of the route of hormone administration. 2. The prevention of growth inhibition by liver powder is not accompanied by other evidences of diminished cortisone activity, *i.e.*, despite normal growth such animals exhibit physiological, anatomical and biochemical changes of the same degree as do cortisone treated rats without liver supplement.

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In vitro Effect of Autologous Lipemic Plasma on Platelet Suspensions.* (20973)

CHARLES W. LOUGHRY AND JACK W. COLE.

From Surgical Research Laboratories, Western Reserve University School of Medicine and Department of Surgery, University Hospitals of Cleveland.

It has recently been observed that hyperlipemia, produced by orally or intravenously administered fat emulsion, results in a marked decrease in the number of circulating blood platelets(1). As yet, there is no satisfactory explanation for this phenomenon. It has been postulated that this may be due to intravascular agglutination of platelets. This possibility suggested itself when it was noted that the intravenous injection of heparin during the hyperlipemia resulted in a prompt return of the platelet count toward normal values. This finding offers substantial evidence that platelets are not destroyed during intervals of hyperlipemia but rather supports the concept that they are "clumped" and that heparin promotes redispersion.

Swank's(2) observation that hyperlipemia produced agglutination of red blood cells led us to investigate the possibility that a similar process might prevail with platelets. The following experiments were carried out to determine whether or not platelet suspensions would demonstrate agglutination when incubated *in vitro* with autologous lipemic plasma.

Methods. All glassware used in these experiments was siliconated. A platelet suspension was prepared from 150 cc of whole blood obtained from normal healthy adult males according to the method of Stefanini(3) with minor modification. The donors were then given 250 cc of intravenous fat emulsion† over a 2-hour period. Platelet counts (Rees-Ecker) were obtained on the donor's blood prior to the administration of the Lipomul and at varying intervals during its administration. When a significant fall (40-50%) in the individual's platelet count was noted, a 10 cc blood sample was obtained. The blood was centrifuged and the supernatant plasma removed.

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† Lipomul—supplied by Dr. Robert Heinle of the Upjohn Co.

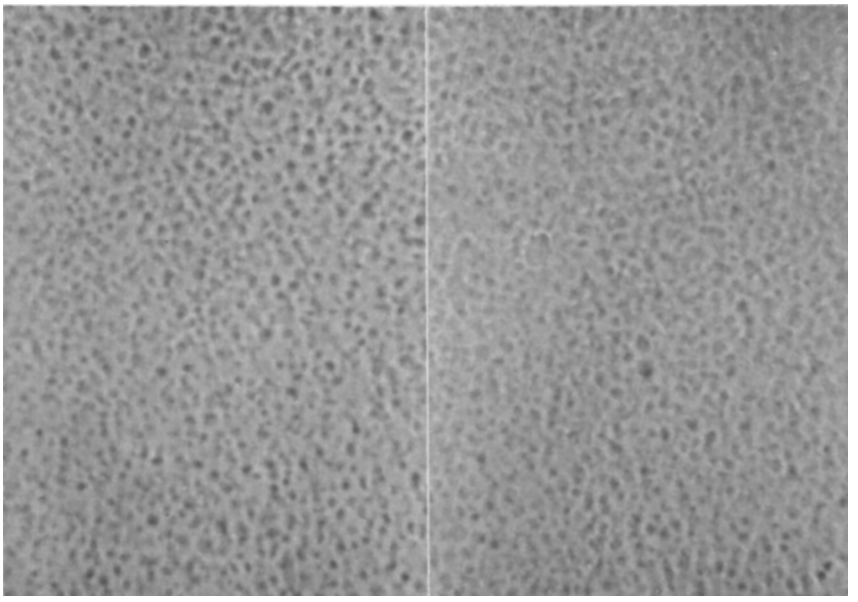

FIG. 1. Microscopic appearance, after 16 hr of incubation, of platelet suspensions mixed with autologous lipemic plasma (left) and physiological saline (right). (× 322)

One drop of the donor's previously prepared platelet suspension and one drop of the donor's hyperlipemic plasma were then mixed on a glass slide with a cover slip and sealed with paraffin. A similar preparation was made mixing saline and the platelet suspension for a control. Several such preparations were made for study. The slides were then incubated at 37°C for 16 hours and examined microscopically. In addition, siliconated stoppered test tubes containing 1 cc of the platelet suspensions and 1 cc hyperlipemic plasma and/or saline were incubated and observed microscopically after a 16-hour incubation period at 37°C.

Results. After the administration of 250 cc of intravenous Lipomul to one normal adult the platelet count fell from 460000/cu mm to 265000/cu mm, thus establishing that in this individual the hyperlipemia had produced a significant decrease in the circulating blood platelets. Similar results were noted in 4 additional subjects. However, when the plasma which was obtained at the time of the maximum platelet depression was incubated with a platelet suspension previously prepared from the same individual no agglutination was noted during a 16-hour period of observation, in either the slide or test tube preparations. The platelets were evenly dispersed and no significant morphological alterations were apparent in either the experimental or control mixtures. Representative fields are shown in Fig. 1. Identical results were obtained in 10 such experiments.

Summary. 1. *In vitro* mixing and incubation of blood platelets and autologous lipemic plasma does not result in apparent platelet agglutination.

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