EXPERIMENTS WITH POLIOMYELITIS IN THE RABBIT.

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Plates 77 to 80.

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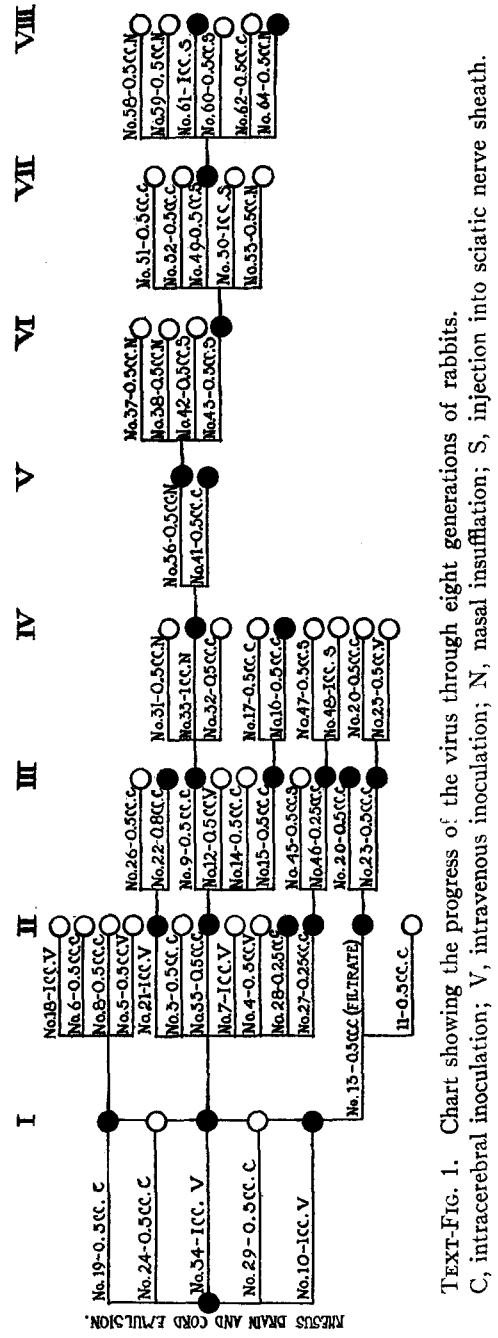
Inoculation of the common laboratory animals with the virus of poliomyelitis has met with so little success that the disease has been generally regarded as exclusively limited to man and the monkey. Attempts have been made to transmit the virus to many animals, but of all the animals tested, positive results have thus far been recorded only for monkeys, rabbits, and, perhaps, guinea pigs.

Krause and Meinicke[1] reported the passage of a strain of virus obtained from a human case through seven generations in rabbits. Lentz and Huntemüller[2] report having successfully transferred the virus from rabbit to rabbit by various methods of inoculation. They found the alterations in the brain and spinal cord to be slight as compared with those in monkeys. On the other hand, Römer and Joseph,[3] Landsteiner and Levaditi,[4] Leiner and von Wiesner,[5] and Flexner and Lewis[6] all failed to transfer the disease to rabbits. The most striking results, perhaps, have been reported by Marks,[7] who carried a strain of poliomyelitic virus derived from a *rhesus* monkey through seven generations of young rabbits varying in weight from 350 to 550 gm. The animals that succumbed developed no paralysis, but died in convulsions. No lesions definitely characteristic of poliomyelitis could be found on microscopic examination. Marks says: "The disease thus produced in rabbits cannot be recognized as poliomyelitis," although he concludes that filtrates of the nervous tissues of monkeys dying from experi-

[1] Krause, P., and Meinicke, E., *Deutsch. med. Wchnschr.*, 1909, xxxv, 1825.
[2] Lentz and Huntemüller, *Ztschr. f. Hyg. u. Infectionskrankh.*, 1910, lxvi, 481.
[3] Römer, P. H., and Joseph, K., *München. med. Wchnschr.*, 1910, lvii, 2685.
[4] Landsteiner, K., and Levaditi, C., *Compt. rend. Soc. de biol.*, 1909, lxvii, 787.
[5] Leiner, C., and von Wiesner, R., *Wien. klin. Wchnschr.*, 1909, xxii, 1698.
[6] Flexner, S., and Lewis, P. A., *Jour. Exper. Med.*, 1910, xii, 227.
[7] Marks, H. K., *Jour. Exper. Med.*, 1911, xiv, 116.



