted to hospital at 6 p.m. on January 9, 1949, as a case of ruptured ectopic pregnancy. Her last pregnancy occurred four years previously. She had experienced acute pain in the right iliac fossa on rising early that morning; she had felt faint and sick and had returned to bed. The pain had persisted and spread across the lower abdomen to the left side. Twice during the day she had been sick, and on trying to get up the feeling of faintness had returned. There had been no shoulder-tip pain, vaginal bleeding, or urinary symptoms. The date of her last menstrual period was December 8, 1948, and she was normally regular every 28 days for 3–4 days. It was unusual for her period to be a few days late in this way.

The patient was pale and shocked, showing shallow rapid respirations. Her pulse was weak and 88 per minute; her blood pressure was 110/70. Abdominal examination showed guarding across the lower abdomen, maximal in the right iliac fossa, with tenderness in the same region. Shifting dullness could be elicited. On vaginal examination there appeared to be a doughy mass in the right fornix, which was displacing the uterus to the left, but tenderness was pronounced and the examination was not conclusive. Although there was no vaginal bleeding, the patient's general condition, together with the abdominal and vaginal findings, supported a diagnosis of intraperitoneal bleeding from ruptured ectopic pregnancy, and laparotomy was decided upon.

At operation the abdomen was found to contain about 2 pints of clotted and liquid blood. No lesion of the uterus or tubes could be seen. On the surface of the right ovary was a small vessel running across a retention cyst which had evidently ruptured. The vessel was bleeding steadily. The small cyst was resected from the remaining ovarian tissue, the bleeding controlled, and the ovary sutured. Although a careful search by palpation was made of the rest of the abdomen, no further lesion was discovered which might have caused the bleeding, and it was concluded that this tiny vessel was to blame. The abdomen was closed and the patient returned to the ward with an intravenous blood drip running.

An uninterrupted recovery was made. There was some lower abdominal pain on the first and second days following operation, but thereafter her stay in hospital was uneventful. She was discharged on the fourteenth day.

Pathological Report.—The small cyst removed proved to be a corpus luteum showing some areas of haemorrhage.

Whilst haemorrhage of this magnitude would necessitate laparotomy in any event, it might be of value to know that rupture of a corpus luteum or Graafian follicle can cause severe bleeding; thus prolonged and extensive search for other causes, to the detriment of the patient, might be avoided.

C. J. DEWHURST, M.B., M.R.C.O.G.

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Rupture of Oesophagus during Childbirth

Spontaneous rupture of the oesophagus has often been reviewed, from the first reported case in 1723 of Baron de Wassenaer, the Grand Admiral of Holland, by Hermann Boerhaave, to the last review by Barrett in 1946. The most common site for perforation is just above the diaphragm, longitudinally, in the left postero-lateral aspect of the oesophagus. The commonest error at necropsy is failure to dilate the oesophagus with water *in situ* and thus demonstrate the rupture. The following case was associated with a prolonged labour, a cause not previously recorded.

CASE REPORT

The patient, a married woman aged 37, was pregnant for the first time. No history of oesophageal disease or abnormality was obtained. The expected date of delivery was June 5, 1949, but the pregnancy proceeded normally until June 11, when labour started. She made no mention of pain in the chest, but it must be remembered that fairly strong labour pains were occurring and she was a little mentally distressed, apart from being physically tired. A moderate dyspnoea developed some 36 hours before delivery and abdominal distension had occurred about the same time. In view of the fact that the dyspnoea was most marked when lying down, abdominal distension was blamed. On June 15 at 2.45 p.m. the temperature rose to 101° F. (38.3° C.). Intramuscular penicillin was given, and by 8 a.m. on June 16 the temperature had dropped to 97.2° F. (36.2° C.). Previously at 1 a.m. the patient had had a mild shivering attack, and a catheter specimen of urine showed a well-developed urinary infection.

By 9 a.m. on June 16 the cervix was fully dilated and the contractions were powerful. Delay in the second stage occurred: ethyl chloride and open ether were given and forceps applied. A healthy female infant was born at 12.35 p.m. A very severe post-partum haemorrhage of approximately 80 oz. (2.27 l.) followed. After the anaesthetic the patient failed to recover consciousness. The breathing was stertorous and the lips and nails were cyanotic. Three pints (1.7 l.) of blood and two bottles of plasma were given, but at 12.45 a.m. on June 17 death occurred.

A post-mortem examination was carried out twelve hours after death. Abdominal distension was marked, but cyanosis was not a feature. No surgical emphysema was present. On opening the abdomen the distension was found to be due to a large amount of gas in the small intestine. The uterus had contracted satisfactorily and the placental site was clean. In the chest, the whole of the left thoracic cavity was filled with a slightly offensive dirty brown fluid. The right side contained approximately half a pint (280 ml.) of clear pale-yellow liquid. Inflation of the oesophagus was not carried out, but 1 in. (2.5 cm.) above the cardia on the left postero-lateral aspect a rupture was demonstrated. Blackening and autolysis of the oesophagus and posterior mediastinum were such that its exact size was difficult to define, but it was in the neighbourhood of ½ in. (1.25 cm.). In the posterior oesophageal wall, running longitudinally upwards, was a thin pouch of mucous membrane about ¾ in. (1.9 cm.) in depth. The mouth of this pouch was situated at the site of rupture and from its structure and position was doubtless due to a splitting of the epithelium and was not congenital in origin. An absence of epithelium around the tear was found by Collis, Humphreys, and Bond (1944) which could not be demonstrated on the opposite wall of the oesophagus. There was no evidence of previous oesophageal abnormality. The lower lobe of the left lung was completely collapsed and the upper lobe partially so. Bronchopneumonic changes, confirmed histologically, were present in both lobes. The mediastinitis was practically confined to the posterior mediastinum and there was no evidence of surgical emphysema.

I consider this case to be one of spontaneous perforation. However, it is possible that the degree of autolysis present may have hidden some other underlying pathology that could not be demonstrated at necropsy. Both from the history and from the post-mortem appearances the rupture doubtless occurred about 48 hours before death.

I wish to express my thanks to Mr. H. Burt-White for permission to publish this case, and to Dr. L. H. D. Thornton and Dr. E. M. Martland for their advice and criticism.

H. W. H. KENNARD, M.B., B.Chir.,
Assistant Pathologist, Salisbury Area Pathological Service.

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