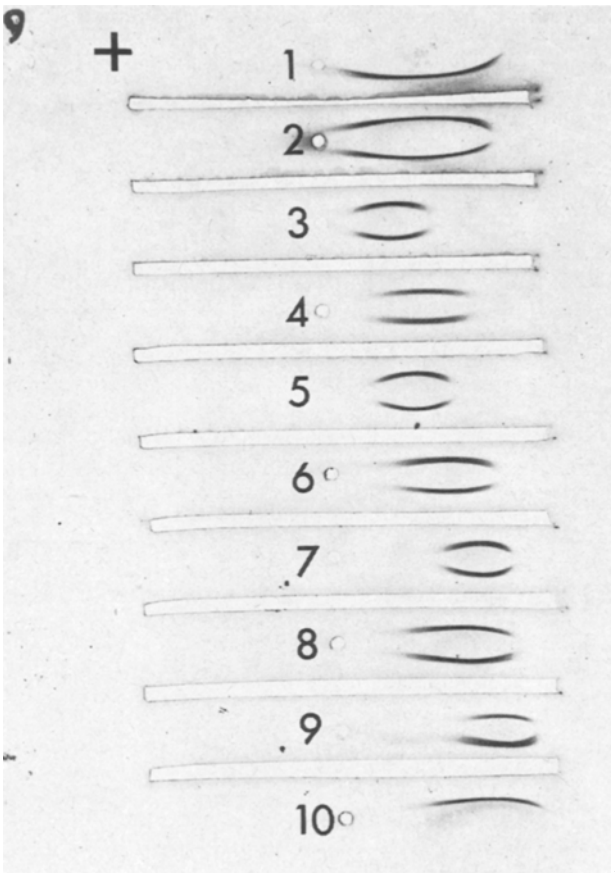

Fig. 2. Comparison of the electrophoretical mobilities of some hypogammaglobulinaemic sera and some isoelectric focusing fractions of IgG. The mobilities for each serum and IgG fraction, in this particular study, are given in bracketts. (1) Normal IgG; (2) serum h 5 (54%); (3) IgG fraction, ph 6.8 (40.9%); (4) serum h 15 (52%); (5) IgG fraction pH 7.0 (43.5%); (6) serum h 4 (63%); (7) IgG fraction pH 8.45 (76.1%); (8) serum h 10 (70%); (9) IgG fraction pH 8.95 (84.8%); (10) serum h 6 (74%). Anti Fc (IgG) serum was used to fill the troughs.

but had less IgG2 than expected. Additional studies proved that residual IgG had 'spontaneously' split into Fab and Fc-like pieces, and the recorded mobility corresponded to the Fc fragment.

Discussion and conclusions. Our results seem to prove that in hypogammaglobulinaemia, the abnormal electrophoretic mobilities of the residual IgG (previously reported by GOLEBIOWSKA and ROWE[6]) depend in the majority of cases on the distribution of IgG sub-classes.

Large amounts of IgG2, and possibly IgG4, due to the fast electrophoretic mobilities of these sub-classes[1,2,9] will lead to an overall fast mobility of the residual IgG. In the absence of IgG2, the mobility will reflect that of IgG1 and IgG3, both slow moving sub-classes[1,9].

Table II. Correlation between electrophoretical mobility and IgG sub-class distribution in 11 hypogammaglobulinaemic sera

	Titres Serum	Mobility	IgG1	IgG2	IgG3
Fast	h 7	52%	1/16	1/2	0
	h 5	54%	1/16	1/16	0
	h 9	54%	1/8	1/16	0
	h 15	57%	1/8	1/16	0
Medium	h 13	60%	1/16	1/8	1/4
	h 14	62%	1/32	1/8	0
	h 16	65%	1/32	1/16	1/4
	h 4	66%	1/6	1/4	1/2
	h 8	67%	1/8	1/4	1/2
Slow	h 10	70%	1/16	1/1	1/4
	h 6	75%	1/16	0	1/1
Normal IgG (Start. Dil. 1 mg/ml)			1/16	1/8	1/1

Summary. The correlation between the electrophoretic mobility of residual IgG in hypogammaglobulinaemic sera and the distribution of IgG sub-classes was studied. It was found that IgG from sera with raised levels of IgG2 show fast electrophoretic mobility, and sera with very low or absent IgG2 show a slow moving IgG. IgG3 was absent or just detectable in fast moving IgGs, and present in higher titers in some slow IgGs.

Résumé. La correlation entre la mobilité electrophorétique de l'IgG résiduelle des sérums hypogammaglobulinémiques et la distribution des sous-classes de l'IgG est l'objet de cet étude. Les IgG résiduelles de mobilité rapide ont des titres élevés d'IgG2 et celles de mobilité lente ne présentent que des traces d'IgG2.

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Birmingham 15 (England), 9 February 1970.

[9] W. D. TERRY and J. L. FAHEY, Science *146*, 400 (1964).

