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Platelet deficiency a factor in diminished coagulability of the blood in anaphylaxis.

By E. W. SCHULTZ.

[From the Department of Bacteriology and Experimental Pathology, Stanford University, California.]

Delay or loss of coagulability of the blood in anaphylaxis has long been recognized. It is generally attributed to excess of antithrombin or to diminished thromboplastin. While making a study of the blood fibrin in canine anaphylaxis by the method of Foster and Whipple¹ observations were made which we believe throw further light on the phenomenon. In making the blood fibrin studies, approximately 9 cc. of blood, obtained by cardiac puncture, are discharged into a centrifuge tube containing 1 cc. of a 1.5 per cent sodium oxalate solution. The samples are then centrifuged at moderate speed for thirty minutes. Exactly 2 cc. of the oxalated plasma is discharged into 40 cc. of clotting solution. This consists of 0.125 per cent calcium chloride in physiologic salt solution. Normal oxalated plasma usually forms a jelly-like coagulum within one hour, whereas the plasma in fatal anaphylaxis may remain uncoagulated for days. Occasionally a feeble coagulum forms after several days. During the course of the work several thromboplastic agents were employed, but it was found that a small quantity of platelets obtained from normal plasma gave us the most trustworthy results. They are obtained as follows: Several hundred cubic centimeters of normal oxalated blood obtained by cardiac puncture from a fasting dog is centrifuged at a slow speed until the red cells have been well sedimented and a more or less turbid plasma remains. The plasma is then pipetted off into 15 cc. centrifuge tubes, and centrifuged at high speed (2700 r.p.m.) for about one hour. At the end of this time several millimeters of a pearly white sediment will be found in the tips of the tubes. This sediment on microscopic examination proves to be composed chiefly of platelets. The tubes are carefully rinsed to remove adherent plasma, and the sediment is dispersed in a little saline by vigorous shaking.

¹ Foster, D. P., and Whipple, G. H., *Am. J. Physiol.*, 1922, lvii, 365.

An opalescent fluid results, which on being added to the clotting solution containing the anaphylactic plasma usually brings on coagulation within a few minutes, while the controls often fail to show coagulation for days.

It has also been possible to induce coagulation with the sediment obtained from the anaphylactic plasma of the same animal, but here it is necessary to pool the sediment from several tubes to get anything like the prompt coagulation which one may obtain from a single tube of normal plasma. The sediment obtained from anaphylactic plasma is appreciably less than that obtained from normal plasma. This would seem to confirm the opinion that the diminished coagulability is due primarily to a reduction in the number of platelets, which normally supply the necessary thromboplastin. These observations are in accord with those of Achard and Aynaud² and Pesci,³ who noted a reduction in the number of platelets in anaphylaxis. The reduction is considered to be due to clumping of the platelets as a result of physico-chemical disturbances. This explanation is supported by the observations of v. Behring,⁴ who found clumped platelets in the capillaries and smaller arterioles of the pia and the choroid plexus in guinea pig anaphylaxis. Studies on the actual platelet count in canine anaphylaxis and on the fate of the platelets will be undertaken shortly.

² Achard, Ch., and Aynaud, M., *Compt. rend. Soc. de biol.*, 1909, lxxvii, 83.

³ Pesci, E., *J. de physiol. et path. gen.*, 1921, xix, 242.

⁴ v. Behring, E., *Deutsch. med. Wchnschr.*, 1914, xl, 233.