was from 12 to 18 mm. Hg and lasted for 40 minutes. The heart rate usually showed a slight increase after both the epinephrine and the control solutions, the increase not exceeding 10 beats per minute. In contrast, the glomerular kidney responded with a tremendous polyuria (as great as 500× the control rate) to doses of epinephrine that ranged from 242 to 3 micrograms per 100 gm. body weight. The larger increases in urinary rate after epinephrine occurred in those experiments in which the control rate was low. A significant and prolonged rise in the blood pressure occurred after the epinephrine, the rise in one experiment being from 6 to 14 mm. Hg and lasting for approximately 80 minutes. The heart rate fell markedly after the injection of the drug (from 120 to 60 beats per minute in the above experiment) but gradually rose to the control level. This fall in heart rate was interpreted by Lutz[5] as the response of an unbalanced parasympathetic mechanism in an organ lacking a sympathetic accelerator innervation. Control injections did not change the heart rate or blood pressure significantly.

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On the Nature of Insulin Convulsions.

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With the injection of doses of insulin just adequate to induce convulsions, the animal will usually exhibit only clonic convulsions, but if twice that dosage be given to this same animal, tetanic convulsions usually result. If the low sugar level be the sole cause of the convulsions, then why would their character change with the increased dose? With doses of insulin adequate to render an animal convulsive, or even sub-convulsive, animals, as well as man, exhibit evidence of a functionally modified nervous system long before the true blood sugar level has fallen to zero and before any convulsions appear. Some of the evidences of this functional modification are marked salivation, profuse sweating, pupillary changes (dilation just before the onset of the convulsions and hippus often accompanied by nystagmus during the seizure), marked restlessness and increased sensitivity to stimuli such as light or noise.

[5] Lutz, B. R., *Am. J. Physiol.*, 1930, **94**, 135.

The barbiturates are being employed both clinically as well as experimentally for the prevention of insulin convulsions. In a search for the mechanism for this action of the barbiturates, the question arose as to whether the drug modified the effect of a convulsive dose of insulin upon the blood sugar. Table I summarizes some of the results.

TABLE I.*

Blood Sugar Levels after Insulin (2 units per kilo) Plus Phenobarbital (40 mg./kg.) Injected Simultaneously. Normal-fed Rabbits.

		Time after injection in hours			
Rabbit No.	Initial mg.	½ mg.	1 mg.	2 mg.	3 mg.
444	79	27	27	14	27
452	112	40	40	16	28
451	97	59	16	10	16
1124	120	71	14	14	92

* Somogyi's Reagent No. 1 for the micro method was used. Peters and Van Slyke, *Quantitative Clinical Chemistry*, Vol. II, p. 466 (Somogyi, M.), Williams and Wilkins, Baltimore, 1935. All values below 27 mg. were obtained by extrapolation.

The resulting values approximate the true sugar level of zero as found by Dotti.[1] In no case, however, were there any signs either of convulsions, or even of the prodromal signs. Although no glucose was administered, none of these animals died despite the fact that the true blood sugar levels remained at or near the zero level for at least the 3-hour observation period. Apparently, the protective mechanism of the barbiturate is independent of the blood sugar concentration. This would seem to indicate either that the drug so affects the central nervous system that it does not respond to the low sugar concentration in the usual manner, or that insulin has some effect on the central nervous system which is unrelated or, at most, related only indirectly to the low sugar level.

[1] Dotti, L. B., *J. Biol. Chem.*, **1934, 104, 535.**