the injection of 1 cc. of immune rabbit serum was approximately the same.

A subcutaneous injection of 1 mg., but not 0.1 mg., of soluble specific substance 24 hours after sensitization desensitized the guinea pigs completely to a subsequent intracardial injection (24 hours later) of soluble specific substance, but not to an injection of cellular carbohydrate. One mg. of the latter, however, desensitized entirely to the soluble specific substance and partially (no fatal shocks) to the cellular carbohydrate.

When the soluble specific substance of Avery and Heidelberger was added to the minimum dose of antipneumococcus serum necessary to sensitize and the mixture injected immediately into guinea pigs, the animals were not sensitized to a subsequent intracardial injection of this substance but were to the cellular carbohydrate. On the other hand, when the cellular carbohydrate was substituted for the soluble specific substance in this experiment, no sensitization was demonstrated toward either the soluble specific substance or the cellular carbohydrate.

In the foregoing experiments, the entire mixture of serum and carbohydrate was injected. In previous experiments,[1] in which the precipitates were removed, the supernatant antiserum, after precipitation by the soluble specific substance, sensitized the guinea pig to the cellular carbohydrate but not to the soluble specific substance. Antiserum, after precipitation by the cellular carbohydrate, however, failed to sensitize the guinea pig to either substance.

The results of these experiments correspond with those previously reported and record additional data concerning the broader activities of the cellular carbohydrate as compared with the soluble specific substance of Avery and Heidelberger.

7283 P

Failure of Antipneumococcus Horse Serum to Sensitize Guinea Pigs to Anaphylactic Shock with Specific Carbohydrates.

RACHEL BROWN.

From the Division of Laboratories and Research, New York State Department of Health, Albany.

Previous experiments have demonstrated noteworthy differences in antipneumococcus sera from the horse and from the rabbit. For

example, immune horse serum, in striking contrast to immune rabbit serum, intensifies the purpuric activity of the type-I pneumococcus cellular carbohydrate in mice.[1] Also, these 2 sera behave very differently with respect to the fixation of complement in the presence of specific carbohydrates[2] and precipitation in the presence of the partial hydrolysis products of type-III soluble specific substance.[3] Finally, marked differences have been reported[4, 5] in the passive sensitization of the guinea pig to the soluble specific substance by immune horse and rabbit sera; the latter sensitizes, while the former does not. Evidently there are fundamental differences in the action of the sera of the horse and the rabbit immunized with the pneumococcus, concerning which it is important to obtain further information.

In the experiments here summarized, type-I antipneumococcus horse serum, alone or in combination, was used as the sensitizing agent and the homologous soluble specific substance or the cellular carbohydrate as the test material for sensitization. The observations of others have been limited to the soluble specific substance, but in these experiments similar results were obtained with both carbohydrates. The sensitizing dose was given to guinea pigs intraperitoneally and the test dose intracardially 24 or 48 hours later.

The problem of passive sensitization with antipneumococcus horse serum was considered from the point of view that this serum either lacked a factor necessary for the sensitization of guinea pigs or contained an inhibiting substance.

The first group of experiments demonstrated that type-I antipneumococcus horse serum did not become a passive sensitizing agent for anaphylactic shock when mixed with normal rabbit serum, antisheep amboceptor produced in the rabbit, heterophile antibody, or with the sera of rabbits which had been inoculated with any one of the following: type-I antipneumococcus horse serum, the soluble specific substance, the cellular carbohydrate or the ether-soluble fraction of the pneumococcus. Thus, none of these added sera (inactive in themselves) was capable of supplying a missing factor in the horse serum.

The second group of experiments was divided into 2 series. From the first series it was shown that the sensitizing action of minimal

[1] Wadsworth, Augustus, and Brown, Rachel, *J. Immunol.*, 1933, **24**, 349.

[2] Avery, O. T., and Heidelberger, Michael, *J. Exp. Med.*, 1925, **42**, 367.

[3] Heidelberger, Michael, and Kendall, F. E., *J. Exp. Med.*, 1933, **57**, 373.

[4] Avery, O. T., and Tillett, W. S., *J. Exp. Med.*, 1929, **49**, 251.

[5] Mehlman, Julia, and Seegal, B. C., *J. Immunol.*, 1934, **26**, 1.

doses of type-I antipneumococcus rabbit serum was not affected by the addition of normal or type-I antipneumococcus horse serum. In the second series neither inactivation of type-I antipneumococcus horse serum at 56°C. nor treatment with the homologous bacterial cells or agar rendered the serum capable of passively sensitizing guinea pigs. These experiments, therefore, failed to demonstrate any inhibiting action of the immune horse serum.

A further attempt to activate the serum by a combination of the above methods, in which normal rabbit serum was added to type-I antipneumococcus horse serum, after treatment with a suspension of pneumococci (type-I) and centrifugalization, was likewise unsuccessful.

7284 C

A Note on the Spread of Poliomyelitis Virus in Monkeys.

JOHN A. TOOMEY.

From the Department of Pediatrics, Western Reserve University, and the Division of Contagious Diseases, City Hospital, Cleveland, Ohio.

Jungeblut and Spring[1] transected the spinal cords of 2 monkeys at the level of the first lumbar vertebra and then injected them intracerebrally with poliomyelitis virus. One animal developed a condition which might have been poliomyelitis, but when separate emulsions, made from the upper and lower parts of the cord, were injected into other monkeys, the disease was not reproduced. Postmortem autolysis prevented a positive histological diagnosis. The second animal developed poliomyelitis. Histologically, there was an absence of lesions considered typical for poliomyelitis in sections taken from the lumbar cord, while those obtained from the cervical area were positive. Injections of an emulsion made from the upper or cervical section of the cord of this monkey reproduced the disease, while an emulsion made from the lower or lumbar section did not do so when injected into another animal.

Previously[2] I reported that when the virus of poliomyelitis was injected into the sciatic nerves of Macacus rhesus monkeys, the disease was produced in the cervical area even though the cord had

[1] Jungeblut, C. W., and Spring, W. J., PROC. SOC. EXP. BIOL. AND MED., 1930, **27**, 1076.

[2] Toomey, John A., PROC. SOC. EXP. BIOL. AND MED., 1934, **31**, 502.