TEXT-FIG. 1. Chart showing the progress of the virus through eight generations of rabbits.
C, intracerebral inoculation; V, intravenous inoculation; N, nasal insufflation; S, injection into sciatic nerve sheath.

mental poliomyelitis are not wholly innocuous to young rabbits. Marks further states that "not all strains of the virus can be transmitted successfully to even a small fraction of individuals of all varieties of domesticated rabbits." This fact, which we have corroborated, may explain the negative results of other investigators.

Römer and Joseph[8] have observed that guinea pigs in the laboratory occasionally die of a paralytic disease. They were unable to transfer poliomyelitic virus from monkeys to guinea pigs, although Römer[8] found that the spontaneous paralysis was due to a filterable virus. Neustaedter[9] claims to have carried a strain from a guinea pig presumably infected through contact with a monkey into one other guinea pig and back again to a monkey.

In the course of our experiments we have inoculated a few guinea pigs. The lesions in those that succumbed were somewhat similar to the lesions seen in the rabbits. This part of our work is not sufficiently advanced to warrant conclusions. Moreover, we have no criterion by which to establish what is and what is not poliomyelitis, except by reproducing the disease in monkeys. It is, therefore, hazardous to affirm or deny the identity with poliomyelitis of these aberrant conditions in other animal species.

The difficulty of obtaining monkeys on account of the war led us to consider other animals that might be susceptible and therefore suitable for experimental purposes. We selected the rabbit because successful results had previously been obtained with these rodents and because rabbits offered a good chance to compare the action of poliomyelitic with rabic virus. We accordingly inoculated rabbits with poliomyelitic virus from a *rhesus* monkey infected by intracranial inoculation with a strain sent us from The Rockefeller Institute for Medical Research by Dr. Harold L. Amoss. We have obtained positive results in young rabbits and have succeeded in transferring the virus from rabbit to rabbit through eight successive generations.

Intracranial inoculations have been used for the most part, although infection has taken place through intravenous injection, also through injection into the sciatic nerve sheath, and even after introduction of the virus into the anterior nares, upon the uninjured nasal mucosa. A few intraperitoneal injections were tried, but with negative results.

[8] Römer, P. H., *Ergebn. d. inn. Med. u. Kinderh.*, 1912, viii, 1.

[9] Neustaedter, M., *Jour. Am. Med. Assn.*, 1913, lx, 982.

All inoculations are by no means successful. Over one-half fail; we obtained positive results in twenty-two rabbits out of a total of fifty-four inoculated in various ways. Of five rabbits inoculated with the virus from the *rhesus* monkey two have failed to show symptoms, although kept under observation for 5 months.

The age incidence of poliomyelitis in man is indicated by the name "infantile paralysis." In our experimental work we obtained positive results in young rabbits only. We failed to infect three full grown rabbits with *rhesus* virus, although this same virus caught in three out of five young rabbits. Furthermore, rabbit virus from the second and third generations was transferred to rabbits 8 weeks old with negative results. Thereafter we used only young animals under 6 weeks old in our experiments. There seems to be a parallel between rabbits and man as far as susceptibility of the young is concerned. These facts seem to furnish a striking example of natural immunity acquired during the period of adolescence.

The incubation period in our observations of twenty-two rabbits has been variable, the shortest being 2 days, the longest, 41. In this respect our experience is similar to that of Lentz and Huntemüller,[2] who found the incubation period to be of uncertain length, sometimes as long as 2 months, but usually between 7 and 11 days. The average period of incubation in our observations was 12 days. Curiously enough, the shortest period, 2 days, as well as the longest, 41 days, was after intracranial injections.

The virus so far has given no evidence of increasing adaptation to rabbits, or of becoming fixed to any degree, the number of failures being as great, and the period of incubation as variable, in the eighth generation as in the first.

