

intervals of about two minutes, produces only in some cases an increase in the blood sugar.

3. Ligation of all branches of the hepatic artery running to the liver produces no change in the sugar content of the blood.

4. Stimulation of the tissues adjacent to the portal vein — after doubly ligating and cutting — causes hyperglycemia in about fifty per cent. of the cases. Such stimulation does not usually cause any change in arterial blood pressure.

As a result of these four groups of experiments it would appear that local asphyxia of the hepatic lobule consequent upon changes in blood supply is a less likely explanation of hyperglycogenolysis than is the hypothesis which assumes the presence of glycogenolytic secretory fibers in the great splanchnic nerves.

If such fibers control the production of glycogenolytic ferment by the liver, we might expect atropin to paralyze the fibers. My observations in this direction, however, show that stimulation of the N. splanchnicus in atropinized dogs causes the usual hyperglycemia. Atropin itself, however, produces a hyperglycemia which makes the observation of doubtful value.

Experiments are in progress to determine by the method of Bang, Lyungdahl and Bohm<sup>1</sup> whether there is an increased amount of glycogenolytic ferment in the liver after stimulation of the great splanchnic nerve.

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### **Prevention of syphilis in *Macacus Rhesus* by atoxyl.**

By **SIMON FLEXNER.**

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The drug atoxyl has been employed successfully in causing the rapid disappearance of syphilitic lesions in human beings and in preventing the development of the specific inoculation eye lesions in rabbits. Metchnikoff reported recently that the same drug would prevent the development of the specific lesion in monkeys even if administered some days after the inoculation.

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<sup>1</sup> *Beiträge zur chemischen Physiologie und Pathologie*, 1907, ix, p. 408 ; x, p. 1 ; x, p. 312.

The experiment which I wish to report was made upon five Macac monkeys. They were all inoculated over both eyebrows with scrapings from a syphilitic papule of the tongue which had been shown by the dark field illumination microscope to be rich in living *Spirocheta pallida*. Two of the monkeys remained as controls and the other three received 0.15 gram atoxyl subcutaneously, one day, eight days and fifteen days respectively after the inoculation. Only the two controls developed specific lesions. Three months later two of the atoxyl-treated monkeys (one having died in the interim) and another control monkey were inoculated over the right eyebrow with virus from a primary syphilitic lesion rich in *Spirocheta pallida*. Within three weeks one of the atoxyl-treated monkeys and the control monkey developed specific lesions, showing the pallida, over the right eyebrow.

This experiment is an example of the power which atoxyl possesses of suppressing the development of syphilitic lesions when given as late as fifteen days after inoculation with active virus, and indicates that this suppression is not attended with the production of a state of immunity to the virus.

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**Further notes on a rat tumor.**

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On several occasions we reported to this Society upon a rat tumor that has been transplanted for more than two years. The tumor was originally described as a sarcoma. In a recent report<sup>1</sup> we described its transformation into a malignant adenoma. This change in histological structure was attended with the acquisition of the property, hitherto absent, of producing metastasis in lymphatic glands. The reverse changes, namely from the adenoma into sarcoma, had been noted by Ehrlich, Leo Loeb and others in mouse cancers and were attributed to gradual or rapid proliferation and predominance of the stroma of the tumors or of a corresponding tissue derived from the host. Since our rat tumor

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<sup>1</sup>This journal, 1908, v, p. 52.