secretion¹. In the present experiments, attempts were made to determine whether electrical stimulation of other areas would similarly result in mammary secretion.

Virgin female rats were injected subcutaneously daily with 10 µg. oestradiol for 10 days to develop their mammary glands. For the following 5 days, groups of rats were electrically stimulated for 30 sec. twice daily in the regions of the head, lumbar vertebrae, nasal mucosa and nipples. Control rats were injected twice daily with saline for 5 days. On the sixteenth day, the rats were killed, and the right inguinal mammary glands were removed for histological preparations. The mammary glands of the controls regressed to a bare duct system, whereas almost all the electrically stimulated rats showed maintenance of the mammary lobule-alveolar system and initiation of lactation. These results suggest that electrical stimulation of the central nervous system induces release of prolactin and adrenocorticotrophic hormone from the anterior pituitary, since both hormones are required to induce mammary secretion in the oestrogen-primed rats.

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HÆMATOLOGY

Anti-i : a New Cold Antibody

In 1956 Wiener and his colleagues¹ described an antibody occurring in a case of cold antibody hæmolytic anæmia which they called anti-I. The I antigen has an almost universal distribution, but five examples of i cells were found in testing 22,000 donors. It was considered that the Ii phenotypes arose by the action of two allelomorphous genes *I* and *i*, individuals of the phenotype *i* being *ii*.

More recently, Jenkins *et al.*² have described an English donor whose cells are *i* and whose serum contains a naturally occurring anti-I that is not auto. Using these cells, the antibodies in a number of other cases of cold antibody hæmolytic anæmia were also found to have I specificity. At the same time Tippet *et al.*³ have reported a number of blood samples that are extremely weak in I although not as weak as the *i* donor of Jenkins *et al.* The serum of these people also contains anti-I that is not auto.

The results of the above workers show clearly that *i* cells also contain some I, but an enormous gulf separates them from the I of normal adults. Cord-blood cells were also found to be extremely weak in I.

Recently, we encountered a remarkable antibody that appears to clarify the Ii relationship in human red cells. This antibody was found in the serum of a patient not yet diagnosed but probably suffering from a reticulosis.

The serum of this patient weakly agglutinated his own and 500 normal adult red cell samples at low temperature (average titre at 12° C. about 1 in 16). Tests with three examples of *i* cells and five examples of the slightly stronger variant described by Tippet *et al.* all gave strongly positive results with titres ranging from 1 in 32,000 up to 1 in 250,000, the three *i*

samples all giving maximum titres. More than 40 cord blood samples were tested and all gave strong reactions similar to adult cells of the *i* phenotype.

Repeated absorptions of the serum with various cells showed that a single antibody was present reacting powerfully with *i* and cord cells and weakly with adult I cells. Clearly this antibody has anti-*i* specificity. Electrophoretic studies have shown that the antibody is present in the β-globulin fraction of the serum.

Tests on a number of infants of various ages show that within 18 months of birth the *i* reaction becomes steadily weaker while the I reaction becomes stronger until the normal adult relationship of strong I and weak *i* is established. It appears that all normal adult and infant samples have some I and some *i*, and cells that are strong in I are weak in *i* and vice versa.

It is clear that the abnormality found in the cells of the rare adults of the *i* phenotype is not that they are *i*, for everyone is probably *i* for a few weeks of life, but that the I has failed to develop. This type of reaction surely cannot reflect the action of *I* and *i* as allelomorphous genes. We think that the abnormality found in these *i* adults is caused by the absence of a genetically controlled factor that is essential for the proper development of I.

Our results have shown that the anti-*i* serum is able to place people very precisely on the I scale in an indirect way by measuring the amount of *i* that they possess. In view of this the following terminology in the I group system is suggested:

Details of these results will be submitted for publication elsewhere.

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¹ Wiener, A. S., Unger, L. J., Cohen, L., and Feldman, J., *Ann. Intern. Med.*, **44**, 221 (1956).

² Jenkins, W. J., Marsh, W. L., Noades, J., Tippet, P., Sanger, R., and Race, R. R., *Vox Sang.*, **5**, 97 (1960).

³ Tippet, P., Noades, J., Sanger, R., Race, R. R., Sausais, L., Holman, C. A., and Futtner, J., *Vox Sang.*, **5**, 107 (1960).

Ageing of Hæmoglobin

THE problem of ageing of protein molecules has been considered by several authors. Kunkel¹ in man, Ranney² in the rabbit, have attempted to demonstrate the ageing of hæmoglobin, taking advantage of the metabolic heterogeneity of hæmoglobins described by us in 1950³. They observe, indeed, during the life of the red cells, an equalization of the specific activities of hæmoglobin fractions which were unequal initially. These authors were using iron-59, and it is known that iron is re-utilized after the death of the red cells: this prevents observation of an eventual crossing of the specific activity curves of different fractions in the course of the ageing of the