The symptoms also vary. They can, however, be divided roughly into two classes: (1) A type which we designate the progressive type, in which the rabbit first becomes inactive, loses weight, and appears weak. This is followed by partial or complete paralysis of one or more of the extremities (Fig. 1), which usually progresses until death. This corresponds somewhat to the syndrome most commonly seen in experimental poliomyelitis in monkeys. The duration of the disease varies from 1 or 2 days to 1 week. The paralysis is usually flaccid, but occasionally it is spastic. Sometimes the paralysis be-

gins locally. As a rule, it is easy to determine that the paralysis is a true palsy and not simply "weakness." (2) The other group of symptoms, which we designate the fulminating type, is more explosive in character, develops suddenly, and terminates in a very short time; it never extends over more than 2 days and usually lasts only a few hours. The overshadowing symptoms are extreme weakness and marked dyspnea. As an illustration of the rapid course of the symptoms of the fulminating type the case of Rabbit 21 is cited. This rabbit was inoculated intravenously on Aug. 18 with 1 cc. of a rich emulsion of virus from a first generation rabbit (No. 34). On Sept. 12, 25 days later, it still appeared to be normal. In about an hour after this observation it was found by one of us lying flat on its side, breathing in slow, labored gasps. There was great general weakness, the rabbit being unable to raise its head, but no paralysis of the extremities was demonstrable. Within a few minutes the animal was dead, the whole syndrome lasting less than an hour. This is an extreme instance of the fulminating type, and is of special interest since it follows a long period of incubation. We have seen combinations of the two types with symptoms resembling Landry's paralysis.

Most of the animals in which the symptoms lasted over 2 or 3 days lost weight rapidly. In some cases the loss of weight was the first observed symptom.

That the rabbit virus is filterable is shown by the protocol of Rabbit 13 of the second generation. This rabbit was inoculated intracranially with 0.5 cc. of a Berkefeld filtrate of brain and cord emulsion from Rabbit 10. Six days later the hind legs became paralyzed, and the animal died 2 days after onset with symptoms of the fulminating type. The unfiltered virus from Rabbit 13 proved infectious for two other rabbits (Nos. 20 and 23).

We have inoculated a monkey with virus from Rabbit 49 of the seventh generation. The monkey died after an incubation period of 4 weeks, with symptoms somewhat resembling the fulminating type seen in the rabbits. The gross pathological findings were congestion of the pia, especially in the region of the medulla, and hyperemia and hemorrhage of the gray matter of the medulla and cord. The microscopic lesions were similar to those seen in the rabbits and not typi-

cal of experimental poliomyelitis in monkeys. The monkey was a South American species which we have since found to be highly resistant to poliomyelitic virus derived from *rhesus* monkeys. This interesting observation is being made the subject of further study.

Flexner found that *Capucinus* monkeys were resistant, although *Cebus*, another South American species, were susceptible, but less so than Old World monkeys. He therefore concludes that the *Platyrrhines* are less susceptible than the *Catarrhines*.[10]

The intracranial inoculations were done by making a small incision in the skin, near the midline, drawing this to one side, and introducing the needle of the syringe directly through the frontal bone, which is thin and soft in young rabbits. The virus is then injected slowly into the region of the lateral ventricle. On withdrawing the needle, the skin slides back and acts as a valve to cover the small opening in the bone.

The emulsion of virus in all cases was made by grinding in a mortar, portions of the brain and cord with salt solution, and filtering through several layers of sterile gauze.

In Table I are briefly summarized the results of the rabbits which succumbed to intracranial inoculation.

Fourteen rabbits inoculated intracranially failed to show symptoms.

The intravenous inoculations were made into the posterior auricular vein with an emulsion of the central nervous matter prepared as above described.

Table II contains a condensed summary of the rabbits which succumbed to intravenous inoculations.

Six rabbits inoculated intravenously failed to show symptoms.

In order to inject the virus into the sciatic nerve sheath, a short incision was made in the skin above the nerve, the tissues were dissected sufficiently to render the nerve visible, and the needle was then inserted into the sheath in the central direction. The emulsion was prepared as above.

Table III gives a condensed summary of the rabbits which succumbed as a result of injection into the sciatic nerve sheath.

Five rabbits inoculated into the sciatic nerve sheath failed to develop symptoms.

[10] Flexner, S., *Jour. Am. Med. Assn.*, 1910, lv, 1105.

TABLE I.

Intracranial Inoculation.

No. of rabbit.	Amount of emulsion.	Rabbit generation.	Period of incubation.	Type of symptoms.*
	cc.		*days*	
19	0.5	I	19	P.
35	0.5	II	5	P.
27	0.25	II	31	F.
28	0.25	II	34	F.
13	0.5	II	6	F.
20	0.5	III	41	F.
23	0.5	III	9	P.
22	0.8	III	10	P.
46	0.25	III	10	Convulsions.
15	0.5	III	2	F.
9	0.5	III	14	P.
16	0.5	IV	13	P.
41	0.5	V	12	P.

