6989 P

A Note on the Chemotherapy of Experimental Poliomyelitis of Monkeys.*

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Probably because only monkeys are known to be susceptible to the virus of poliomyelitis among the lower animals, little thought and attention have been given to chemotherapeutic investigations in this disease on account of the expense involved. Furthermore the results of medicinal treatment of acute anterior poliomyelitis in human beings have yielded no interesting or encouraging "leads" or suggestions for chemotherapeutic investigations and individual case reports have so far failed to receive confirmation. Young, Hill and Scott[1] have recorded alleged benefit in the treatment of 2 cases of poliomyelitis with mercurochrome by intravenous injection, but this compound given to other cases under the personal observation of one of us did not result in any curative activity.

Having on hand a number of *Macaccus rhesus* monkeys which had been used for experiments on vaccination against poliomyelitis[2] but which developed the disease following intracerebral inoculation with the virus as a test for any possible acquired immunity, we have thought it worth while to institute treatment with a variety of chemical compounds as soon as definite signs and symptoms of poliomyelitis developed which terminated the experiments on vaccination. Under these conditions the experimental disease was allowed to produce pronounced paralysis before the drugs were given by intravenous injection but we thought it worth while to use the animals in these therapeutic tests with the hope that some encouraging results for further work in this field may be secured.

The compounds selected, the doses per kilogram of weight and the number given each animal are shown in the accompanying table. All compounds were administered by intravenous injection every 3 days for 5 to 8 injections. Each compound was given to 1 or 2 animals. As previously stated all of the monkeys showed unmistakable paralysis developing 6 to 9 days after intracerebral inoculation with virus before treatment began.

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[1] Young, Hill and Scott, *Arch. Surg.*, 1925, **80**, 813.

[2] Kolmer, J. A., and Rule, A. M., *J. Immunol.*, in press.

TABLE I
Ineffectiveness of following compounds in experimental poliomyelitis of monkeys.

Compounds	No. Treated	Dose per Kilo*	No. Doses	Results
Hexamethylamine	2	.025 gm.	8	died
Metaphen	2	.0005 "	8	"
Mercurochrome	1	.005 "	6	"
Pregl's solution of iodine	2	.5 cc.	8	"
Sodium and gold thiosulphate	1	.0003 gm.	7	killed
Sodium ricinoleate	2	.005 "	5	"
Neoarsphenamin	1	.02 "	8	died
Neutral acriflavin	1	.010 "	6	"
Tryparsamide	2	.03 "	6	"
Bismuth Potass. Tartrate	1	.002 "	5	killed

*By intravenous injection every 3 days; treatment begun upon first signs of poliomyelitis following intracerebral inoculation with virus 6 to 9 days previously.

In every instance paralysis progressed under treatment; 7 succumbed to the disease while the remaining 3 were chloroformed when it was apparent that treatment was ineffective.

6990 C

Concerning the Absorption of Unsplit Protein Through the Lacteals.*

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It is commonly assumed that allergic responses to ingested foods are due to the passage of unchanged protein from the bowel into the blood. Alvarez, who had seen a few cases in which unfavorable reactions to food appeared to be worse when patients ate much fat with the offending protein, suggested to me that an unguarded pathway might be through the lacteals and thoracic duct into the blood stream. In this way the protein would escape possible change in the intestinal wall and in the liver. Somewhat in favor of this view is the reported observation that food reactions are less likely to occur if the patient first takes a dose of hydrocarbon oil; theoretically, it might block the lacteals.

A number of investigations, inspired by the contributions of Uhlenhuth,[1] and of Hamberger and Sperk,[2] attempted to identify

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1 Ascoli, M., and Vigano, L., *Z. f. physiol. Chem.*, 1903, **39**, 283.