

red cells. While both develop marked activity only after coagulation has occurred, the activity exerted by red cells before coagulation has occurred is much weaker than is that of platelets. When, therefore, platelet activity of other source is minimal and when other conditions are unfavourable to coagulation, the development of red cell activity tends to be blocked. In the thrombin generation test coagulation is retarded, because the blood has not been exposed to surface activation. In the thromboplastin generation, test dilution of the constituents retards coagulation. In both cases when red cells are present, but no platelets, the red cell activity develops only slowly. By varying conditions, however, red-cell activity can easily be demonstrated in both types of test.

The difference in behaviour of platelets and red cells makes a quantitative comparison impossible, unless the conditions are specified. Moreover, since the removal of all platelets tends to block the development of red-cell activity, it might be thought that this activity would be unimportant under physiological conditions. This requires further investigation, but so far the evidence obtained tends to indicate that red cells contribute to coagulation under natural conditions and are partly responsible for the normal coagulation time found in thrombocytopenic conditions. Certain clinical differences between the hæmorrhagic states of thrombocytopenia and of coagulation defects, such as the difference in frequency of hæmarthroses, may be explicable in terms of this red-cell action.

JAMES A. INGLIS  
JUNE W. HALLIDAY

Department of Pathology,  
University of Queensland.

<sup>1</sup> O'Brien, J. R., *J. Clin. Path.*, **12**, 45 (1959).

<sup>2</sup> Macfarlane, R. G., and Biggs, R., *J. Clin. Path.*, **6**, 3 (1953).

### Thalassæmia in an English and in a German Family

REPORTS of thalassæmia (major and minor) in British residents of the United Kingdom are few<sup>1-6</sup>. Equally few are reports of the occurrence of this abnormality in subjects of German extraction<sup>7-14</sup>.

Recently, we have been studying the frequency of the carrier form of this abnormality (thalassæmia minor) in subjects of Greek, Egyptian, Syrian, Armenian, Italian, Ethiopian and Sudanese (Northern, 'Arab' and Southern, 'Negro') origin, using, as an absolute criterion, the presence of an increased erythrocyte osmotic resistance associated with abnormal morphological characteristics of the erythrocytes<sup>15,16</sup>. During our testing of healthy school children in Khartoum for this purpose we detected one English and one German girl whose erythrocytes repeatedly showed an increased osmotic resistance and, on unstained peripheral smears, anisopoikilocytosis and microcytes. Both girls (aged nine years and thirteen years respectively) were in good general health and appeared normal on clinical grounds. The abnormality was not found in fifteen other British and five other German children tested.

Similar studies carried out on the parents of the English girl (their only child) revealed that both carried the same abnormality and that both were in good general health. The mother was born in Cornwall and was of mixed Spanish-Irish-Cornish stock; she has a brother and a sister both of whom are in

good health. The father was born in Malta of British parents. There is a possibility that there was some French admixture in his ancestry.

Examination of both parents of the German girl and of one brother showed that the father was carrying the abnormality and that the mother and brother were normal. The mother was born in Kothlen, Anhalt, had a fair complexion and looked and felt well. She was of 'pure' German stock. The father was born in Korrig, Saarburg, and was of a dark complexion with heavy bushy eyebrows. He had suffered from malaria and had occasionally complained of easy fatigability. A recent hæmoglobin test yielded results within normal limits. His sister and his mother are said to have a similar dark complexion. His family had had close ties in Lorraine some two centuries ago.

The evidence of ties with continental Europe in the ancestry of the three parents having the thalassæmia abnormality in these two families raises the possibility that the abnormality entered both these families through their foreign antecedents. It is difficult to prove this point in the absence of any systematic work on the frequency of this abnormality in Spain and France as well as in Great Britain and Germany. Chernoff<sup>17</sup> lists only five references in the literature to thalassæmia in Spanish subjects, while there are only two reports of this abnormality in French subjects<sup>18,19</sup>.

It is to be hoped that the significance of the thalassæmia abnormality in these countries will emerge in the not too distant future.

F. VELLA  
S. A. IBRAHIM

Department of Biochemistry,  
Faculty of Medicine,  
University of Khartoum.

<sup>1</sup> Grinnan, A. G., *Amer. J. Roentgenol.*, **34**, 297 (1935).

<sup>2</sup> Bush, C. E., *Brit. J. Radiol.*, **10**, 491 (1937).

<sup>3</sup> Bywaters, E. G. L., *Arch. Dis. Childhood*, **13**, 173 (1938).

<sup>4</sup> Israels, L. G., Suderman, H. J., and Hoogstraten, J., *Lancet*, **ii**, 1318 (1955).

<sup>5</sup> Israels, M. C., and Turner, R. L., *Lancet*, **ii**, 1363 (1955).

<sup>6</sup> Havad, C. W. L., Lehmann, H., and Scott, R. B., *Brit. Med. J.*, **i**, 304 (1958).

<sup>7</sup> Graser, E., *Z. f. Kinderh.*, **62**, 698 (1941).

<sup>8</sup> Freudenberg, E., and Esser, M., *Ann. Paediat.*, **158**, 128 (1942).

<sup>9</sup> Rohr, K., *Helv. Med. Acta*, **10**, 31 (1943).

<sup>10</sup> Heilmayer, L., Muller, W., and Schuboth, H., *Klin. Wochschr.*, **29**, 333 (1951).

<sup>11</sup> Pribilla, W., *Deutsch. Arch. Klin. Med.*, **198**, 223 (1951).

<sup>12</sup> Martin, H., *Verhandl. Deutsch. Gesellsch. Inn. Med. Kong.*, **58**, 728 (1952).

<sup>13</sup> Schaefer, R., *Monatsschr. Kinderh.*, **102**, 108 (1954).

<sup>14</sup> Middlebrook, J. E., *New England J. Med.*, **255**, 815 (1956).

<sup>15</sup> Silvestroni, E., and Bianco, I., *Amer. J. Human Genet.*, **1**, 83 (1949).

<sup>16</sup> Silvestroni, E., and Bianco, I., *Abnormal Haemoglobins*, 242 (C.I.O.M.S. Sympos., Blackwell Sci. Pub., Oxford, 1959).

<sup>17</sup> Chernoff, A. I., *Blood*, **14**, 899 (1959).

<sup>18</sup> Christiaens, L., Carlier, C., Crampon, and Goudemand, M., *Arch. Franc. Pediatr.*, **7**, 575 (1950).

<sup>19</sup> André, R., Bessis, M., Dreyfus, B., Jacob, S., and Malassenet, R., *Rev. Hémat.*, **13**, 31 (1958).

## GERONTOLOGY

### Expected Rate of Senescence and Age-dependent Mortality in Fish

It is usual in fisheries investigations to assume that the mortality of wild fish from natural causes does not rise significantly with age, and calculations on this basis give reliable results for most economically important species<sup>1-3</sup>. Age is sometimes invoked to explain the disappearance of fish<sup>4</sup>: at high ages a number of species show a decline in numbers which