* In this and the following tables, the letter P indicates symptoms of the progressive type; F, symptoms of the fulminating type.

TABLE II.

Intravenous Inoculation.

No. of rabbit.	Amount of emulsion.	Rabbit generation.	Period of incubation.	Type of symptoms.
	cc.		*days*	
34	1.0	I	18	F.
10	1.0	I	3	F.
21	1.0	II	25	F.

TABLE III.

Inoculation into the Sciatic Nerve Sheath.

No. of rabbit.	Amount of emulsion.	Rabbit generation.	Period of incubation.	Type of symptoms.
	cc.		*days*	
43	0.5	VI	8	P.
49	0.5	VII	6	F.
61	1.0	VIII	20	F.

In order to introduce the virus into the nose, the following procedure was adopted. The rabbit was lightly etherized, held on its back, and the emulsion of the virus then dropped into the anterior nares from an ordinary medicine dropper, care being taken not to injure the mucosa. There was usually a little sneezing immediately after the fluid was introduced.

Table IV is a condensed summary of the three rabbits which succumbed as a result of intranasal insufflation.

TABLE IV.

Intranasal Insufflation.

No. of rabbit.	Amount of emulsion.	Rabbit generation.	Period of incubation.	Type of symptoms.
	cc.		*days*	
33	0.5	IV	2	F.
36	0.5	V	2	F.
64	0.25	VIII	2	F.

Six rabbits inoculated by intranasal insufflation failed to develop symptoms.

It will be seen that, judging from the limited data at hand, this is a particularly virulent way of infecting rabbits with the virus. The period of incubation is short—only 2 days—and the symptoms in all three cases were of the fulminating type. These represent only three out of nine rabbits tested by introducing the virus into the nose. The other six failed to take. Particular attention was paid to the lungs of the rabbits in this group at autopsy. There was no evidence of pneumonia, or even of congestion of the lungs. On microscopical examination it was found that the medulla was markedly congested, whereas the cord was only slightly affected.

The gross lesions consist of injection of the vessels of the pia, hyperemia of the gray matter of the medulla and cord, and more or less marked edema throughout the brain and cord. The microscopic lesions are distributed rather uniformly throughout the gray matter of the cord and medulla in the progressive type. In the fulminating group (those exhibiting symptoms of respiratory failure without

paralysis of the skeletal muscles), the lesions are more marked in the medulla.

The microscopic lesions consist of capillary congestion, focal hemorrhages into the gray matter, degeneration of the large motor cells, and infiltration with cells of uncertain origin (Figs. 2, 3, 4, 5, 6, 7, and 8). These cells for the most part seem to be proliferated glia cells, and therefore appear to be different from the infiltrating lymphocytes of the lesions of poliomyelitis in man. The congestion of the capillaries and small arterioles is conspicuous. The infiltrating cells when stained with eosin and methylene blue have large, vesicular nuclei with a number of conspicuous chromatin granules. The cytoplasm is scanty and homogeneous. They are scattered throughout the gray matter, and are also grouped in satellite arrangement around the nerve cells, but the perivascular infiltration, so typical of the lesions of poliomyelitis in man and the monkey, is absent in the rabbit. Punctate hemorrhages are numerous, sometimes every vessel in a field being ruptured. The nerve cells show all stages of degeneration. Chromatolysis is common and satellitosis is an almost constant feature. A moderate degree of round cell infiltration is occasionally seen in the meninges. The accompanying plates illustrate the different features of the lesions. The complete picture gives the impression of a severe intoxication of the gray matter of the cord and medulla.

The following are the protocols in brief of the rabbits that succumbed, arranged chronologically, as in Text-fig. 1.

Rabbit 34.—Age 6 wks. Weight 660 gm. 1st generation. 1 cc. of *rhesus* virus intravenously. Incubation period 18 days. Died in the night with no observed symptoms.

Rabbit 19.—Age 5 wks. Weight 600 gm. 1st generation. 0.5 cc. of *rhesus* virus intracranially. Incubation period 19 days. Death in 4 days. Paralysis of front legs with respiratory symptoms.

