by the increase in the cell counts (Table II). As determined by the Student t-test, the spleens of the mouse group II contained significantly ($p < 0.05$) more cells between the 4th and 14th day after immunization than those of the corresponding control group I. Rather reduced cell numbers were detected per 100 mg wet spleen weight in the pertussis-treated mouse group II. This suggests that the multiplication of spleen cells is not alone responsible for the elevation of the spleen weights. As to the spleen weights of 20-month-old mice, 2 findings should be especially noted: 1. The average spleen weights of the senile mice were sometimes higher, as compared to those of the 3-month-old controls. The same applies to the spleen indexes (Table I) and cell counts (Table II). 2. In the aged mouse groups III and IV the spleen weights, spleen indexes and cell counts differed considerably, as is evident from the relatively high standard errors. Contrary to expectations, the injection of PO into senile mice (group IV) did neither effect significantly the elevation of spleen weights nor spleen indexes (Table I) nor the cell numbers (Table II).

The events leading to splenomegaly after the injection of *B. pertussis* cells are unknown. According to MORSE[2], it is not due to a production of new cells. This was concluded from the finding that the mitotic figures were no more prominent in the spleens of the pertussis-treated group than in those of the corresponding controls. On the other hand, it was demonstrated by histological investigations that proliferative changes occurred in the spleens of the treated mice, these being especially pronounced in the periphery of the follicles and in the red pulp[7]. This suggests that non-appearance of the characteristic splenomegaly in senile mice treated with PO is due to: 1. reduced 'accessible reserve of small lymphocytes'[2] or its diminished capacity to be mobilized and/or 2. reduced proliferative potential of the lymphoreticular tissue.

Zusammenfassung. Die Injektion abgetöteter Zellen von *Bordetella pertussis* bewirkt bei 3 Monate alten NMRI-Mäusen eine erhebliche Zunahme des Milzgewichtes verbunden mit einem Anstieg des Gesamtzellgehaltes der Milz. Diese charakteristischen Effekte sind bei Mäusen im Alter von 20 Monaten nicht zu beobachten. Ihr Ausbleiben im Alter ist vermutlich zurückzuführen: 1. Auf eine geringere Reserve an kleinen Lymphozyten bzw. deren verminderte Mobilisierbarkeit und/oder 2. auf eine Verminderung der Proliferationsfähigkeit des lymphoretikulären Gewebes.

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Ropalocytosis - a New Abnormality of Erythrocytes and their Precursors

During the course of our studies on a case of 'Hairy Cell' leukaemia, we have observed a startling and hitherto undescribed abnormality in the form of the erythrocytes and their precursors. 'Hairy Cell' leukaemia is a rare disease characterized by the presence of numerous cell processes arising from the surface of the leukaemic cells[1-4]. The nature of the leukaemic cell is uncertain, but it is probably related to the reticulum cell or lymphocyte.

A normochronic, normocytic anemia is commonly associated with this condition but no other abnormality of erythrocyte form has been reported to occur, nor did we find any such change with the light microscope in blood films and bone marrow smears from our case, apart from an occasional burr cell. However, in ultrathin sections of bone marrow and peripheral blood cells examined with the electron microscope, a remarkable alteration of form was seen in some of the erythrocytes, reticulocytes, and normoblasts (Figures 1 and 2). The alterations in form are often quite complex so that no two cells look exactly alike, but a basic feature seems to be the production of numerous branched and nubranched cell processes which in ultrathin sections often appear club-shaped. Such processes arise either from small foci or more extensive areas of the cell surface. This may culminate in the production of most bizarre forms which bear little resemblance to the original shape of the red blood cell. This abnormality is quite different from any of the well known alterations of erythrocyte morphology such as acanthocytes[5], helmet cells or schistocytes[6], sickle cells or the discocyte to echinocyte transformation in the normal and pathological cell[7]. Further the above mentioned abnormalities are discernable with the light microscope, while the new alteration of morphology we are now reporting can be characterized only with the electron microscope.

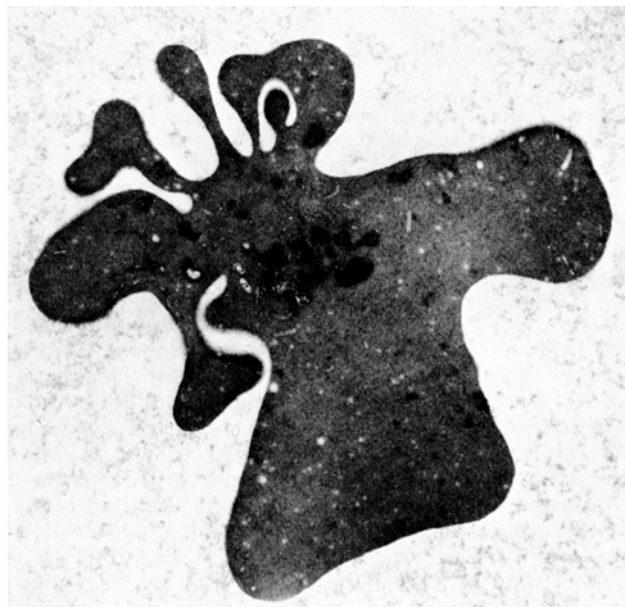

Fig. 1. Reticulocyte from peripheral blood showing alteration of overall shape and cell processes. ×10,000.

