Recent Experimental Work on the Pathogenesis of Multiple Sclerosis. (*Journ. Amer. Med. Assoc., vol. cvi, p. 2117, June 20, 1936.*) *Brickner, R. M.*

The author makes a summary and comparison of recent experimental work on multiple sclerosis. In recent times the toxæmia point of view has gained support from several sources. The term "toxæmia" is used to refer to the action of any myelinolytic substances on the myelin sheaths. Hallervorden and Spatz (1928) described cases in which the predominant lesions were perivascular concentric rings of demyelinization which suggested as a cause, diffusion of a demyelinizing agent from a central point.

Testing of toxæmia theory has been carried out by the attempt to produce demyelinization in animals by the injection of toxins, and also by efforts to identify a toxin the presence of which is suspected. Ferraro has produced multiple areas of demyelinization by the injection of small doses of potassium cyanide into monkeys and cats. Some of these lesions had the concentric appearance noticed by Hallervorden. It is noted here that cyanide is a part of our intermediary metabolism. Putnam and his collaborators have produced patchy demyelinization in dogs by the employment of tetanus toxin, carbon monoxide and the injection of cod-liver oil emulsion, resulting in the formation of emboli. The presence of similar emboli or thrombi in individuals dying with multiple sclerosis is also attested by these workers.

Rivers and Schwentker have produced patchy demyelinization by the frequently repeated intra-muscular injections, over a long period, of an alcoholic extract of brain lipoids.

It is noted that in all these experiments pictures resembling multiple sclerosis have been produced, but the disease itself has not. The studies of Cone, Russel and Horwood into the role of lead-poisoning in multiple sclerosis yielded some evidence that this may be a factor.

The author conducted experiments in search of a lipolytic agent in the blood of patients with multiple sclerosis by the immersion of the spinal cord of rats in plasma from these patients. The results showed that this plasma had a demyelinizing action not present in controls.

These observations were confirmed by Weil and Cleveland employing serum instead of plasma. It was never possible, however, to obtain results diagnosable microscopically as multiple sclerosis.

Weil and Crandall have obtained demyelinization in the nervous system by ligation of the pancreatic and cystic ducts in dogs, and a demyelinating substance was also demonstrated in the blood. This substance was unaffected by heat, and a similar substance was demonstrated in the urine of patients with multiple sclerosis, and by Karady in the duodenal contents.

Sachs and Steiner have announced the discovery of a complement-fixation test for multiple sclerosis which is comparable to the Wassermann reaction.

The most recent phase of work on lipolytic action has been concerned with changes occurring in the blood during remission of the disease, and it is probable that a different lipolytic agent. enzymic in character and an esterase, is present. The serum esterase is high during remission and low during the active stage of the disease.

Quinine hydrochloride stimulates the action of this esterase.

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Cerebral Control of Fat Metabolism. (*Orvosi Hetilap., vol. lxxx, pp. 336–8, 1936.*) *Molnar, I., and Marsovszky, P.*

Experiments with hares showed that an increase of cerebral pressure diminished the fat content of liver, kidneys and musculature. The increase of cerebral pressure was effected by intra-cisternal injections of kaolin suspensions. The observed changes in fat content were not caused by insufficient nutrition.

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