Rabbit 10.—Age 4 wks. Weight 550 gm. 1st generation. 1 cc. of *rhesus* virus intravenously. Incubation period 3 days. Died in the night with no observed symptoms.

Rabbit 21.—Age 5 wks. Weight 600 gm. 2nd generation. 1 cc. of virus of Rabbit 34 intravenously. Incubation period 25 days. Death in 1 hour. Symptoms very explosive in character; complete prostration and marked dyspnea.

Rabbit 35.—Age 6 wks. Weight 730 gm. 2nd generation. 0.5 cc. of virus of Rabbit 34 intracranially. Incubation period 5 days. Hind legs became

paralyzed. Paralysis remained stationary for 5 days, then showed tendency to improvement. Chloroformed.

Rabbit 27.—Age 3 wks. Weight 290 gm. 2nd generation. 0.25 cc. of virus of Rabbit 34 intracranially. Incubation period 31 days. Death in 1 day. Paralysis and dyspnea.

Rabbit 28.—Age 3 wks. Weight 255 gm. 2nd generation. 0.25 cc. of virus of Rabbit 34 intracranially. Incubation period 34 days. Death in 1 day. Weakness and dyspnea.

Rabbit 13.—Age 4 wks. Weight 540 gm. 2nd generation. 0.5 cc. of Berkefeld filtrate of emulsion of virus of Rabbit 10 intracranially. Incubation period 6 days. Death in 2 days. Paralysis of hind legs and respiratory distress.

Rabbit 20.—Age 5 wks. Weight 645 gm. 3rd generation. 0.5 cc. of virus of Rabbit 13 intracranially. Incubation period 41 days. Death in 1 day. Explosive symptoms; prostration and marked dyspnea.

Rabbit 23.—Age 4 wks. Weight 410 gm. 3rd generation. 0.5 cc. of virus of Rabbit 13 intracranially. Incubation period 9 days. Death in 5 days. General weakness and dyspnea.

Rabbit 22.—Age 5 wks. Weight 620 gm. 3rd generation. 0.8 cc. of virus of Rabbit 21 intracranially. Incubation period 10 days. Death in 7 days. Paralysis progressive.

Rabbit 46.—Age 5 wks. Weight 380 gm. 3rd generation. 0.25 cc. of virus of Rabbit 27 intracranially. Incubation period 10 days. Death in 1 day. Died in convulsions.

Rabbit 15.—Age 6 wks. Weight 520 gm. 3rd generation. 0.5 cc. of virus of Rabbit 35 intracranially. Incubation period 2 days. Symptoms progressed rapidly; respiratory distress and great weakness. Chloroformed.

Rabbit 9.—Age 4 wks. Weight 440 gm. 3rd generation. 0.5 cc. of virus of Rabbit 35 intracranially. Incubation period 14 days. Death in 7 days. Progressive paralysis with respiratory distress.

Rabbit 16.—Age 4 wks. Weight 430 gm. 4th generation. 0.5 cc. of virus of Rabbit 15 intracranially. Incubation period 13 days. Death in 8 days. Symptoms progressive in type with dyspnea.

Rabbit 33.—Age 6 wks. Weight 620 gm. 4th generation. 0.5 cc. of emulsion of virus of Rabbit 9, half the amount dropped in each nostril. Incubation period 2 days. Death in 1 day. Symptoms of the fulminating type with paralysis of hind legs towards the last.

Rabbit 41.—Age 5 wks. Weight 560 gm. 5th generation. 0.5 cc. of emulsion of virus of Rabbit 33 intracranially. Incubation period 12 days. Death in 4 days. Symptoms of progressive type.

Rabbit 36.—Age 4 wks. Weight 380 gms. 5th generation. 0.25 cc. of heavy emulsion of virus of Rabbit 33 in each nostril. Incubation period 2 days. Death in 2 days. Symptoms of fulminating type with paralysis.

Rabbit 43.—Age 5 wks. Weight 450 gm. 6th generation. 0.5 cc. of emulsion of virus of Rabbit 36 injected into sciatic nerve sheath. Incubation period 8

days. Symptoms progressive in type. Complete paralysis on 3rd day. Chloroformed.

Rabbit 49.—Age 4 wks. Weight 310 gm. 7th generation. 0.5 cc. of emulsion of virus of Rabbit 43 into sciatic nerve sheath. Incubation period 6 days. Death in 2 days. Symptoms fulminating in type.