The defects in the normoblasts although at times quite considerable were generally of lesser degree and only a few of these cells were affected. However, severely altered reticulocytes were frequently seen. The most flamboyant morphological alterations, however, were exhibited by rare erythrocytes (Figure 2) in the peripheral blood, the rest of the red cells being completely free of such defects. The paucity of abnormal erythrocytes in the peripheral blood would suggest their rapid destruction.

The underlying cause of this process is not clear. Marked alterations in form of red cells are most commonly observed in situations where normal red cells are altered by an abnormal plasma environment, e.g. acanthocytes and echinocytes[8], or where mechanical factors fragment and disrupt the cells as in schistocyte formation[6,9]. The abnormal form in the present case is, however, totally different from these and also differs from the small tubular protrusions, described by BESSIS and BRICKA[10], in cells undergoing agglutination.

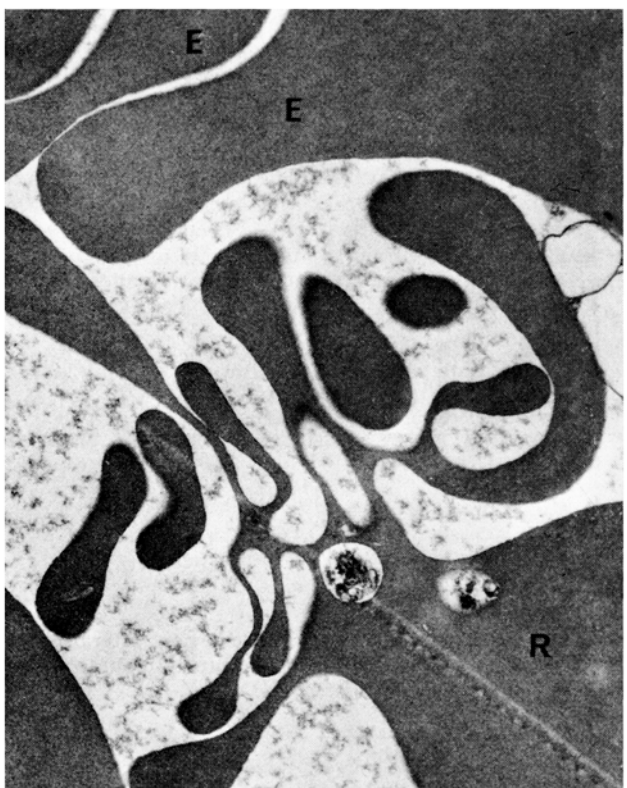

Fig. 2. Some morphologically normal erythrocytes (E) and a ropalocyte (R) with branched and unbranched club-shaped processes found in the peripheral blood. ×17,500.

The defect may lie in the cell membrane but whether this is an entirely intrinsic abnormality of the affected cells or whether extrinsic factors are involved, cannot be ascertained at the present. In keeping with the custom of naming erythrocyte abnormalities we would like to propose a name for this picturesque alteration of erythrocyte morphology. Since the basic defect here seems to be a production of processes which in ultrathin sections appear club-shaped and the greek for club is ropalon, we propose that this abnormality is called ropalocytosis.

Detailed study of the leukaemic and erythropoietic cells from this case are in progress and will be reported later. The purpose of this communication is to draw attention to this intriguing alteration of erythrocyte form, so that it may be further investigated in future cases of this rare condition.

Zusammenfassung. Bei leukämischer Retikulose wurden in Ultradünnschnitten im Elektronenmikroskop abnorm deformierte Erythrozyten, Reticulozyten und Normoblasten mit Einbuchtungen und Pseudopodien-artigen Ausstülpungen, die in gewöhnlichen Ausstrichpräparaten nicht auftreten, nachgewiesen.

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Effects of Some Membrane-Active and Other Compounds on Thrombin-Induced Platelet Aggregation

Thrombin-induced platelet aggregation appears to play a central role in both thrombosis and hemostasis[1]. Numerous agents have been reported to inhibit the aggregation of platelets induced by thrombin, but the mechanisms of action of most such agents are unclear. Recently, increasing evidence has accumulated to indicate that thrombin-induced platelet aggregation is mediated through changes in the platelet plasma membrane, possibly through changes in platelet adenosine triphosphatase (ATPase)[2] or acetylcholinesterase (AChE)[3] activities or by alteration of cyclic adenosine monophosphate (cAMP) levels[4,5]. Accordingly, a group of compounds were tested to determine their effects on thrombin-induced platelet aggregation. This group included steroids, antibiotics, adrenergic blocking agents, antiacetylcholine compounds, and others, many reported to influence plasma membrane phenomena[6,7]. Also tested for comparison were cAMP and dibutyryl cAMP.

All studies were conducted using the macroscopic platelet aggregation test of BRINKHOUS et al.[8], which is sensitive to small changes in the degree of aggregation of platelets. The test system contained thrice washed human platelets[8] mixed with equal parts of 1.08 m*M* $CaCl_2$, bovine thrombin 0.5 units per ml, 50 m*M* *tris* (hydroxymethyl) aminomethane buffer pH 7.4 (*Tris* buffer), and test agent in *Tris* buffer. Test agents were incubated at 25°C for 15 min with platelets prior to addition of calcium and thrombin. Aggregation was