the baby was removed from the mother as soon as possible it seems probable that the infection was acquired by contact at birth rather than due to intrauterine transmission. Of particular interest is the myocarditis in the infant. Saphir and Wile (1942) first drew attention to the occurrence of myocarditis in poliomyelitis, but it is by no means constant. Saphir (1945) described myocarditis in 10 of 17 patients examined after death. The second case reported by Baskin *et al.* showed histological evidence of severe acute and healing myocarditis. The lesions in the heart in the case here reported were widely separated and could not be described as severe, the intervening muscle being normal. The child died from bulbar paralysis and not myocardial failure.

REFERENCES

Baskin, J. L., Soule, E. H., and Mills, S. D. (1950). *Amer. J. Dis. Child.*, **80**, 10.
Saphir, O. (1945). *Amer. J. Path.*, **21**, 99.
—— and Wile, S. A. (1942). *Amer. J. med. Sci.*, **203**, 781.
Severin, G. (1939). *Nord. Med. (Hygiea)*, **1**, 55.

Medical Memorandum

Spontaneous Rupture of the Spleen

A case of spontaneous rupture of an apparently normal spleen is recorded because of the comparative rarity of such a condition. Cases have been reported from time to time, and about 40 cases have been recorded within the past 20 years.

CASE REPORT

An agricultural labourer aged 46 was admitted to hospital on January 8, 1951, at about 1 a.m. He had felt off colour for about a week, and had had a headache followed by repeated bleeding from both sides of the nose. Two days before admission he felt pain in the epigastrium after moving some milk-churns, but this passed off, leaving him feeling as though he had influenza. He had some pain in the back all next day and stayed in bed, still thinking he had influenza. The same night at 10.30, while in bed, he felt a sudden severe pain beneath the left costal margin and in the praecordium. He got out of bed and fainted. He was admitted as a case of possible perforated gastric ulcer. In his own words, he had always been "as fit as a fiddle." However, on closer questioning he admitted having been ill with malaria for 10 days in 1926 while serving in the Army in India; there had been no recurrence.

On examination he looked pale and ill; he had marked dyspnoea, and felt faint when sat up in bed, but did not seem notably anaemic, being rather dark-skinned. His temperature was 98° F. (36.7° C.), pulse 100, and respirations 25. The heart sounds were faint, the rhythm was regular, and the pulse beat was soft. There were a few crepitations at the left base, with slightly diminished breath sounds and impairment of the percussion note. The abdomen was very rigid all over the epigastrium and deep palpation was not possible. The liver dullness was not altered, and bowel sounds were present. There was no shifting dullness. He had been able to drink tea without nausea or any worsening of his pain.

Perforated gastric ulcer was not thought to be the diagnosis, but possibilities considered were early diaphragmatic pleurisy, coronary thrombosis, or, possibly, acute pancreatitis. Operation was not carried out at once because it was thought he would not survive a laparotomy without resuscitation. *X*-ray films of the chest and abdomen taken later in the morning showed no abnormality, and a leucocyte count showed total cells, 11,000 per c.mm. (polymorphonuclears 75%, eosinophils 2%, basophils 1%, monocytes 5%, lymphocytes 17%). Perforation was therefore thought to be most unlikely, and the patient was kept under observation. His condition did not deteriorate, and he took tea by mouth without discomfort or vomiting. The next day his condition was slightly better, but he still had the abdominal pain and rigidity. The bowel sounds were also present. At a consultation with the medical specialist, Dr. A. H. Banton, coronary thrombosis and pleurisy were excluded. As the patient was now more fit a laparotomy was arranged. After preliminary intravenous saline he was taken to the theatre with the drip *in situ*—the tentative diagnosis being acute pancreatitis.

Operation.—On February 9, under thiopentone, nitrous oxide, and curare anaesthesia, a midline upper abdominal incision was made. The peritoneal cavity was found to contain free blood. This was traced to a spleen surrounded by enormous clots. Accordingly blood was transfused instead of saline, and the approach to the spleen was rapidly improved by a transverse incision through the left rectus muscle. The bleeding had taken place between the spleen and its capsule, and the huge clots were mostly intracapsular. After separating these a normal-sized spleen came to view; the pedicle was ligated and the spleen removed after separating the adhesions which were anchoring the capsule to the diaphragm. The spleen looked normal and appeared to have bled from its surface, there being no appreciable rupture in its substance. The adhesions of the splenic capsule to the diaphragm were remarkable for small pearly nodules of fibrous tissue of an icing-sugar appearance. After removing much blood from the peritoneal cavity the abdomen was closed. Altogether 5 pints (3.3 litres) of blood was transfused into the patient, who made a quick and uneventful recovery.

Pathological Report (Dr. E. J. Harries).—"Spleen of about normal size. Capsule shows slight perisplenitis. On section, dark red. Some blood clot remains attached to outside of capsule. Also fragments of tissue from diaphragm. Microscopical examination: The splenic capsule is thickened, but is lost over most of the organ. The haemorrhage seems to have been mainly subscapular but has split the capsule, leaving a thin 'pseudo-capsule' limiting the parenchyma. Stains reveal free iron in and on each side of the capsule, suggesting that the haemorrhagic accidents have at least a slightly longer history than the clinical history suggests. The parenchyma shows reticulum hyperplasia but no definite evidence of specific disease such as malaria. The arterial system in the organ appears normal. The diaphragmatic fragments are similar to the thickened capsule described, and are probably adhesions which have in the past separated and become smoothed off."

COMMENT

There was evidence of previous disease only so far as the perisplenic adhesions are concerned. Furthermore, it is reasonable to assume that the capsule may have been torn from the spleen while the patient was lifting the milk churns, though this was a normal part of his work. The bleeding was slow until the capsule ruptured 36 hours later; the severe pain then came on, associated with signs of shock. These findings fit in with those of Babson and Morgan (1946). It is possible also that the perisplenic adhesions may have originated during a short attack of malaria, 25 years previously, though this was not a pathological malarial spleen in any other sense. A perusal of the literature of the past eight years suggests that there may be two pathological types of spleen which rupture spontaneously: those with intrasplenic haemorrhages in a physiologically engorged spleen or a spleen with diseased vessels, the haematoma separating the capsule late; and those which produce extrasplenic haemorrhages from the surface of the spleen, separating the capsule early. Both types occur either with dramatic suddenness or with preliminary malaise, depending on how long the bleeding goes on before the capsule ruptures.

In the above case it is likely that the splenic capsule was torn from an engorged spleen by the trivial effort of moving a milk churn.

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REFERENCE

Babson, W. W., and Morgan, P. (1946). *Amer. J. Surg.*, **72**, 97.