coma and died on the 11th day after admission. Gross and histological findings of the brain showed hemorrhagic softening of both dentate nuclei and a generalized perivascular infiltration in many parts of the brain and demyelinization of the white matter of the cortex. Both areas of softening and perivascular infiltration were noted in the basal ganglia.

These last 2 cases we believe illustrate conditions which must be present but which do not guarantee the findings of a Herpes virus. Brain material, preferably from the region of the basal ganglia, should be used for inoculation; the cases should be in the acute febrile stage, either of the initial acute encephalitis or of an exacerbation of the disease in a chronic case.

We have failed to find Herpes virus in a case of encephalitis following mumps, in another following whooping cough and in another very acute adult case—no one of which differed in any obvious way from these positive cases we have just discussed.

We wish to express our gratitude to Dr. Josephine B. Neal, who has furnished us with the histories and material for all the cases to which reference is made, and to Drs. Orton, Stevenson, and Wolff for the histopathological descriptions.

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Susceptibility of Diabetic Dogs to Tuberculosis.*

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(Introduced by Frederick P. Gay.)

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It has been believed for a long time that diabetes predisposes human beings to infections, including tuberculosis, and that when a diabetic individual develops tuberculosis the disease is extremely severe, and rapidly fatal. The incidence of tuberculosis is almost 3 times as great in diabetics as in the general population (Banyai[1]). Fitz[2] found that 63% of his cases died in less than one year(19 of 31 cases). While most writers agree on these findings (Gotten,[3]

* This work was made possible by a grant from the Josiah Macy, Jr., Foundation.

[1] Banyai, A. L., *Am. Rev. Tub.*, 1931, **24**, 650.

[2] Fitz, R., *Am. J. Med. Sci.*, 1930, **180**, 192.

[3] Gotten, H. B., *Am. Rev. Tub.*, 1931, **24**, 668.

Adams[4]), they have been disputed by many others. Joslin,[5] and Rosenberg and Wolf[6] feel that the prognosis is not bad in cases of tuberculosis complicated by diabetes. It is, therefore, obvious that much confusion prevails concerning the increased susceptibility of the diabetic to tuberculosis. It was hoped that animal experimentation might help to clarify this problem.

Our experiments include first a study of tuberculosis in the diabetic dog. A series of dogs was pancreatectomized. Eight survived, 7 of which developed severe diabetes while one had only a slight increase in blood sugar. As noted on the accompanying chart the animals survived from 16 to 95 days after infection. The average blood sugar is also included in the chart. Small doses of insulin were given daily to offset coma, and at the same time the animals remained severely diabetic. To replace some of the enzymes of which pancreatectomized animals are deprived, we fed them daily with desiccated pancreatic substance incorporated in their food. Twenty-four hours after operation, the animals were injected intraperitoneally with a human strain of tubercle bacillus (H37) in doses of 1 mg. per pound of weight.

As recorded on the chart, 7 of the 8 animals were macroscopically positive for tuberculosis (87.5%). In 6 of the 7 that have so far been checked histologically the diagnosis was confirmed, while one animal was negative both macroscopically and microscopically. The lungs were more severely involved than the retroperitoneal glands or the liver. The spleen was never found positive on microscopic study. Caseation was encountered only once (in the retroperitoneal glands), and the tuberculosis was never severe. This may be due to the high natural resistance of the animal, or to the fact that the strain of infecting bacilli, which we have kept in our laboratory for the past 10 years, appears to have lost a good deal of its original virulence.

The experiment was controlled with 8 animals. These were laparotomized and closed without interference with the pancreas and infected 24 hours after operation with the same strain (H37), by the same route, and with the same dose. It suffices to say that although 4 of the controls were questionably positive macroscopically, they were all negative on microscopic examination.

In conclusion it appears that the dog is highly refractory to this

[4] Adams, S. F., *Arch. Int. Med.*, 1927, **37**, 861.

[5] Joslin, E. P., Treatment of Diabetes Mellitus. Lea & Febiger, Phila., 1928.

[6] Rosenberg, N., and Wolf, G., *Klin. Wnschr.*, 1927, **1**, 1935.

TABLE I.
Experimental Results on Susceptibility of Diabetic Dogs to Tuberculosis.

Diabetic Animals Pancreatectomized and Infected							Control Animals Laparotomized and Infected				
			Macro.Path.		Micro.Path.						
No.	No. days surviving infection	Aver. blood sugar: mg./100cc.	— or +	Organs involved	— or +	Organs involved	No.	Died or killed	No. days surviving infection	Macro. — or +	Micro. — or +
1	16	130	+	Liver	+	Lungs, liver, mesent. lym. nodes	1	D	9	Lung, liver and spleen?	—
2	29	292	+	Lungs, liver, spleen	+	Lungs,	2	D	13	—	—
3	31	312	+	Lungs, liv., spl., retroper. glands	+	Lungs, retroper. glands	3	D	21	Liver and kidney?	—
4	34	338	—		—		4	K	31	Liver?	—
5	42	349	+	Lungs, liver	+	Liver, retroper. glands	5	D	37	—	—
6	46	314	+	Lungs, liver, spleen			6	K	42	Kidney and liver?	—
7	47	365	+	Lungs, liver	+	Lungs, liver	7	K	46	—	—
8	95	399	+	Lungs, liv., spl., mes. and retro. gl.	+	Liver, retroper. glands	8	K	95	—	—

(All animals infected with H37, intraperitoneally, 1 mg. per lb. weight.)

particular human strain of tubercle bacillus (H37), but becomes definitely susceptible when rendered severely diabetic by pancreatectomy.

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II. Experiments on Immunization with Haptens.

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Experiments were previously reported[1] confirming the findings of Gonzalez and Armangué that the antigenic properties of alcoholic

[1] Landsteiner, K., and Jacobs, J., PROC. SOC. EXP. BIOL. AND MED., 1932, **29**, 570.