the fibrin there is a well defined rise in the hemotocrit values, frequently from 10 to 20 per cent above the normal. The maximum hematocrit readings are generally attained by the tenth minute (average on thirty dogs in fatal shock), immediately following which the fibrin generally reaches its lowest level. This would suggest that the drop in fibrin is probably due to an escape of plasma proteins incident to the increased permeability of the capillary endothelium recognized in anaphylaxis, although the possibility of its partial destruction cannot be excluded.

The following table illustrates the points in question. The fibrin is expressed in milligrams per 100 cc. of blood; the hematocrit (cells) in per cent.

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The blood platelets in canine anaphylaxis.

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It is well recognized that in canine anaphylaxis the blood generally becomes incoagulable, often remaining fluid for days. This loss of coagulability of the blood has been attributed to an excess of antithrobin or to a diminution of thromboplastin. Recently one of us noted,[1] in connection with studies on the blood fibrin in canine anaphylaxis, that the addition of a small quantity of platelets to the clotting solution, into which the oxalated anaphylactic plasma had been discharged, induced prompt coagulation in samples which otherwise (in controls) remained uncoagulated for hours, sometimes for days. This suggested the desirability of making platelet counts. While a diminution in the platelets in anaphylaxis has been reported,[2, 3] we were especially interested in the counts obtained in fatal canine anaphylaxis, particularly since changes in the coagulability of the blood are more prominent in the dog than in other animals.

[1] Schultz, E. W., PROC. SOC. EXP. BIOL. AND MED., 1925, xxii, 343.

[2] Archard, Ch., and Ayanud, M., *Compt. rend. Soc. de biol.*, 1909, lxvii, 83.

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

Of a number of current methods employed in counting platelets we found none that gave us entirely satisfactory results. We finally adopted the method of Rees and Ecker,[4] using, however, instead of their diluting fluid that recommended by Leake and Guy.[5] This combination gave us entirely satisfactory checks. The time allowed for settling in the counting chamber was reduced to a constant minimum to prevent undue evaporation of the diluting fluid and to insure strictly relative counts. The blood samples were obtained from the common carotid artery by incision. A rubber covered artery clamp was placed proximal to the opening and the blood was allowed to flow freely before the samples were taken. The animals were sensitized to horse serum,

Group I—Deep Shock Terminating Fatally.

Dog No.	Normal Platelet Count	Platelet Counts During Shock.						Time of death; minutes
1	576,000	minutes	1.5	4.5				12
		count	475,000	310,000				
2	398,000	minutes	3.0	6.0				9
		count	280,000	210,600				
3	380,000	minutes	1.0	6.5	12.0	18.0	25.0	26
		count	290,000	158,700	141,600	99,860	98,720	
4	398,800	minutes	7.0	15.0	20.0			28
		count	227,000	161,000	132,600			
5	528,000	minutes	3.5	8.5	17.5	26.5		30
		count	440,200	296,000	208,600	150,100		
6	530,100	minutes	4.5	9.0	15.0	30.0	45.0	65
		count	398,600	281,000	236,000	152,800	160,200	
7	483,000	minutes	3.0	9.0	18.0	23.0		29
		count	401,000	304,000	272,600	170,800		
8	492,000	minutes	5.0	13.0	19.0	29.0		30
		count	401,600	298,400	203,000	210,800		

Group II—Light Shock not Terminating Fatally.

1	852,800	minutes	2.0	11.0	16.0	31.0	45.0
		count	824,000	804,900	762,400	710,000	821,600
2	492,100	minutes	6.5	15.5	33.0	75.0	
		count	389,600	398,400	352,000	522,000	
3	469,000	minutes	7.0	17.0	27.0	42.0	52.0
		count	349,100	297,200	296,000	312,400	322,600

[4] Rees, H. M., and Ecker, E. E., *J. Am. Med. Assn.*, 1923, lxxx, 621.

[5] Leake, C. D., and Guy, E. F., *J. Am. Med. Assn.*, 1925, lxxxiv, 890.

0.3 cc. per kilo subcutaneously, followed on the third day with the same dose intracardially, and the shocking dose, 1 to 2 cc. per kilo, was injected intracardially.

Our results show a decrease in the platelets ranging from 47 to 71 per cent below the normal count, depending on the time at which the samples were taken. The drop is as a rule progressive, becoming more marked as the state of shock continues. In arriving at these figures the shrinkage of the blood volume and consequent relative increase of the cells during shock, was not taken into consideration. Correction for this would show a further reduction in the platelet counts. In the case of non-fatal shock the decline in platelets is not so marked. Two non-sensitized dogs injected with the equivalent amount of horse serum showed no material difference in the platelet count.

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Elimination of streptococci in blood stream through the biliary system in the dog.

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Streptococci injected into the blood stream of the dog, even in massive doses, are removed from the circulating blood with surprising rapidity. Blood cultures made at short intervals show a precipitous decline from more than ten thousand colonies per cubic centimeter to zero within four to six hours. Examination of the tissues within two or three hours show sparsely scattered cocci in the liver, spleen and lungs. In later examinations the organisms are generally difficult to find. Although the leucocyte count rises appreciably following the injections, sometimes fifty per cent or more within five hours, it is difficult to find cocci in the leucocytes in smears prepared at short intervals during the experiment.

While it cannot be denied that many of the organisms are filtered out in the capillary and sinusoidal beds and picked up by phagocytic cells, especially in the spleen, liver and bone marrow,