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Toxicity of Rabbit Anti-Dog Lymphocyte Serum to Human Cell Cultures

In previous work[1], rabbit anti-dog lymphocyte serum plus rabbit complement was found to have a rapid cytocidal effect on blood lymphocytes of patients with chronic lymphocytic leukemia but had little or no effect on normal human lymphocytes. In this study, the antiserum was tested against cells in long term cultures which had been derived from blood of normal persons and of patients with acute leukemia and from BURKITT's lymphoma. The method of cell culture is described by MOORE et al.[2].

The cells suspended in RPMI 1640 were incubated for 90 min at 37 °C with fresh rabbit serum (10%) and with a rabbit anti-dog lymphocyte serum in varying dilutions. The number of viable cells was counted by means of a

special slide chamber and phase microscopy. The titer was defined as the dilution of antiserum which killed 90% of the cells.

The rabbit anti-dog lymphocyte serum was toxic to the cells in all 5 cultures (Table). The cells of MOORE No. 5287 were the most sensitive (titer 1:3000) and of Burkitt's lymphoma, HRIK, the least sensitive (titer 1:30). Cultures CEE, MOORE and CORMACK were derived from normal persons and culture BARNES from a patient with acute leukemia. The degree of toxicity did not seem to be correlated with the derivation of the culture.

In recent parallel experiments, the rabbit anti-dog lymphocyte serum had titers up to 1:40 for lymphocytes of 6 normal persons (median > 1:10) and up to 1:1000 for lymphocytes from 12 patients with chronic lymphocytic leukemia (median 1:100). These findings are in accord with the previous study[1].

The rabbit anti-dog lymphocyte serum plus rabbit complement was, on the average, more toxic to the human cells in cultures than to blood lymphocytes of patients with chronic lymphocytic leukemia[3].

Titer of rabbit anti-dog lymphocyte serum which killed 90% of cells in established human cell cultures and in lymphocyte suspensions

Cell cultures	*Titer (median)*
Derived from blood of normal persons	
CEE No. 1120	1:300
CORMACK No. 8068	1:1000
MOORE No. 5287	1:3000
Derived from patients with acute leukemia or lymphoma	
BARNES No. 4277	1:1000
BURKITT's lymphoma – HRIK	1:30
Lymphocyte suspensions	
Lymph nodes of 2 dogs	1:10,000
Derived from blood of	
6 normal persons	> 1:10
12 patients with chronic lymphocytic leukemia	1:100

Zusammenfassung. Die Kombination von Kaninchen Anti-Hund Lymphozytenserum und Kaninchen Komplement war für menschliche Blutzellen in in-vitro-Kultur sowie für Blutlymphozyten von Patienten mit chronischer lymphatischer Leukämie toxisch.

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R. SCHREK, F. W. PRESTON and A. A. DIETZ, Blood *33*, 555 (1969).

[2] G. E. MOORE, R. E. GERNER and H. A. FRANKLIN, J. Am. med. Ass. *199*, 519 (1967).

[3] This study was aided by grant No. CA-07541 from the National Cancer Institute, United States Public Health Service, and by grants from the Leukemia Research Foundation, Inc., and the John A. Hartford Foundation, Inc.

Rheogoniometric Viscosity Measurements of Whole Human Blood at Minimal Shear Rates Down to 0.0009 sec^{-1}

Low shear rates down to 0.052 sec^{-1} applied to certain blood systems led some investigators to the conclusion that blood has Newtonian characteristics. Our modifications to the Weissenberg rheogoniometer permitted measurements down to 0.0009 sec^{-1} and, in some cases, down to 0.0006 sec^{-1}. The data obtained at these low shear rates demonstrate that blood has non-Newtonian behavior and exhibits a yield stress. These findings are considered significant in the physiology and pathology of blood circulation.

Blood viscosity is markedly augmented as the flow of blood approaches a standstill and the shear rate progresses to zero. Such situations exist physiologically in the living circulation, when the flow velocity is extremely low, such as in post-capillary vessels and with the occurrence of stasis or cessation of blood flow. Therefore, it needs to be established whether or not blood has a yield stress and to determine what the flow characteristics are at extremely low shear rates.

One of us (A.L.C.) proposed in 1942 that blood behaves as a non-Newtonian fluid and might well have a yield stress[1]. Since at that time techniques were not available for the measurement of viscosity at very low shear rates, this problem could only be speculated upon. MERRILL, WELLS et al.[2] studied whole blood viscosity at 'low' shear rates and concluded, according to CHIEN, GREGERSEN et al.[3], that whole blood exhibited a yield stress. CHIEN et al.[3], who repeated some of these studies, reported in 1966 that the results at low shear rates with the GDM viscometer were not reliable, because of limitations of

[1] A. L. COPLEY, L. C. KRCHMA and M. E. WHITNEY, J. gen. Physiol. *26*, 49 (1942).

[2] E. W. MERRILL, E. R. GILLILAND, G. COKELET, A. SHIN, A. BRITTEN and R. E. WELLS JR., J. appl. Physiol. *18*, 255 (1963).

[3] S. CHIEN, S. USAMI, H. M. TAYLOR, J. L. LUNDBERG and M. I. GREGERSEN, J. appl. Physiol. *21*, 81 (1966).