Secretin powder S I, (Ivy) which is relatively stable, was used as a standard.

A unit has been arbitrarily defined as the amount of gall bladder contracting material present in 0.2 cc of solution which, when injected intracardially into 30 g frogs, brings about contraction in 50% of 30 experiments.

The activities of 3 crude secretin preparations from dog duodena and a powdered cholecystokinin, prepared according to Ivy's pH 1802 method, have been compared to the standard S I powder. The powders were used in strength of 0.030, 0.025, 0.020, and 0.015% for the S I and 0.1 and 0.2% for the cholecystokinin. The crude secretin preparations were used in dilutions of 1:30, 1:25, 1:20 and 1:15. It was found that 0.2 cc of a 0.020% S I solution gave contraction in 47% of 32 experiments. Cholecystokinin powder in 0.1% solution gave contraction in 19% of 16 experiments while in a 0.2% solution, it gave a gall bladder contraction in 80% of 20 experiments. The crude secretin preparations A, A_1, and B in a dilution of 1:25 gave gall bladder contractions respectively in 50% of 8 experiments, 56% of 32 experiments, and 47% of 30 experiments. Converting these results into terms of units, 1 mg of S I would be the equivalent of 25 units. 1 mg cholecystokinin would be the approximate equivalent of 3 units. Undiluted secretin preparations would contain the equivalent of approximately 125 units per cc or the equivalent of 5 mg of S I per cc.

10662 P

Immunization of Mice by Intranasal Instillation of Nasopharyngeal Washings from Cases of St. Louis Encephalitis.*

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The 1937 outbreak of encephalitis in St. Louis afforded an excellent opportunity for further study of the disease. Despite the fact that the virus of encephalitis has never been demonstrated in nasal secretions procured from patients during the acute phase of the

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disease,[1, 2] it is held by some workers that the mode of spread is by way of the upper part of the respiratory tract. It is well known that the disease is easily produced in mice by the instillation of mouse-brain virus directly into the nares. Furthermore, Armstrong[3] has reported that 30 to 60% of mice which had withstood an intranasal instillation of the virus may have become immune and would survive a subsequent intracerebral injection of an amount sufficient to kill normal controls.

In the present experiments nasopharyngeal washings from patients with the disease were given intranasally to mice on 2 successive days in order to attempt to demonstrate the presence of active virus. This route was used since material containing bacteria can be used as the inoculum, thus avoiding possible loss of virus by filtration or other means of removing the contaminating bacteria. Since none of the mice receiving intranasal instillation of nasopharyngeal washings developed recognizable symptoms of the disease, we attempted to determine whether any immunity was conferred by these instillations.

About 3 weeks after the last instillation, the mice were given an intracerebral inoculation of an amount of virus which was sufficient to kill all (20) control mice. Of the 40 test mice, 14 (35%) survived the injection. This observation encouraged us to make further trials. Accordingly, washings from 15 patients during the acute stage of the disease were instilled into the nares of a series of mice. However, in these experiments 7 to 8 instillations were made. None of these animals developed clinical signs of the disease, but a similar test for immunity showed an even higher incidence of survivors. Of the 164 test mice, 81 (49%) survived the subsequent intracerebral injection, while all of the 54 control mice died.

These experiments suggest that virus is present in the secretions of the upper respiratory tract during the clinical disease and the mode of transmission may be by means of such discharges.

Since the above findings were obtained, no further clinical cases have been available for study so that there has been no opportunity to repeat these experiments on a larger scale. We are reporting them at this time because of the possibility that cases may occur again this summer, at which time other workers might attempt the detection of the virus in the nasopharyngeal washings of patients and particularly of contacts.

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Attempts to Infect Guinea Pigs with the Virus of St. Louis Encephalitis.*

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It is known that passage of influenzal virus directly from human patients to mice is extremely difficult,[1] but that passage to ferrets and then to mice is readily accomplished. It is also probably true that passage of smallpox virus directly from patients to calves or rabbits is difficult, while previous passage through monkeys converts the virus to vaccinia which is then more readily infectious for calves and rabbits.[2, 3, 4] Reasoning by analogy, it was suspected that susceptibility to St. Louis encephalitis virus might be transmitted to a wider variety of animals if brain-tissue of suitable animal species were used as inoculum. In spite of the fact that previous attempts to infect guinea pigs with human brain material[5, 6] or mouse-brain virus[7, 8] have been unsuccessful, we have considered this worthy of another trial with the view of using guinea pig brain virus for the inoculation of other animals.

In the first series of experiments, begun in the fall of 1937, mouse-brain virus suspended in Locke's solution was introduced intra-

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