Haemorrhage Mimicking Intravascular Haemolysis

Str.—We were interested to read the letter of Dr. Jeanne D. Reeve (12 June, p. 654) on haemorrhage mimicking intravascular haemolysis. We had a similar case recently that caused considerable diagnostic difficulty since we did not recognize the possibility of an intra-abdominal haemorrhage simulating an episode of acute intravascular haemolysis. A brief description is given as we feel that this syndrome merits wider recognition.

The patient was a 31-year-old negro woman, who presented with lower abdominal pain for three days. She was three months pregnant and was admitted for observation with a tentative diagnosis of an ectopic pregnancy. She was observed for the next three days, during which time she had some further abdominal pain, started to pass small amounts of blood per vaginum, and was noted to have a haemoglobin of 6.4 g/100 ml. She was transfused with two pints of blood and a D. and C. was performed since she was then considered to have a missed abortion. Nine hours postoperatively she collapsed pulseless with no recordable blood pressure, but recovered following the administration of intravenous Rho-macrogol (0.5 ml) and dextrose and hydrocortisone. Postoperative vaginal bleeding was minimal and no explanation for this collapse was found at that time. Two days postoperatively she started to pass dark red urine and six days postoperatively she was transferred to Guy's for investigation.

In this case she did not appear obviously ill but still complained of intermittent lower abdominal pain, worse on micturition, and was noted to be mildly icteric. Investigation showed a haemoglobin of 8.7 g/100 ml, with a reticulo-cytosis of 20%. Total bilirubin was 4.5 g/100 ml and direct-reacting 1.5 mg/100 ml. Plasma haptoglobins were absent and methaemalbuminemia present. The urine contained free haemoglobin, haemosiderin, and excess urobilinogen. A sternal bone marrow aspirate showed marked erythropoietic hyperplasia. It was thought that she had had an episode of acute intravascular haemolysis but this could not be explained. There was no evidence for a mismatched transfusion; Coombs test, Ham's test, and Donath-Landsteiner tests were all negative; blood cultures grew no organism, malaria parasites were not found; haemoglobin studies were normal. Other postoperative tests included an examination of faeces for occult blood, liver function tests, plasma urea and electrolytes, plasma proteins, and chest x-ray. A midstream specimen of urine contained 30 leucocytes/mm³, but no bacterial growth.

Within a few days her haemoglobinuria cleared completely. Her haemoglobin rose gradually during the next two weeks to 9.8 g/100 ml and the reticulocyte count dropped to 7.5%. Postoperatively it was clearly shown that the patient had developed acute pain in the left iliac fossa with tenderness and signs of peritoneal irritation. At laparotomy there was much old blood in the pelvis and within the abdominal cavity was the placenta attached to the fimbrial end of the left fallopian tube. Postoperatively she made a rapid recovery and six weeks later had a normal haemoglobin, 14.4 g/100 ml, with no evidence of haemolysis.

In retrospect it seems likely that the characteristics of an acute intravascular haemolytic episode were due to the intra-abdominal haemorrhage, which presumably caused the episode of acute collapse and the haemoglobinuria. The absence of haptoglobins, the presence of methaemalbuminemia, and the urinary features were due to reabsorption of haem pigment from the peritoneum. We were unaware of this syndrome until we read the letter of Dr. Reeve and hope that this report will help to emphasize it.—We are, etc.,

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Familial Hyperparathyroidism

Str.—In their paper on familial hyperparathyroidism (10 July, p. 87) Dr. P. Marsden and colleagues agree that this condition probably represents a form of the syndrome of multiple endocrine adenomatosis. Many patients with this syndrome have a gastrin-producing tumour and the highest association is with parathyroid tumours.1 Surely an indication existed here to perform gastric secretory studies on these patients together with immunosases for circulating gastrin, as reliance on clinical and radiological findings for the diagnosis of a Zollinger-Ellison state is likely to be fallacious?

Likewise, an opportunity was not taken to assess the effect on gastric secretion of changing levels of serum calcium before and after parathyroidecemy.2 I am, etc.,

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1 Ballard, H. S., Frame, B., and Hartsock, R. J., Medicine, 1964, 43, 481.