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Effect of Human Blood Serum on Bile Salt Hemolysis.*

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In previous reports^{1, 2} the effect of blood serum on the toxicity of bile salts injected intraperitoneally into the white mouse and the hemolytic effect of these salts dissolved in normal saline for red blood cells were observed. It was found that hemolysis occurred in dilutions of 1:200 to 1:6400. This large variation in hemolytic range might have been due in part to exposure of the cells under diverse circumstances of collection and in part to the heterogeneous type of individual from whom they were collected.

The present report concerns the effects of blood serum on hemolysis by bile salts after the stock product had been purified as explained in a previous article.¹ Different concentrations of this purified salt were made up in normal saline and in fresh human serum (not older than 3 hours). A drop of fresh homologous blood was then added to the saline and serum dilutions and the results observed in 3 and 24 hours. The hydrogen ion concentrations of the solutions were computed but the variation was not sufficient to influence the results. The surface tension of the several solutions of like concentration of the salt was taken by the drop method and this variation was found in each instance to be so slight that at least by the present perfection of technique estimation of the small difference was impossible. The tubes in which the solutions were mixed were allowed to stand at room temperature. Approximately 24 bloods were tested.

When human serum was used as the vehicle and readings made in 3 hours, complete hemolysis occurred in the tubes containing 2.5% bile salts, partial hemolysis in tubes containing 1.25%, and no hemolysis in those containing 0.625%. When normal saline was used as a vehicle complete hemolysis occurred in all tubes containing 0.625% bile salts, in $\frac{1}{2}$ of the tubes containing 0.3125% and $\frac{1}{3}$ of the tubes containing 0.10625%, partial hemolysis in $\frac{1}{2}$ of the tubes containing 0.3125%, and $\frac{1}{3}$ of the tubes containing 0.10625% and no hemolysis in $\frac{1}{3}$ of the tubes

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¹ Williams, J. W., *PROC. SOC. EXP. BIOL. AND MED.*, 1932, **29**, —.

² Williams, J. W., *PROC. SOC. EXP. BIOL. AND MED.*, 1930, **27**, 913.

containing 0.10625%, and all tubes containing 0.053125%. After standing 24 hours there was a shift of hemolysis, in some instances several dilutions higher when normal saline was used as a vehicle. On the other hand, with the blood serum vehicle no such marked shift was noted.

The foregoing results indicate that both white mice and red cells are protected by blood serum to a considerable degree from the toxic effects of bile salts. This substantiates our belief that either the serum has a neutralizing effect or that the serum molecules by affording a surface capable of being coated by bile salts diverts in part or toto this substance from the red cell and thus prevents or delays hemolysis.

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Peripheral Course of Sensory Nerves Supplying Arteries of Lower Extremity.

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Lumbar sympathetic gangliectomy abolishes the reflex vascular spasm, the central feature of Raynaud's disease. The immediate relief of the accompanying pain is so striking that it has led clinical observers to conclude that some type of sensory nerve to the lower extremity is sectioned in the course of the sympathectomy. This view is strengthened by the experiments of Johnson¹ and of Kuntz and Farnsworth,² which demonstrated that certain of the dorsal root components of the lower dorsal and upper lumbar spinal nerves pass to the lumbosacral sympathetic trunks and are distributed from them to the lumbosacral plexus.

During the course of experiments in arterial visualization, we noted that cats under sodium amytal anesthesia reacted in a characteristic manner when a concentrated solution of sodium iodide was injected into the femoral artery. The entire body stiffened with the legs straining at the leashes; hyperpnea; dilatation of the pupils; tossing of the head accompanied by vocalization, the outcry possess-

¹ Johnson, S. E., *J. Comp. Neurol.*, 1921, **33**, 85.

² Kuntz, A., and Farnsworth, D. I., *Proc. Soc. Exp. Biol. and Med.*, 1928, **25**, 808.