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.

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A Note on the Spread of Poliomyelitis Virus in Monkeys.

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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.

been transected and removed in the region of thoracic X. The only connection between the upper and lower parts of the cord was by way of the sympathetic nerve fibers.

In these animals, the pathological picture of the distal or lumbar sections of the cord showed complete tissue degeneration, practically an autolysis in all but one specimen as well as a diffuse, but sparse, round cell infiltration and a few leucocytes. There was neither dilatation of nor cuffing about the capillaries. In the one specimen from the lumbar area referred to in the foregoing report, there was slight cellular degeneration, a relative decrease in the number of neurones in the anterior horn cell area and the same diffuse, but sparse, round cell infiltration. There was a lack of capillary dilatation and cuffing. The capillaries in the proximal or cervical sections of the cord were dilated and had cuffing about the vessels. There was also a generalized infiltration of round cells and anterior horn cell degeneration. The cells seen in the cervical cord sections took the contrasting stains, while the whole section from the lumbar cord, save for the lymphocytes present, was a pinkish red.

I agree with Jungeblut and Spring[1] that the spinal fluid plays no essential part in the transfer of the virus along the central nervous system. From my own work, however, I could not understand why, if the virus could travel up the sympathetic nerve fibers from the sciatic nerve to the cervical area, it could not just as well travel by way of the sympathetic system from the upper to the lower part of the cord.

In experiments where the cord is cut and the virus injected intracerebrally, it is obvious that the spread of the virus along the main pathway, *i. e.*, the cord itself, is interrupted, and the virus, in order to reach the lower part of the cord, would have to travel by the somewhat devious route of the sympathetic nervous system, if at all. It was logical, then, to assume that the amount of virus might not be so great in the distal portion of the cord as it would be in the proximal or cervical end. The time element might also play a part, since a sufficient concentration of virus might not have spread to the lumbar area before the animal had died.

The authors[1] correctly inferred that broad conclusions could not be drawn from their experiments and it was thought that further experiments along the same line should be done.

Four monkeys were transected in the region of dorsal IX, and ½ inch of the cord was removed. Transection was made at this height in order to make sure that the virus would not be transferred

along the normal nerve fiber connections between the last thoracic and the first 2 lumbar nerves. The animals were then injected intracerebrally with 1.5 cc. of a 2% emulsion of a potent poliomyelitis virus. All 4 monkeys developed tremor and weakness of the muscles of the arms and upper back from 6 to 7 days after the injection. Suddenly, within 24 hours after the weakness and tremor had begun, or 7 to 9 days after the injection of the poliomyelitis virus, all of the animals died so that at first it was not absolutely evident that the animals had poliomyelitis. The abrupt onset of clinical symptoms, with death occurring so soon after, was in contrast with the usual clinical picture seen in monkeys used in other transection experiments where the sciatic nerve had been used for transference of the virus. The latter animals had been injected with virulent poliomyelitis virus and gradually went through all the stages usually seen in monkeys (weighing from 4 to 6 pounds) that develop experimental poliomyelitis, such as furring, tremor, paresis, paralysis, complete quadriplegia and usually death.

Sections were taken from the cervical and lumbar portions of each cord for microscopic examination. Histologically, the cervical sections from all 4 animals were typical enough to make a diagnosis of poliomyelitis and they looked similar to those taken from the same area in animals in which the virus had traveled from the sciatic nerve along the sympathetic system to the upper part of the cord. The lower sections of the cord in 2 specimens looked like sections of the lumbar area previously described herein (after sciatic injection), the cellular elements being nearly autolyzed with the same diffuse, but sparse, round cell infiltration and all the cellular elements, save for the lymphocytes, stained a homogenous pink. There was no dilation of the capillaries, perivascular infiltration or edema.

The histological sections from the lumbar cords of the other 2 animals showed only a relative decrease in anterior horn cells, occasional cuffing, definite capillary increase with dilatation and some sparse, but diffuse, round cell infiltration with an occasional leucocyte present. The cells that were present took the contrasting hematoxylin eosin stain.

Separate emulsions were made from the remaining pieces of the lumbar cord from each of the 4 monkeys and each one was injected into each of 4 other animals. The cervical cord was not tested for pathogenicity since this point was not in question.

For reasons stated previously, it was decided to use a greater concentration of lumbar cord emulsion for the transfer experi-

ments and to inject 2 cc. of a 10% emulsion of poliomyelitis virus intracerebrally for passage transmission of the disease. The amount of lumbar cord remaining after section was only enough to make the required 10% emulsion in one instance. The other 3 animals received the same amount of virus, however, though in a greater dilution of saline. Three of the 4 animals contracted typical poliomyelitis; 2 developed complete quadriplegia and died; one developed weakness of one leg and paralysis of the other and recovered. The remaining animal had furring, but did not develop paralysis.

The 2 animals which developed quadriplegia had been injected with emulsions made from lumbar cords of animals whose sections took the contrasting stains. The others were injected with material made from devitalized lumbar cord emulsions from animals whose sections took the acid pink stain.

Although virus was present in the lower cord, it must have been relatively decreased as compared to the amount present in the upper cord. This is comprehensible when one remembers that, in transecting the cord, the normal blood supply may be disturbed to such an extent that degeneration of nerve fibers of the lower cord would naturally often follow the operation. Since the degeneration of nervous tissue is obvious in some specimens and the area is not completely normal even in the non-degenerated ones, it might be presumed that the absorption of the virus might be so slow that the presence of the small amount that would have been absorbed before the death of the animal would be difficult to demonstrate in passage experiments. It is interesting to point out that after a nerve is sectioned and degeneration of the axis cylinder has set in, absorption of poison ceases.[3] In the experiments herein described, complete degeneration may not have set in, but enough changes may have taken place after these transection operations to accomplish practically the same effect and thus limit absorption of the virus.

It was interesting to note the modification of the clinical picture in the experimental animal brought about by the method of producing the disease as described.

Summary. In transected animals, transfer of the virus can occur from the upper to the lower part of the cord after intracerebral injections of the virus of poliomyelitis as well as from the lower to the upper part of the cord after sciatic injection, presumably along the sympathetic nerve fiber connection.

[3] Meyer and Ranson, quoted by Zinsser, Hans, in "Resistance to Infectious Disease," Ed. 4, New York, The Macmillan Co., 1931, p. 43.