

test was too painful to carry out properly. Palpation confirmed that the swelling extended from the symphysis to the umbilicus, and from the midline to the right as far as the linea semilunaris. The shape was oval, with the large diameter in the vertical axis. The swelling was smooth, firm, and tense, without impulse, extremely tender, and did not move with respiration. Percussion of the swelling gave a dull note. Rectal and vaginal examinations and urinalysis were negative.

The case resembled an acute surgical abdomen, and torsion of an ovarian cyst or torsion of the omentum was a possible diagnosis. As mentioned before, the shoulder-raising test was too painful to carry out and no deduction was made whether the swelling was in the abdominal wall or was intraperitoneal, but it seemed more superficial than would have been expected of an intraperitoneal lesion. However, operation was decided upon mainly because a serious intraperitoneal lesion could not be discounted.

**Operation.**—A right sub-umbilical paramedian incision over the swelling revealed a bulging and dusky-coloured rectus sheath. Dark, tarry blood clot exuded immediately the sheath was incised. A clot the size of a large grape-fruit was removed from in front of and behind a longitudinal split in the rectus muscle which extended from umbilicus to symphysis. There was considerable bleeding welling up from behind the rectus muscle in the region of the deep epigastric vessels, and this was only partially controlled by under-running sutures, so that packing was necessary. The wound was closed on either side of the pack.

The pack controlled the bleeding well and was removed after 36 hours. A full course of penicillin was administered. The wound healed satisfactorily and the patient was discharged 21 days later. The follow-up revealed no obvious abnormality of the rectus muscle, which functioned very well.

#### DISCUSSION

That trauma in some degree or other is the exciting factor in all cases appears certain. But where this traumatic factor is slight—e.g., coughing (the factor in this case)—then there will be a predisposing factor, and the main ones seem to be (1) senility, with its degenerative changes in the walls of blood vessels; (2) debilitating illness, with its degenerative changes in muscle; and (3) pregnancy, with its effect of stretching the muscle fibres of the rectus abdominis and the deep epigastric blood vessels.

Correct diagnosis is seldom made, the most popular one being torsion of an ovarian cyst. Diagnostic points are a history of paroxysm of coughing followed by pain, with the early appearance of a mass which may steadily increase in size and which becomes more prominent and fixed on raising the shoulders. Operative treatment is usually undertaken because of the doubt in diagnosis, but where a definite diagnosis of haematoma of rectus muscle is made then there is the choice of conservative treatment. If the haemorrhage is already considerable or is progressing operation should be undertaken; Teske (1946) gives an overall mortality of 4%, and Thomas (1945) states that there is a 13% mortality rate in the obstetric group.

G. H. MOORE, F.R.C.S. Ed.,  
Surgeon, North Lonsdale Hospital,  
Barrow-in-Furness.

#### REFERENCES

- Teske, J. M. (1946). *Amer. J. Surg.*, **71**, 689.  
Thomas, R. C. (1945). *J. Obstet. Gynaec. Brit. Emp.*, **52**, 580.

### A Case of Melaena Complicating Congenital Pyloric Stenosis

The following case of congenital hypertrophic pyloric stenosis is worthy of record on account of an unusual complication—melaena—occurring after operation.

#### CASE REPORT

A male infant aged 6 weeks, an only child, was admitted to the Royal Salop Infirmary on July 16, 1947. He had had projectile vomiting for over a week. He had been breast-fed, but his mother had given him complementary feeds of half-cream milk food after each feed for a few days. The weight had risen from 7 lb. 15 oz. (3.6 kg.) to 9 lb. 6 oz. (4.25 kg.) before the onset of vomiting, but had fallen to 7 lb. 4 oz. (3.29 kg.) on admission.

Congenital pyloric stenosis was diagnosed and operation was decided upon (July 21). The stomach was first washed out, and then under local analgesia a Rammstedt's operation was performed. There was a well-marked pyloric tumour. In separating the circular muscle

fibres the duodenal mucosa was inadvertently opened and was sutured with fine catgut. In view of this perforation an intravenous saline drip was set up and feeding by mouth withheld until July 23, when half-strength half-cream milk food was given, the drip being discontinued on July 24. Apart from two small vomits he made good progress until Aug. 1, when his weight was 8 lb. 3½ oz. (3.73 kg.). He then started vomiting bile-stained material, and this lasted two days, being relieved after gastric lavage and rectal saline. Feeding by mouth was resumed.

