forms disorders in the evidence, natal haematopoietic stem the best of erythrocyte, granulocytic leucocyte, reticulum cell.

R. van Furth J. W. M. van der Meer

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1 Moore, M. A. S., and Metcalf, D., British Journal of Haematology, 170, 18, 279.

Grendon, Warwickshire.

KENNETH T. FARN

Reticulum Cell not a Haematopoietic Stem Cell

SIR,—In their article on myeloproliferative disorders in the series on blood and neoplastic diseases (16 November 1974, p. 399) Drs. S. M. Lewis and L. Szur state that the "reticulum cell is thought to be the haematopoietic precursor cell from which originate the erythrocyte, granulocytic leucocyte, and megakaryocyte." In a diagram they illustrate this view and suggest the relationship with myeloproliferative disorders. There is no experimental evidence, however, that the reticulum cell can be the haematopoietic stem cell. Ontogenetic studies showed that the haematopoietic stem cell originates in the yolk sac, is next present in the fetal liver, and finally in the bone marrow. To the best of present knowledge the pluripotent haematopoietic stem cell in postnatal life has the morphology of a small round cell like a small lymphocyte but quite distinct from that of the reticulum cell. The reticulum cell, on the other hand, is a large cell with specific morphological features and forms reticulin fibres.

In our view it is more appropriate to make a distinction between cells deriving from the haematopoietic stem cell and those from mesenchymal origin (see fig.) Such a distinction based on morphological, cytochemical, functional, and kinetic characteristics offers ample opportunity for the classification of myeloproliferative and other neoplastic diseases.—We are, etc.,

J. W. M. VAN DER MEER

BRITISH MEDICAL JOURNAL 9 AUGUST 1975 371

Pemphigus Induced by D-Penicillamine

SIR,—We have observed six cases of bullous cutaneous eruptions with features of pemphigus in patients who were (or had been recently) receiving d-penicillamine (DP) for seropositive rheumatoid arthritis. The clinical aspect of the eruption was in one case truly mucocutaneous pemphigus vulgaris and in the remaining five cases a purely cutaneous bullous disease, which presented three times as pemphigus seborrhoeicus and once as dermatitis herpetiformis. The diagnosis of pemphigus was based in all cases on: (1) the Tzanck cell test; (2) histological demonstration of acantholysis; and (3) circulating antibodies to intercellular substance, the titre of which was rather low except in one case in which it reached 1/1600.

The bullous disease developed during DP treatment in five cases (in one on resuming treatment after an interval). In the sixth patient the pemphigus started after DP had been withdrawn for several months owing to a previous non-specific bullous rash. (Another case, seen in collaboration with Dr. C. Grupper, showed a similar delayed onset.) The evolution of the disease followed two patterns: a benign one, rapidly stopped by moderate doses of corticosteroids, and a classical course, sometimes very difficult to control.

Before these six patients were seen at our clinic a previous case had been reported in a patient under treatment for Wilson's disease. Since our first reports of this condition and our further observations we have received verbal communication of three more French cases, making a total of 10 known cases of DP-induced pemphigus.

Two kinds of DP produced by three pharmaceutical firms share the similarity of inducing the 10 cases of pemphigus: French extractive DP (Hôpitaux de Paris); Knoll's extractive DP, and Bayer's synthesized DP.

The pathogenic implications of these data may afford a new approach to research on the aetiology of pemphigus.—We are, etc.,

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Sequela of Delayed Spontaneous Respiration in Breech Infants

SIR,—The current controversy about vaginal versus caesarean delivery for breech infants has led me to follow up infants from a series of 106 vaginal breech deliveries without death from tentorial tear to see whether infants who were kept alive by prompt intubation and prolonged positive pressure respiration (P.P.R.) developed gross long-term sequelae. The three who failed to breathe spontaneously within 20 min have all been traced and only one is grossly handicapped, being spastic and slightly mentally retarded at 3 years. This child was born at 40 weeks gestation (birth weight 3.7 kg) after concealed accidental haemorrhage, was shocked at birth, and was treated with P.P.R. for 30 sec. The second infant, who was born at 37 weeks (birth weight 2.35 kg) with a true knot in the cord and without a palpable apex beat. He was treated with cardiac massage and P.P.R. for 60 min. At 12 years he is of average intelligence with no sign of spasticity. The third infant was born at 34 weeks (birth weight 2.3 kg) with a prolapsed cord and received P.P.R. for 40 min. He had numerous childhood illnesses and was slow in developing speech and learning to read, but at 12 years he has overcome these difficulties, is tall and well built, and is "coltish" in his movements without being too clumsy. In the first case forceps were applied to the after-coming head; in the other two the Mauriceau-Smellie-Weit technique was used for delivery.

All four infants who failed to breathe spontaneously within 20 min after toxic accidental haemorrhage in Neligan's series, however delivered, developed cerebral palsy. (Our case, taken with his, leads us to the conclusion that one should desist from P.P.R. after 20 min in any infant after concealed accidental haemorrhage.)