hypertransfused rats, which, unlike the plasma iron in normal animals, is not further elevated following radiation (Gurney, C. W., and Taylor, K. T., unpublished results). It must be stressed that only effective erythropoiesis is being measured, and future investigation must also include a study of the blood-forming organs to assess the degree to which stem cells damaged by irradiation undergo differentiation and then succumb while still erythrocyte precursors.

In addition to affording a means for the simple quantitative determination of irradiation damage and recovery of a population of cells, we believe this model offers possibilities for investigation into the nature and kinetics of the stem-cell compartment, and it is for these reasons that these preliminary observations are reported.

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Development of the Islets of Langerhans in Man

Very little has been published about the differentiation of the α and β cells in the development of the pancreatic islets in man. The only work since the introduction of the modern granule stains is that of Ferner and Stoeckenius[1] (based on twelve fœtal pancreases), and Schultze Jena[2] (based on six fœtal pancreases).

The material for the present work was 36 pancreases from human fœtuses with weights ranging from 34–3,000 gm. (10 wk. gestation to full term), stained by Gomori's chrome-alum hæmatoxylin phloxin method[3] and/or by chrome-alum hæmatoxylin with Gomori's one-stage trichrome counterstain. Eleven pancreases were from fœtuses between 10–20 wk. gestation. It is the islets in these early months of gestation, in particular, which have not been adequately described before.

Four stages are observed in the development of the islets. These overlap so that any one pancreas will show islets in more than one stage :

(1) State of budding islets. In the third gestational month, clusters of islet-cells bud off from the ducts. Differentiated α and β cells are already present. The β cells form a central cluster, around which ungranulated islet-cells and occasional α cells are ranged.

(2) Bipolar stage. As the islets grow, the β cells become grouped at the tip of the islet farthest from the duct, while the α cells form a calyx at the base of the islet. The proportion of ungranulated cells diminishes. Islets in this bipolar phase are observed from the fifth to the eighth gestational month.

(3) 'Mantle-islet' stage. The α cells at the base of the islets proliferate and grow right round the β cells. The islet now has a kernel of β cells and a shell of α cells. Some ungranulated cells are still present among the α cells.

There is a good deal of overlapping between the bipolar and mantle-islet stages. Mantle-islets are seen as early as the fifth gestational month, and dominate the picture from about the sixth gestational month.

(4) Stage of mature islets. From the eighth gestational month onward, a few islets can be found where the α and β cells have the more-or-less haphazard distribution characteristic of the mature adult islet, although the majority are 'mantle-islets' right up to term and beyond.

A detailed account of this work, with photomicrographs, will be reported elsewhere.

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PATHOLOGY

Serological Tests for Toxoplasmosis

Interest in various serological tests for toxoplasmosis has increased considerably in recent years. Siim and Lind[1], in a preliminary communication, have recently described a flocculation test using toxoplasma antigen adsorbed on to acrylic particles. They stated that preliminary results of absorption experiments showed the existence of more than one toxoplasma antigen. The following experiments carried out in this Laboratory may be of interest.

A high-titre human antitoxoplasma serum was absorbed with toxoplasma-infected mouse peritoneal exudate at room temperature (20° C.). This removed almost all the hæmagglutinating, complement-fixing and dye-test antibodies. Absorption of the same serum overnight with human red cells coated with toxoplasma antigen (as used in the hæmagglutination test of Jacobs and Lunde[2]) removed the hæmagglutinating antibody but the dye test and complement-fixation antibodies were unaffected.

Control tests were set up using a normal human serum, absorbed with toxoplasma suspension, and a normal human serum absorbed with red cells coated with toxoplasma antigen. This experiment excluded effects due to elution of soluble toxoplasma antigen. Absorption of the antitoxoplasma serum with human unsensitized cells did not alter the titres of the dye, hæmagglutinating or complement-fixing antibodies. The results are summarized in Table 1.

The results of this experiment were substantiated by using a HeLa cell tissue culture toxoplasma antigen to absorb a hyperimmune serum from another patient. The effect of using a tissue culture antigen