Rabbit 64.—Age 4 wks. Weight 400 gm. 8th generation. 0.25 cc. of emulsion of virus of Rabbit 49 into each nostril. Incubation period 2 days. Death in 2 days. Symptoms of fulminating type.

Rabbit 61.—Age 4 wks. Weight 340 gm. 8th generation. 1.0 cc. of emulsion of virus of Rabbit 49 into sciatic nerve sheath. Incubation period 20 days. Died in night with no observed symptoms.

DISCUSSION AND SUMMARY.

The poliomyelitic virus obtained from an experimental monkey has been passed through eight generations in rabbits. It shows no signs of dying out. On the other hand, it gives no evidence of becoming more pathogenic to the species through successive passage. The period of incubation remains variable and the percentage of takes has not increased. Whether eventually a virus can be obtained which is of heightened virulence to rabbits is problematic.

All inoculations are by no means successful. The animals show great individual differences in susceptibility to the virus, as is evidenced by the fact that out of fifty-four rabbits inoculated, only twenty-two, or about 40 per cent, succumbed. This fact may explain the negative results of other investigators. At several points in the series of experiments it was thought that the strain had died out. As many as six rabbits have been inoculated one after the other before the virus would catch again.

The age of the rabbits is important in considering the susceptibility. From the limited data at our command, adult rabbits are resistant, and there appears to be an abrupt increase in resistance between the age of 6 and 8 weeks; that is, rabbits under 6 weeks are more susceptible to the virus. There seems to be a parallel between the age incidence of this disease in rabbits and spontaneous poliomyelitis in man. The age incidence of poliomyelitis in man is indicated by the term "infantile paralysis."

Several methods of inoculation have proved successful; thus the rabbits have succumbed as a result of introducing the virus directly

into the brain, by injecting it into a peripheral nerve, or directly into the circulation, or by placing it upon the uninjured nasal mucosa.

The symptoms produced show more or less departure from the symptoms of poliomyelitis as seen in the spontaneous disease in man and in the experimental disease in the monkey. There are two distinct pictures recognizable. In one there is paralysis of one or more of the extremities which progresses until death, resembling somewhat the symptoms of the experimental disease in the monkey. This we have designated the progressive type. The other group is included in what we have called the fulminating type. The symptoms are explosive in character, with extreme weakness amounting to prostration, terminating in death in a few hours, attendant upon respiratory failure. The mode of inoculation seems to have little effect upon the type of symptoms produced.

The period of incubation is variable and apparently does not depend upon the method of inoculation. The period varied from 2 to 41 days, with an average of 12 days. The two extremes both occurred after intracranial injection. After intranasal insufflation the incubation period was short, being in each case 2 days, followed by symptoms of the fulminating type. The placing of the virus into the nose seems to be an effective method, but is as uncertain as other routes, as only three out of nine rabbits tested in this manner succumbed. The disease produced by this route was particularly virulent.

The virus shows no tendency to become fixed. The period of incubation is as variable in the eighth generation as in the first, and the virus has shown no tendency towards increasing virulence through successive passage, in these respects differing from the virus of rabies.

We have found the virus to be filterable. An emulsion of the central nervous matter of a rabbit of the first generation passed through a Berkefeld filter, and injected intracerebrally into another rabbit, resulted in death, preceded by symptoms of the fulminating type. Virus (unfiltered) from this rabbit was transferred successfully to two other rabbits.

The lesions, while definite and consistent throughout the series, lack the distinctive features of the pathologic picture of poliomyelitis in man and the monkey. Capillary congestion, punctate hemor-

rhages, degeneration of the motor cells, satellitosis, and more or less cellular infiltration of the gray matter of the cord and medulla are found, but perivascular infiltration is absent and the infiltrating cells are not lymphocytic in character.

