35, and 39 weeks, and after delivery. Unlike other tests for folic-acid deficiency, which often give abnormal results in pregnancy, there is a steady decline in the amount of formimino-glutamic acid excreted in the urine throughout pregnancy. It is suggested that this is due to the utilization of increasing amounts of histidine for protein synthesis in pregnancy.

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PORTAL PHLEBOTHROMBOSIS IN THE PUERPERIUM: A REPORT OF THREE CASES

BY

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Mesenteric vascular occlusion has long been recognized as a cause of infarction of both small and large bowel. It was not, however, until the work of Warren and Eberhard (1935) that the venous type was clearly differentiated from the arterial and accepted as a distinct clinical entity. It invariably takes the form of thrombosis in the portal system and has been classified clinically (Skandalakis, 1958) as primary (idiopathic) or secondary. Few cases, however, will be regarded as idiopathic if all the possible aetiological factors are carefully assessed.

However, infarction of bowel resulting from thrombosis in the portal system during pregnancy and the puerperium has very rarely been reported, and only 11 cases were found in a review of the world literature. Of these, four followed abortions, two occurred during pregnancy, and four during the puerperium. One case was not proved. Only two cases seem to have been definitely primary in nature (Bucura, 1923; Heimrath, 1961). No survivors were reported.

Three further cases of portal phlebothrombosis during the puerperium are here presented.

Case 1

A housewife aged 27 attended the antenatal clinics of the Southampton Group of Hospitals during her third pregnancy in 1961. Her two previous pregnancies had terminated in abortion—the first in 1958 at 24 weeks and the second in 1960 at 8 weeks.

The third pregnancy was uneventful. Her haemoglobin was recorded as 83% on booking. She was admitted to the Southampton General Hospital at 38 weeks, and after a normal labour lasting 13 hours was delivered of a living female infant weighing 5 lb. (2.270 g.). The placenta was expressed after five minutes and the total blood loss did not exceed 300 ml.

The puerperium was uneventful and her haemoglobin was 71% on the fifth day. The child did not regain its birth weight until the 12th day after delivery, and the patient therefore remained in hospital. On the 17th day, when about to be discharged, she complained of continuous abdominal pain, which she said had been gradually worsening for 48 hours. That morning she had passed two loose stools of normal colour, and vomited her breakfast. She appeared well, but her temperature had risen to 99.4° F. (37.4° C.) and her pulse rate to 100. On examination there was definite tenderness with some guarding in the epigastrium and around the umbilicus, but there was no rebound tenderness and normal bowel sounds were heard. On vaginal examination the uterus was found to

be well involuted. The symptoms and signs remained unchanged during the day, but that evening the abdominal pain became generalized and much more severe, and several times she vomited bile-stained fluid. During two hours the pulse rate rose to 120, the blood-pressure fell from 120/80 to 90/50, and she was noted to be pale, cold, and sweating. Considerable abdominal distension was obvious and there were generalized guarding, tenderness, and rebound tenderness. No bowel sounds were to be heard.

An intravenous infusion of plasma was begun, gastric suction was carried out, and 500 ml. of blood-stained fluid was aspirated. After 1,500 ml. of plasma and 1,200 ml. of blood had been transfused her condition had improved sufficiently for laparotomy to be undertaken.

On opening the abdomen a large quantity of blood-stained fluid escaped, and numerous distended coils of small bowel of a dark reddish-black colour were seen. Patchy discoloration was found to commence about 2 feet (60 cm.) from the duodeno-jejunal flexure, and it was obvious that the distal jejunum, ileum, and caecum had undergone infarction. As normal pulsation was felt in the superior mesenteric artery and its branches, this was thought to be the result of venous thrombosis. The area of involved bowel was so extensive that resection was not thought practicable and the abdomen was closed.

Despite massive blood and plasma infusion, intravenous heparin, close electrolyte control, and, ultimately, intravenous hydrocortisone and noradrenaline drip, the patient's condition steadily deteriorated and she died 48 hours later.

At necropsy the abdomen was distended. Small bilateral serous effusions were present in the pleural cavities. The peritoneal cavity contained a blood-stained effusion and the surfaces of the lower jejunum, ileum, and caecum were dull, dark red in colour, and covered with a fibrinous exudate. A firm thrombus occluded the superior mesenteric and portal veins (Fig. 1). The vein walls appeared healthy. There was diffuse fatty infiltration of the liver parenchyma. Several pancreatic veins were occluded by thrombus, but no sign of pancreatitis was present. The uterus showed normal involution with no sign of inflammation, and the uterine veins were patent.

