assumes a purplish tinge when the skin is cut. A considerable amount of epinephrine is also lost when it remains for some time under the skin of dead animals, as is shown in Table I. This indi-

TABLE I.

Recovery of epinephrine from subcutaneous tissue of rat.

0.1 mg. was injected in each case. The values are expressed in % of the amount injected.

	Immediately	Ave.	After 1 hour	Ave.	After 2 hours	Ave.	After 3 hours	Ave.
Dead rats	79; 83; 80; 78	80	65; 60; 61	62	38; 45; 40	41		
Live rats	78; 84; 81; 80	81	47; 51; 43; 58	50	32; 49; 20; 25	32	17; 18; 9; 16; 11	14

cates that the disappearance of epinephrine from the subcutaneous tissue of live animals depends not only on absorption into the blood stream but also on local destruction. The data of Table I can therefore not be used for a calculation of the rate of absorption of epinephrine into the blood stream. It is concluded from the experiments in Table I that epinephrine is still present in the subcutaneous tissue 3 hours after the injection and this direct chemical evidence agrees well with the observation that the hyperglycemia persists for at least 3 hours.

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The Influence of Constant Intravenous Injection of Epinephrine on Blood Sugar of Rats.

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The aim of the present experiments was a determination of the minimal rate of injection of epinephrine which causes a rise in blood sugar of rats. Similar experiments on rabbits could be made without anesthesia but this was unfortunately not possible on rats. Though amytal anesthesia has little effect on the blood sugar of rats, as may be seen in Table I, it undoubtedly lowers the carbohydrate tolerance. Animals in the postabsorptive state with a high liver glycogen content on account of previous carbohydrate feeding were used. 0.1 to 0.2 cc. of blood was withdrawn from a femoral vein by means of a syringe at the beginning and end of the injection. A

third sample was taken 30 minutes after the end of the injection. The temperature of the animals was kept between 97.8 and 99° F., heat being supplied by an electric bulb. The dilutions of epinephrine were made with a slightly acidified physiological salt solution and as a control the same salt solution was injected. The injections of epinephrine lasted for 30 minutes. It is concluded from the data of Table I that 0.0002 mg. per kilo per minute is the smallest rate of injection which produces a definite rise in blood sugar of amytalized rats.

TABLE I.

Influence of epinephrine on blood sugar of rats under amytal anesthesia.

Weight	cc. injected in 30 min.	mg. epinephrine per kilo per minute	Blood sugar (in mg. per 100 cc.)		
			Before injection	After injection	30 min. later
176	1.46	0.001	116	171	155
170	1.43	0.001	122	178	141
173	1.21	0.0005	128	192	
206	1.50	0.0004	108	155	121
205	1.56	0.0004	119	173	123
220	1.88	0.0003	108	163	
203	0.96	0.00025	111	134	119
188	1.59	0.0002	117	128	110
184	1.13	0.0002	114	147	124
194	0.63	0.00015	122	109	114
200	1.14	0.0001	114	115	
223	1.54	0.0001	112	114	104
212	1.50	salt solution	113	105	109

TABLE II.

Response of different species to constant intravenous injection of epinephrine.

Species	Minimal pressoric rate	Minimal hyperglycemic rate
	mg. epinephrine per kilo per minute	
Man	0.00005	0.000025
Rabbit*	0.0006	0.00005
Rat†	0.001	0.0002

* Unanesthetized. † Under amytal anesthesia.

The relation between the pressoric and hyperglycemic rate of epinephrine injection in different species, as determined recently in this laboratory, is shown in Table II. This comparison shows that the carbohydrate metabolism and the vascular system of the rat is decidedly less sensitive to epinephrine than that of the other species investigated.