One of the most striking features of this investigation is the way in which rabbits and monkeys react to the same virus. The disease in the rabbit presents certain clinical resemblances to the experimental disease in the monkey and also to the spontaneous disease in children. On the other hand, the symptoms show marked variation from those seen in the monkey and in man. The picture has not the same constancy in rabbits and could not in most cases be recognized clinically as poliomyelitis. There are still more marked differences in the pathology. While it is true that the brunt of the attack in the rabbit falls upon the gray matter of the cord and medulla, the appearance of the lesions under the microscope shows such differences from the lesions of experimental poliomyelitis in monkeys, as well as the natural disease in man, as to suggest two distinct infections. It is more reasonable, however, to assume that we are dealing with a modified form of poliomyelitis; that the rabbit reacts differently to the virus than the monkey or man; and that the disease produced in rabbits by us and others is in fact poliomyelitis. So far as we know, no other virus produces such differences in two animal species. Smallpox is so profoundly altered in the cow that it took almost 100 years to prove Jenner's assumption that cowpox is a modified form of smallpox. However, the pock of vaccinia is a correct counterpart both clinically and pathologically of the pock of variola. If the virus of poliomyelitis may be so altered in the rabbit as scarcely to be recognizable, may it not be still more profoundly changed in other animals? The conjecture then arises that poliomyelitis, instead of being limited naturally to man and experimentally to monkeys, may in fact occur in other animals in unnoticed or unrecognized form. If this should prove true, it may be a source of human infection and may help to solve the problem of prevention.

EXPLANATION OF PLATES.[11]

PLATE 77.

FIG. 1. Rabbit 19. Paralysis of extremities.

FIG. 2. Rabbit 19. Medulla. Chromatolysis of the nerve cells. × 515.

PLATE 78.

FIG. 3. Rabbit 9. Lumbar cord, showing capillary congestion and hemorrhage. × 180.

FIG. 4. Rabbit 9. Lumbar cord. Numerous punctate hemorrhages, and marked cell degeneration. × 385.

PLATE 79.

FIG. 5. Rabbit 35. Lumbar cord. Cellular infiltration. × 180.

FIG. 6. Rabbit 9. Medulla. Satellitosis around degenerated nerve cells. × 515.

PLATE 80.

FIG. 7. Rabbit 35. Satellitosis. × 515.

FIG. 8. Rabbit 19. Cervical cord. Degeneration of anterior horn cells with satellitosis. × 385.

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[11] We are indebted to Dr. J. P. Bill for the microphotographic work.

FIG. 1

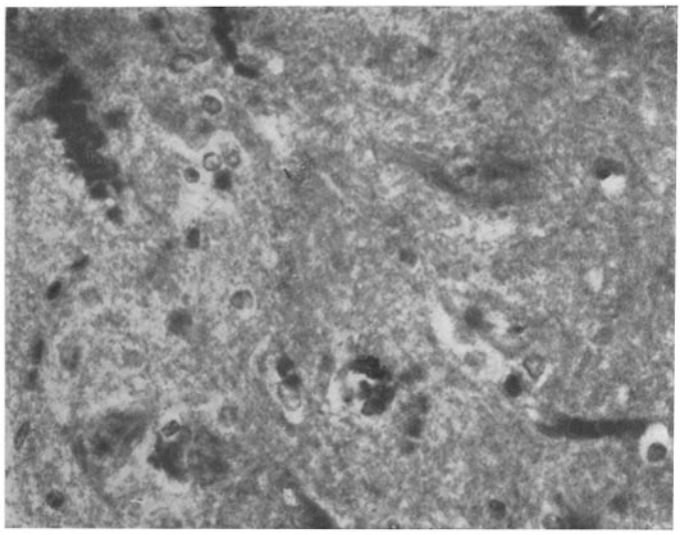

FIG. 2.

(Rosenau and Havens: Poliomyelitis in the Rabbit.

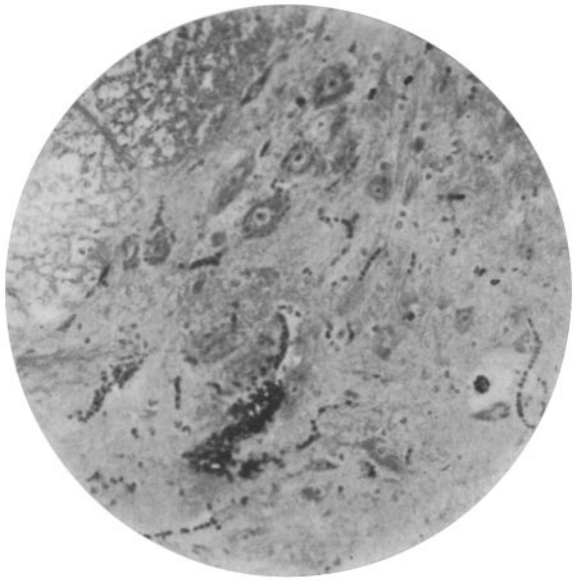

FIG. 3.

