13263

Failure to Produce Neuronal Injury and Necrosis with Poliomyelitis Virus in Rabbits During Insulin Hypoglycemia.*

J. EMERSON KEMPF, MARJORIE E. PIERCE AND MALCOLM H. SOULE.

From the Hygienic Laboratory, University of Michigan, Ann Arbor.

Sandler[1] has reported that by depressing the blood sugar of rabbits to 66 mg % or less he was able to produce neuronal injury in the anterior horns with the MV strain of the poliomyelitis virus. The hypoglycemia was thought to decrease cellular oxidation in the tissues of the central nervous system, thus lowering in some unknown manner their susceptibility to the virus. His technic for the demonstration of this phenomenon was as follows: Rabbits were fasted 24 hours, after which they were injected subcutaneously with 0.6 to 0.8 of a unit of protamine insulin per kg of body weight. The hypoglycemia which resulted extended from 3 to 5 hours and sometimes longer. The blood sugar estimations were made by the macromethod of Folin and Wu. During the period of hypoglycemia, the virus was inoculated intracranially, and the fasting was continued. Of the 4 rabbits[2] treated in this manner death occurred in 14 hours, 2 days, 6 days, and 15 days, and histological examination of the spinal cord revealed moderate to severe necrosis of the anterior horn cells.

In the present report the experiments of Sandler were repeated. Normal rabbits were selected, fasted for 24 hours, a blood specimen taken by cardiac puncture, and the animal then injected subcutaneously with 0.6 to 0.8 units of protamine insulin (Lilly) per kg of body weight. After approximately 2 hours, blood was again taken, and the animals were injected intracranially with 1.5 cc of a 10% suspension of monkey cord infected with the MV strain of poliomyelitis virus. If signs of hypoglycemia such as weakness, restlessness or convulsions developed, 10% glucose was injected intravenously. However, several animals died within 10 hours even though sufficient glucose had been administered to raise the level well over 100 mg %. Data on animals lost in this manner are not included in this report.

Seven animals survived for a satisfactory period and had suitable decreases in blood sugar levels. Four of the 7 developed signs of

* This work was aided by a grant from the Clara Ward Seabury Clinic for the Study of Infantile Paralysis.

1 Sandler, B. P., *Am. J. Path.*, 1941, **17**, 69.

2 Sandler, B. P., personal communication.

TABLE I.
Failure to Produce Neuronal Changes in Rabbits by Means of Intracranial Inoculation of MV Virus During Insulin Hypoglycemia.

Rabbit No.	Blood sugar, mg%	Signs of hypoglycemia	Cause of death	Survival period
19*	60	+	Hypoglycemia	18 hr
38†	49	+	"	20 "
33†	60	+	"	48 "
42†	61	+	Sacrificed	72 "
25*	64	0	"	9 days
17*	63	0	"	9 "
62†	62	0	"	30 "

*Mixed breed.
†New Zealand white breed, males.

hypoglycemia (Table I). No neurological signs of anterior horn damage appeared, and histological studies of the brain and spinal cord revealed no neuronal injury or other evidence of CNS lesions.†

Summary. The intracranial inoculation of rabbits with the MV strain of poliomyelitis virus during insulin hypoglycemia did not produce neurological or pathological signs of anterior horn damage. These findings lend no confirmation to the theory of Sandler that hypoglycemia decreases the cellular oxidation of the central nervous system tissues, thus lowering their susceptibility to neuronal damage by the poliomyelitis virus.

13264

Isolation of Virus from Feces of Mice Receiving Intranasal Inoculations of Epidemic Influenza Virus.

JOHN B. SARRACINO AND MALCOLM H. SOULE.

From the Hygienic Laboratory, University of Michigan, Ann Arbor.

Epidemic influenza virus has been recovered from the lungs of experimental animals injected intraperitoneally with large amounts of virus.[1] No account of the recovery of the virus from feces has been found in the literature. Accordingly, experiments were outlined to detect the presence of the PR8 strain of influenza virus in the feces of infected animals. At the outset it was essential to develop a method for the treatment of the feces which would destroy

† We are indebted to Dr. Konstantin Scharenberg for the histological examination of these tissues.

1 Rickard, E. R., and Francis, T., Jr., *J. Exp. Med.*, 1938, **67**, 953.