between the living condition observed in the field and the limiting conditions observed in the laboratory.

This is a preliminary report.

[1] Bastin, Edson S., Anderson, Belle, Green, Frank E., Merritt, C. T., and Moulton, Gail, *Bull. Am. Assn. of Petroleum Geologists*, 1926, x, 1270-1299,

[2] Beyerinck, W. M., *Centr. für Bakter.*, etc., 1895, Abt. II, i, pp. 1-9 and 104-114.

[3] van Delden, A., *Centr. für Bakter.*, etc., 1903-4, Abt. II, xi, pp. 81-94 and 113-119.

[4] Elion, H., *Centr. für Bakter.*, etc., Abt. II, lxiii, 58-67.

3576

The Effect of Heparin on Anaphylactic Shock in Guinea Pigs.

O. B. WILLIAMS AND F. RENE VAN DE CARR. (Introduced by K. F. Meyer.)

From the Division of Bacteriology, University of California Medical School.

Kyes and Strauser[1] have recently reported the inhibition of anaphylactic shock in pigeons by the intravenous injection of heparin 45 minutes preceding the shocking dose of antigen. They state that it is their belief that many of the symptoms of anaphylactic shock are the result of the formation of fibrin aggregates, and that if the formation of these aggregates be inhibited by a suitable reagent the symptoms will be reduced or eliminated.

In view of the striking results of these workers with pigeons, it seemed to us that it would be worth while to determine the effect of heparin on anaphylactic shock in guinea pigs. The animals were sensitized to horse serum twenty days before testing. Eight controls were tested for efficiency of sensitization, 6 of these dying with classical antemortem symptoms and showing the typical post mortem picture. Two animals exhibited symptoms of severe shock but survived. The guinea pigs receiving heparin were given 5 mg. of heparin in physiological salt solution intracardially for each 100 grams of body weight. The results obtained with these animals are shown in Table I.

As shown in the table, two of the heparinized guinea pigs receiving shocking doses of serum exhibited classical symptoms of shock, one of them succumbing with typical post mortem findings. It may be recalled that Kyes and Strauser report that 1 of 12 heparinized pigeons showed transitory symptoms. We do not feel that there is

any relation between the size of the shocking dose and the occurrence of severe symptoms in the heparinized guinea pigs, since some of the animals showed slight or no symptoms, after receiving the same amount of serum as that given to those which exhibited shock.

TABLE I.

Animal number	Time elapsing between injection of heparin and serum.	Amount of serum	Symptoms	Remarks
1	15 min.	2 cc.	None	
2	15 min.	2 cc.	None	Death 20 min. later from hemorrhage. No pulmonary emphysema. Pericardial sac filled with blood. Free blood in pleural cavity.
3	25 min.	2 cc.	Slight transitory respiratory distress	
4	30 min.	2 cc.	Typical symptoms of shock	Death in 5 min. with typical pulmonary emphysema at autopsy. Blood showed no clot in test tube after 48 hours.
5	40 min.	2 cc.	None	
6	55 min.	1.5 cc.	None	Death about 40 min. later from hemorrhage. Autopsy findings as for No. 2 above.
7	1 hour	1 cc.	Slight transitory respiratory distress	
8	1 hour	1 cc.	None	
9	1 hour and 15 min.	1 cc.	Slight transitory respiratory distress	
10	1 hour and 20 min.	2.25 cc.	Typical symptoms of shock	Recovered
11		None		Death from hemorrhage about 20 min. following heparin. Autopsy findings as for No. 2 and No. 6 above

A check on the clotting time of the blood of two of the control animals showed the formation of a clot at between 15 and 20 minutes, the blood having been collected in a test tube at autopsy. In the cases of the three heparinized animals which died from hemorrhage and of the one which died with symptoms of shock, no clot had been formed at the end of 48 hours by blood collected in a similar manner.

The animals for which we record slight transitory respiratory distress showed symptoms ranging from a single gasp to sneezing and scratching at the nose.

The results obtained appear to indicate that the injection of hep-

arin into the circulation in amounts sufficient to prevent the formation of a blood clot prevents or reduces, in the majority of cases, the symptoms of anaphylactic shock in guinea pigs hypersensitive to horse serum.

[1] Kyes, Preston and Strauser, E. R., *J. Immunol.*, 1926, xii, 419-422.

3577

Further Evidences of the Non-Sympathomimetic Action of Ephedrine.

FLOYD DE EDS AND EDWARD M. BUTT.

From the Department of Pharmacology, Stanford University School of Medicine, San Francisco.

In a previous report[1] it was shown that the most favorable functional state of the sympathetic nerves in blood vessels, *i. e.*, during sensitization by cocaine, failed to give responses to ephedrine, indicating that the cause of the pressor action of ephedrine was not sympathetic stimulation. Further evidence obtained recently along other lines confirms this result.

Three cats (4.5 to 7 cc. per kilo of Fledxt. Ergot, U. S. P.) and 1 dog (1 mgm. per Kg. ergotamine) were ergotized to the point of reversal of the original rises of blood pressure caused by epinephrine, to falls of pressure owing to paralysis of the constrictor sympathetic endings. In these same animals, the rises of blood pressure (before ergot) caused by different doses of ephedrine were invariably obtained when the vasomotor reversals to epinephrine were demonstrated. In other words, the pressor action of ephedrine still occurred after paralysis of the sympathetic constrictor endings, and

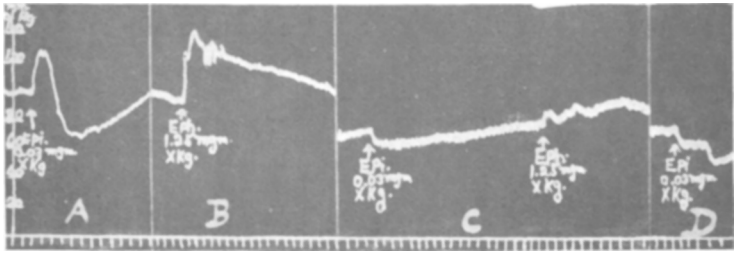

FIG. 1.
Control pressor actions of epinephrine and ephedrine (A and B), and reversal of epinephrine (C and D), but not with ephedrine (C) after ergot in a cat. "Epi" means epinephrine, and "Eph", ephedrine.