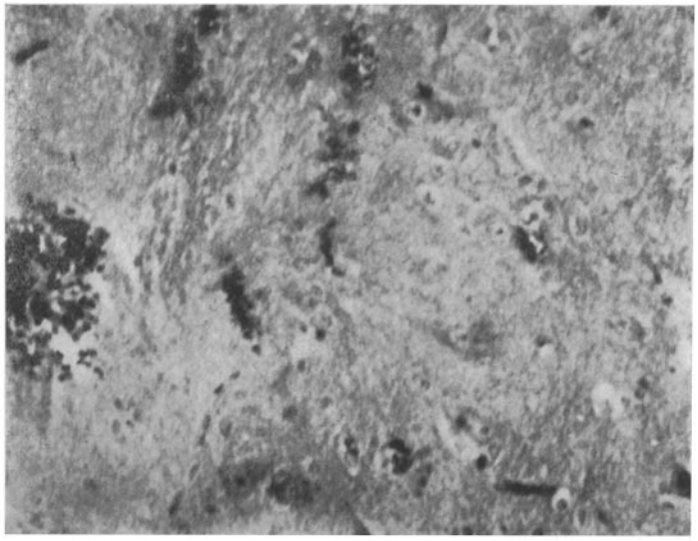

FIG. 4.

(Rosenau and Havens: Poliomyelitis in the Rabbit.)

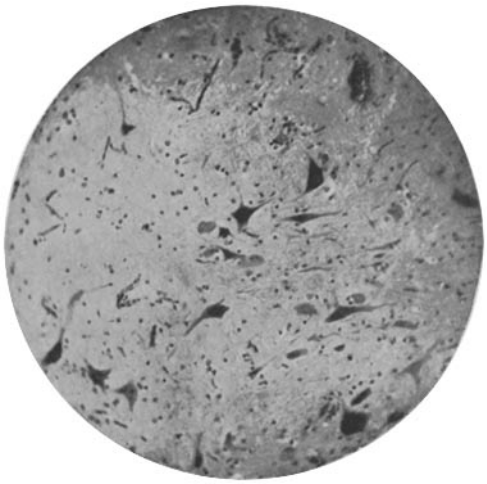

FIG. 5

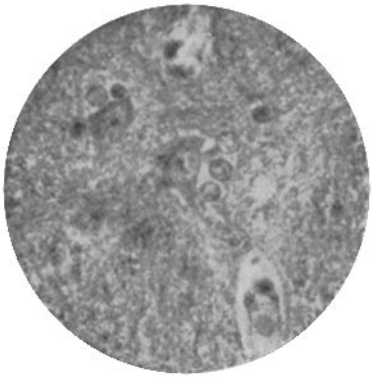

FIG. 6.

(Rosenau and Havens: Poliomyelitis in the Rabbit.)

FIG. 7.

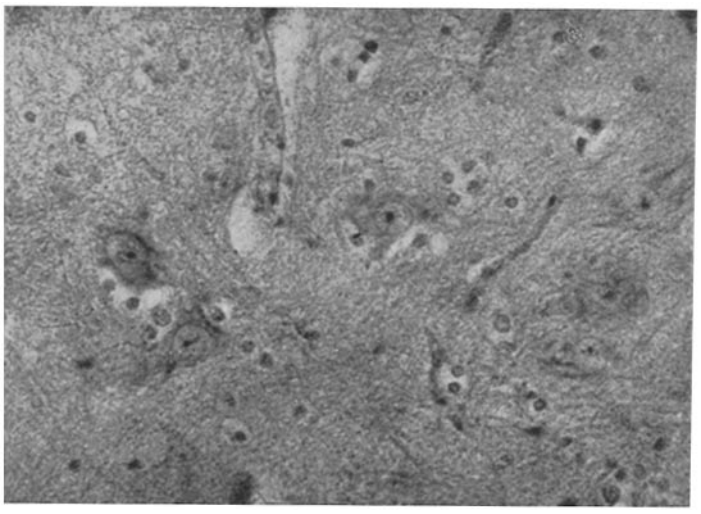

FIG. 8.

(Rosenau and Havens: Poliomyelitis in the Rabbit.)