Case 2

A married woman aged 22 attended her own doctor during her fourth pregnancy in 1951. Her previous confinements had been normal.

On August 9, 1951, she had a normal delivery at home of a living full-term infant after a short labour. The puerperium was quite uneventful until the 13th day, when she had abdominal pain. This continued intermittently for 48 hours; it then became much more severe and she passed blood per

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rectum. She was admitted to Southampton General Hospital on August 24 in a state of collapse. On examination she was pale and sweating, with an imperceptible pulse and unrecordable blood-pressure. The abdomen was distended and there was marked tenderness, guarding, and rebound tenderness in all areas. After energetic resuscitation with dextran and blood, she improved enough for laparotomy to be performed.

On opening the abdomen a large quantity of blood-stained fluid escaped and several feet of small bowel were seen to be distended and of a dark reddish-black colour. Good pulsation was felt in the superior mesenteric artery, and the condition was believed to be due to thrombosis of the superior mesenteric vein. The infarcted bowel was resected and the abdomen closed. After a stormy convalescence she made a good recovery and was discharged eight weeks later. For six months she had frequent loose stools, but these gradually returned to normal.

She was interviewed in December, 1961, and said that, apart from minor coughs and colds, she had remained perfectly well. Her appetite was good and her bowels were regular and normal. She was somewhat overweight, but no abnormality could be detected on physical examination. The liver and spleen were not palpable and there was no detectable ascites. Liver-function tests were within normal limits.

FIG. 1.—Case 1. Thrombosis of the portal and superior mesenteric veins with infarction of the small bowel.

Case 3

The records of this case together with Fig. 2 were kindly supplied by the Department of Pathology of Cambridge University.

A married woman aged 39 years was admitted to Addenbrooke's Hospital on December 16, 1930, and died the same day. The history was that of a normal confinement on October 25. She had remained well until November 9, when she developed abdominal pain and breathlessness and also some swelling of the legs. These symptoms gradually improved, but the abdominal pain became very severe on the day of admission.

At necropsy extensive portal phlebothrombosis was found, which had caused haemorrhagic necrosis of the larger part of the small intestine (Fig. 2), only the first foot or so of the jejunum and the last few feet of the ileum escaping. The intestines were distended with blood and 700 ml. of deeply

blood-stained fluid was found in the abdomen. The liver weighed 1,440 g. Most of the liver substance was very pale, though irregular areas were shrunken and of a darker red colour. The spleen was normal and the uterus normally involuted. Sections of the liver showed areas of local necrosis with degeneration of the liver cells, mainly in the middle zone of the lobule.

FIG. 2.—Case 3. Thrombosis of the mesenteric veins with small-bowel infarction.

Discussion

Three factors, usually referred to as Virchow's triad, are concerned in the formation of every thrombus (G. Payling Wright, 1958). These are: provocative lesions in the vascular intima, changes in the velocity and character of the local blood-stream, and changes in the constituents of the blood. Nothing is known of factors influencing the first two in the normal puerperium, but a rise in platelet count together with an increase in platelet stickiness maximal on the 12th day has been demonstrated (H. Payling Wright, 1942).

In two of the above cases the symptoms were first complained of 15 and 13 days respectively after delivery. In one case, however, although the illness began 14 days after delivery, it was not until some five weeks later that infarction of bowel occurred. This is not as surprising as would appear at first sight, for portal phlebothrombosis can extend slowly and in a recurrent manner (Leger and Holmes, 1960). Indeed, the venograms of Hunt (1958) suggest that cavernomatous transformation of the portal vein can occur *pari passu* with extending thrombosis. Whether this sequence of events is ever initiated in the puerperium is unknown.

In association with pregnancy, portal thrombosis sufficient to cause intestinal gangrene would appear to be particularly dangerous. In none of the cases reported in the literature did the patient survive, and the survival of the patient reported here (Case 2) may be attributed to the successful resection of a large portion of the small bowel.

In conclusion, we consider that the sequence of events here discussed may occur more often than is apparent from a review of the literature. We suggest that similar cases be reported, so that an estimate of the true incidence of the condition can be made and the results of treatment assessed.

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