A temporary improvement occurred, but on Aug. 5 vomiting recommenced and the stools were relaxed. There had been no pyrexia throughout, and no signs of peritonitis. On Aug. 6 the stomach was washed out and rectal saline given. A course of phthalylsulphathiazole, one tablet four-hourly, was started. Next day he was given glucose feeds. A "dark brown" stool was reported. His weight was 7 lb. 8 oz. (3.4 kg.).

On Aug. 8 (the 18th day) the baby was collapsed, pale, and generally oedematous. A plasma transfusion was started. Soon afterwards a large melaena stool was passed. The haemoglobin was 30%. Blood was substituted for the plasma, and about 450 ml. run in slowly. Next day a remarkable improvement had occurred and the oedema had disappeared. The haemoglobin was 92%. Phthalylsulphathiazole was discontinued. After several dark stools the motions had now become a normal colour. Feeding was resumed with half-cream milk food. No further vomiting occurred. Apart from one melaena stool on Aug. 10 the motions remained normal. Unfortunately it was not possible to estimate the prothrombin index.

Further convalescence was uninterrupted, and the baby was discharged on Aug. 26 weighing 8 lb. 5 oz. (3.77 kg.). When seen again on Oct. 7, as an out-patient, his weight was 13 lb. 14 oz. (6.29 kg.) and his mother stated that he had remained well.

#### DISCUSSION

I have found only a few references to bleeding into the bowel in cases of pyloric stenosis. Meader (1936), quoting a case in which "occult blood" had been found in the stools on the seventh day—gradually decreasing after a few days—commented: "Now it is not unusual for these patients to vomit fresh blood within 24 hours following operation, or for some evidence of blood to appear in the first or second stool, but in our experience this phenomenon has not occurred as late as in this instance."

In my case the bleeding was severe, occurring on the 18th day, and was preceded by gastro-enteritis. It was presumably due to ulcer. Infection was probably an aetiological factor. Possibly trauma, particularly as the duodenal mucosa was punctured at operation and subsequently sutured, determined the location of the ulcer.

If infection and trauma are regarded as aetiological factors in peptic ulcer (Illingworth and Dick, 1941) it is surprising that this complication is not more common in pyloric stenosis, since infections of various kinds are not infrequent after operation and the mucous membrane is widely exposed after incising the seromuscular layers.

The importance of breast-feeding in pyloric stenosis in relation to prognosis is well established (Herzfeld and Wallace, 1935; Thompson and Gaisford, 1935; Tallerman, 1938; Levi, 1941; Williams, 1942). Levi (1941) reported a consecutive series of 100 breast-fed babies who underwent a Rammstedt's operation without a death, and contrasted this with a series of 46 babies who were bottle-fed, of whom five died—all from gastro-enteritis. Bottle-fed babies present greater risk of cross-infection owing to their longer stay in hospital; they are less well nourished and are more liable to suffer from dietetic disorders (Williams, 1942).

It is probably significant that my case was artificially fed immediately before and after operation.

I wish to thank Dr. A. D. Rope and Mr. A. D. Haydon, under whose care this case was admitted, for the opportunity of treating it.

H. C. FLETCHER-JONES, M.B., B.S., F.R.C.S.,  
Late Resident Surgical Officer,  
Royal Salop Infirmary.

#### REFERENCES

- Herzfeld, G., and Wallace, H. L. (1935). *Lancet*, **2**, 385.  
Illingworth, C. F. W. and Dick, B. M. (1941). *A Textbook of Surgical Pathology*, 4th ed. Churchill, London.  
Levi, D. (1941). *British Medical Journal*, **1**, 963.  
Meader, H. B. (1936). *Ohio St. med. J.*, **32**, 531.  
Thompson, W. A., and Gaisford, W. F. (1935). *British Medical Journal*, **2**, 1037.  
Tallerman, K. H. (1938). *J. Pediat.*, **13**, 787.  
Williams, H. (1942). *Med. J. Aust.*, **1**, 303.