Female Sterility Produced by Investigation

Sirm,—The letter of Dr. F. W. Wright and Sir John Brody (22 September, p. 632) prompts me to draw attention to another danger in the investigation of infertility in the female, possibly leading to subsequent tubal blockage.

Methylene blue is commonly used to demonstrate tubal patency during diagnostic laparoscopy. The usual preparation is methylene blue injection, U.S.P., 1% in water in 10-ml ampoules (Harvey Laboratories, Inc., Philadelphia). The pH is 6.07. I wish to emphasize that this should be diluted liberally. Because the colour is very strong 1 ml diluted in 1000 ml of sterile normal saline is sufficient to show clearly after spill into the peritoneal cavity.

An average injection would be 10 ml and if given undiluted, straight from the ampoule, can provoke an acute reaction with signs of pelvic peritonitis lasting for some weeks, presumably following a “chemical salpingitis.” There is also the problem of leakage into uterine cavity. The manufacturer’s leaflet relating to intravenous administration of undiluted dye specifies the precaution of slow injection over minutes of a dose calculated on a body weight basis and mentions adverse reactions of nausea, abdominal pain, precordial pain, dizziness, headache, profuse sweating, mental confusion, and the formation of methaemoglobin.—I am, etc.,

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Type IV Hyperlipidaemia in Cord Blood

Sirm,—Though Kwiterovitch et al. have recently reported triglyceride concentrations in cord blood averaging 37 mg/100 ml, with a standard deviation of only 15 mg, in 36 normal infants, evidence was published 11 years ago that a group of babies had relatively high triglyceride levels. Brody and Carlson measured triglycerides and other lipids in cord blood from 52 unselected newborn infants. The mean concentration of their plasma triglycerides was 0.38 mmol/l. (approximately 33 mg/100 ml) but the frequency distribution was significantly skewed to the right. This suggested that a standard deviation could not be calculated and values ranged up to 0.85 mmol/l or 74 mg/100 ml. The upper tail of the distribution might, if one wished, be called relative type IV hyperlipidaemia in the newborn.

Brody and Carlson suggested that genetic factors could be responsible for this phenomenon. However, the late weeks of pregnancy and birth itself are a time of stress and metabolic changes whose intensity varies between individuals. Some degree of fatty liver appears to be quite common in the newborn. Preformed lipoprotein, which carries the fatty acids to the tissues, dil Foto et al. suggested that triglycerides in plasma is the predominant lipoprotein in cord blood but its concentration falls off during the first few days of life, while that of β-lipoprotein increases. There are accompanying major changes in the fatty acid pattern of serum lipids. The phenomenon to which Dr. F. M. Martin and his colleagues draw attention (8 September, p. 544) is interesting and deserves further study, but we cannot yet assume that a proportion of newborns with relatively high triglyceride levels in cord blood will be the individuals who show the better known primary type IV hyperlipoproteinemia in middle age. Careful and prolonged follow-up is needed and it could yet turn out that the cause of “newborn type IV hyperlipoproteinemia” is obstetrical (in the broadest sense of the word) rather than genetic.

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Drug-Induced Platelet Antibodies

Sirm,—We read with interest the letter of Drs. C. Davidson and S. M. Manohitharajah (8 September, p. 545), who reported the presence of specific platelet antibodies in a patient who developed thrombocytopenia while receiving phenylbutazone therapy. We were interested in the incidence of drug-induced platelet dyscrasias it would appear to be more and more relevant to search for medicating antibodies and we would like to report another illustrative case. A 77-year-old woman presented with a 24-hour history of epistaxis and retinal bleeding, with spontaneous bruising for two weeks. She had been taking phenylbutazone regularly for 12 years to alleviate arthritic pain and for three years she had been taking quinoline sulphate as treatment for nocturnal cramps. We were unable to ascertain any recent alteration in dosage or proprietary brand. Examination revealed a pale woman with generalized bruising and purpura over the limbs and trunk. Petechial haemorrhages in the mouth and a flame haemorrhage in the left fundus were noted, together with evidence of recent epistaxis and retinal bleeding. There were no other abnormal clinical findings.

Her haemoglobin level was 9.7 g/100 ml, total leucocyte count 1,200/mm³, and platelet count 10,000/mm³. Bone marrow aspiration revealed a mildly hypochromic marrow with macronormoblasts and delayed maturation of granulopoiesis. Megakaryocytes showed reduced platelet budding. There was no evidence of bone marrow infiltration. Tests for antinuclear factor and L.E. cells were negative, as were Ham’s test and screening for urinary haemosiderin. Serum vitamin B₁₂ and folate levels were normal. The agglutination test was positive for leucocyte and platelet agglutinins but negative for group A and B. Three weeks after drug withdrawal her haemoglobin level had risen to 10.3 g/100 ml, total leucocyte count to 4,600/mm³, and platelet count to 170,000/mm³.

It would appear that the pancytopenia resulted from the combined effects of phenyl-