penic purpura and anaphylactic, tryptic or peptonic shocks.

The results presented in this paper show another possible means for producing anaphylactoid shock, *i.e.*, by injecting intravenously a large amount of an active anti-platelet serum. The similarity of various signs described in purpura with some verified in allergic or anaphylactic phenomena is emphasized.

Summary. Intravenous injection of large amounts of active anti-platelet serum produces in dogs, rabbits, guinea pigs and rats a picture of severe shock quite similar to anaphylactic shock. Relationship between purpura and anaphylactic phenomena is emphasized.

16876

Thermolability of the Bacterium-Phage Complex.

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The infection of susceptible host cells by bacterial viruses may lead to several interesting events affecting primarily the host. A number of investigators have reported an interfering effect on the growth of unrelated viruses[1-3] while others have shown that infected cells no longer divide[2,4] or were incapable of adaptative enzyme production.[5] In the course of our investigations on the effect of sodium chloride on phage formation by staphylococci at elevated temperatures,[6] it was observed that the bacterium-phage complex was much more thermolabile than either the bacterium or phage alone. The present paper is a brief account of these observations.

The K race of phage active upon *Staphylococcus aureus* (K strain) was employed throughout these experiments. Quantitative determinations of [phage] were performed by Gratia's[7] method and viable cell counts were made by plating appropriate dilutions in tryptose agar and counting the colonies developing after 24 hours incubation at 36°C. Stock bacterial cultures were prepared by harvesting the growth from tryptose agar cultures in Roux flasks after incubation at 36°C for 18-24 hours. The suspensions were chilled for one hour in an ice-water bath and were used to make mixtures in tryptose phosphate broth containing 1 x 10^8 organisms/ml. To aliquots of these preparations phage was added in concentrations varying from 5 $\times$ 10^7 to 2 $\times$ 10^8 plaque units/ml; aliquots without added phage were maintained as controls. Samples were removed at once from the experimental mixtures and controls for determination of the viable cell counts.

As soon as samples had been taken, the tubes were immersed in the ice-water bath for an additional hour. At this time aliquots were removed for plaque determinations and viable cell counts. The latter were assumed to be a measure of the uninfected bacteria since infected cells cannot reproduce and form colonies. The plaque counts were considered to indicate the number of infected cells for it is known that phage uptake by bacteria is rapid even at 5°C and that little phage remains unattached to cells after sorption has proceeded for one hour.[8]

1 Delbrück, M., and Luria, S. E., *Arch. Biochem.*, 1942, **1**, 111.

2 Luria, S. E., and Delbrück, M., *Arch. Biochem.*, 1942, **1**, 207.

3 Delbrück, M., *J. Bact.*, 1945, **50**, 151.

4 Cohen, S. S., and Anderson, T. F., *J. Exp. Med.*, 1946, **84**, 511.

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7 Gratia, A., *Ann. Inst. Pasteur*, 1936, **57**, 652.

8 Krueger, A. P., Scribner, E. J., and Brown, B. B., *J. Gen. Physiol.*, 1946, **30**, 25.

With the maximum number of cells infected by phage, the mixtures were next placed in a constant temperature bath and were held at 47.5°C for 40 minutes. In order to ascertain the effect of exposure to heat upon infected and uninfected bacteria, the plaque counts and viable cell counts were repeated.

Table I presents the data from two experiments of the sort just described. The bacterial controls exhibit no significant reduction in number of viable cells during the 40 minute period of exposure to 47.5°C, nor is there any measurable drop in the phage control. On the other hand, plaque counts in the experimental mixtures are reduced to 17% and 14% of the initial titres. The numbers of viable, uninfected cells in the experimental mixtures remain practically constant confirming the data secured from the bacterial controls.

Three other experiments besides the 2 included in Table I were performed with essentially identical results. The average value for thermal destruction of phage adsorbed to bacteria for the set of 5 experiments is 80%.

Normally, phage-infected cells are detectable as plaques; exposure of such complexes to very moderate heat treatment somehow inactivates the phage so that plaques are no longer produced. Normal, uninfected bacteria or phage alone are not affected by the experimental conditions employed. We have considered the possibility that the results secured might be due to the aggregation of the infected cells during the period of heating but no evidence of this mechanism was observable on direct microscopic examination. Furthermore, the effect of phage upon living susceptible cells is to increase the zeta potential[9] and thus to decrease the likelihood of agglutination.

Summary. Staphylococcal phage and the susceptible strain of *Staphylococcus aureus* are not destroyed during a 40 minute period of exposure to 47.5°C. However, when the phage is attached to bacterial cells the plaque count drops to approximately 20 percent of the initial value under like conditions.

TABLE I.
Effect of Heat on Bacterium-Phage Complex.
Treatment 60 min. at 4°C.

Sample	Viable $[B]_0$		$[P]_0$		Treatment	Viable $[B]_f$		$[P]_f$	
	Exp. 1	Exp. 2	Exp. 1	Exp. 2		Exp. 1	Exp. 2	Exp. 1	Exp. 2
Bacterium, phage mixture	1.5×10^8	1.5×10^8	1.0×10^8	2.0×10^8	+ 40 min. at 47.5°C	7.8×10^7	4.5×10^7	6.7×10^7	1.0×10^8
						8.0×10^7	4.3×10^7	1.2×10^7	1.4×10^7
Bacteria control	1.5×10^8	1.5×10^8	—	—	+ 40 min. at 47.5°C	1.5×10^8	1.5×10^8	—	—
						1.4×10^8	1.4×10^8	—	—
Phage control	—	—	1.0×10^8	2.0×10^8	+ 40 min. at 47.5°C	—	—	1.1×10^8	2.0×10^8
						—	—	1.0×10^8	2.0×10^8

$[B]_0$ Initial number bacteria/ml.
$[B]_f$ Final number bacteria/ml.
$[P]_0$ Initial number plaque units/ml.
$[P]_f$ Final number plaque units/ml.

[9] Krueger, A. P., and Mundell, J. H., PROC. SOC. EXP. BIOL. AND MED., 1937, **36**, 317.