synergist may lead to a modification of views pertaining to the nature of the gonadotropic hormones.

7685 C

Hemolysins to which Reticulocytes are Especially Vulnerable in the Plasma of Primary Anemia.

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Ucko[1] has described a saponin-like substance present in normal plasma but in much larger amounts in active cases of primary anemia. It is more toxic to the reticulocytes than to the mature cells. It seemed to us that the presence of such a substance, rather than a structural defect in the reticulocytes of patients with primary anemia, might explain the fall of 30-60% in the reticulocyte count on incubating sterile defibrinated blood of active cases of primary anemia.[2] Blood from normal persons, from secondary anemia cases, or from cases of primary anemia during remission does not have a fall in reticulocyte count when incubated in the same way.[2, 3]

We therefore took blood from 4 active cases of primary anemia just before starting intensive parenteral liver therapy and preserved the plasma on ice for 4-7 days. Washed cells obtained at the height of the reticulocyte rise were then mixed with twice their volume of fresh plasma, and also, in the same proportion, with plasma obtained before treatment. These tubes were incubated at 37° C. All the blood was handled with sterile technique and counts were made only if there was no bacterial growth on incubation.

In one case, the mildest and least jaundiced (Van den Bergh, indirect, 1.25 units), the blood taken during the active stage had to be transported and cells and plasma were not separated until several hours after being drawn. This patient had 2.4 million red cells per cu. mm. before treatment and in this case the reticulocyte count in the tubes containing pre- and post-treatment plasma was the same after 24 hours incubation. The other 3 patients all had counts under 1.4 million and indirect Van den Berghs of 1.5 to 3 units before

[1] Ucko, H., *Z. f. klin. Med.*, 1931, **118**, 22.

[2] Buckman, T. E., and MacNaugher, E., *J. Med. Research*, 1923, **44**, 61.

[3] Mermod, C., and Dock, W., *Arch. Int. Med.*, in press.

treatment. At the time the reticulocyte peak was reached the Van den Berghs had fallen to 0.6 to 0.8 units. In all of these the reticulocyte count fell on incubating the cells in plasma taken before treatment, but not in that taken during the period of high reticulocytosis. In the first 2 cases the proportion of cells to plasma was as 1 to 2 during incubation; in the third case a 1 to 10 suspension was also observed and showed a more striking fall in reticulocyte count in the plasma taken during the acute stage (Table 1).

TABLE I.

The reticulocyte percentage after incubation for 24 hrs. at 37° C. of cells obtained during remission in plasma obtained during remission (Plasma B) and that obtained before treatment (Plasma A). In case P, Plasma A obtained on March 8, plasma B on March 12; in case S, A on April 2, B on April 9; in case M, A on September 11, B on September 16. Counts in plasma B were never lower than in fresh blood unless incubated 30-50 hours.

Reticulocyte Counts After Incubation.

	Case P	Case S	Case M	Case M.*
Plasma A	24	25	19	6.5
Plasma B	29	34	26	28

*Ratio of washed cells to plasma in these tubes 1:10, in all others 1:2.

This phenomenon can not be explained by increased reticulocyte resistance due to treatment, but suggests the presence of a hemolysin, to which reticulocytes especially are vulnerable, in the cases of active primary anemia, and the disappearance of this substance during remission.

7686 C

Absence of Vasoconstrictor Substance in Blood of Rats with Renal Hypertension.

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Using an adaptation of Griffith's and Collins' method for measuring systolic pressure[1] in rats which had developed hypertension as a result of renal insufficiency,[2] we have studied the effect of destroying the central nervous system on the blood pressure of normal and of hypertensive rats. The blood pressure was determined under light ether anesthesia, and then, after inserting a tracheal cannula con-

[1] Griffith, J. Q., Jr., and Collins, L. H., Jr., *Am. Heart J.*, 1933, **8**, 671.

[2] Chanutin, A., and Ferris, E. B., Jr., *Arch. Int. Med.*, 1932, **49**, 767.