practically always infected superficially, may be partially sterilized with little injury to the tissue by rinsing the surface quickly with weak alcohol (60 per cent.). In a large number of preparations from a piece of skin treated in this way, a fair percentage will show no bacterial contamination, and some of the remainder will show only occasional colonies. We have obtained a good growth of epithelium from pieces of circumcision tissue thus treated.

A large number of antiseptics and disinfectants—toluol, chloretone, tricresol, phenol, silver nitrate, hypochlorites (Dakin's solution), argyrol, iodine, potassium cyanide, and bichloride of mercury, have been tested on tissues more diffusely infected. For nearly all of these the strength of solution necessary to kill bacteria (staphylococcus aureus) also injures the cells.

Experiments carried out so far, however, indicate that potassium cyanide and probably also bichloride of mercury are exceptions to this rule. For example, potassium cyanide in 1–2,000 dilution is a very good disinfectant but injures cells very slightly. More complete reports of these experiments will be presented in a subsequent communication.

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Development of immune reactions in serum disease.

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The occurrence of immune reactions to horse serum and their relationship to the development of serum disease in man, we have studied by two methods: first, the sensitiveness of the skin to intravenous injections of 0.02 c.c. of horse serum, undiluted or diluted ten times or one hundred times with 0.85 per cent. NaCl; and secondly, by determining the presence of anaphylactic antibody in the blood serum of the patient by transference to guinea-pigs through passive sensitization.

Eleven patients have been studied, who have received for therapeutic purposes from 4 c.c. to 350 c.c. of horse serum, in the

form of diphtheria antitoxin, antimeningococcus serum or antipneumococcus serum, intravenously, intraspinally or intramuscularly. Nine of the eleven cases developed serum sickness.

All of the cases, whether or not they developed serum disease, showed sooner or later a positive specific reaction to the intracutaneous injection of horse serum. This was never obtained before the seventh day following the first therapeutic injection of horse serum and was first observed between this day and the eighteenth. It was never demonstrable until after the appearance of serum disease.

Anaphylactic antibodies could not be demonstrated in the two cases that did not develop serum disease. In all of the other nine cases these antibodies were found at some time in the serum of the patient. In but one case did they appear before the onset of serum disease and then on the fifth day after the therapeutic injection of horse serum. Neither in this instance nor in any other was the anaphylactic antibody demonstrable in the patient's serum during the early part of serum sickness. In all nine cases the anaphylactic antibody was present in maximum concentration at the close of the serum sickness and in one instance persisted for sixty-eight days after the disease. In two cases in which the original attack of serum sickness was followed by a relapse, the antibodies could not be definitely demonstrated until the end of the relapse, that is twenty-one and twenty-four days after the therapeutic injection of horse serum. In several instances it was possible to sensitize guinea-pigs both passively and actively to horse serum with portions of the same specimen of blood serum drawn from the patients towards the close of the serum sickness, thus demonstrating that some of the proteins of horse serum and antibodies for the proteins of horse serum may exist at the same time in the circulation in man.

These experiments show that anaphylactic antibodies for horse serum appear in maximum concentration in the blood serum towards the close of serum sickness and suggest that their presence in the circulation in large amounts determines the recovery from this disease.