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Response of Experimental Hypertension to a Rice and Fruit Juice Diet.

GEORGE F. DICK AND WILLIAM B. SCHWARTZ.
From the Department of Medicine, University of Chicago.

Dietary treatment of hypertension has received the attention of many investigators. Fishberg[1] in summarizing the evidence concluded that "No dietary treatment is known which has a specifically favorable effect on essential hypertension." More recently Kempner[2] has reported striking therapeutic results in a majority of patients with both "primary" and "secondary" hypertension by the use of a diet of rice, fruit, and fruit juices. Grellman, Harrison, and co-workers[3] have suggested that rigid sodium restriction is responsible for the changes observed by Kempner.

It is the purpose of this communication to record preliminary studies on 12 hypertensive dogs kept for 8 weeks on the Kempner regime. The hypertension in these animals had been produced by a nephrosclerosis which followed the intravenous administration of streptococci, as already reported by Dick.[4] At the time the present studies began, hypertension had been maintained for 2 to 4 years. Blood pressure was determined monthly throughout this period by direct arterial puncture with kymographic recordings through a mercury manometer.

Prior to the institution of the "rice" diet the animals had been fed on meat and dog biscuits provided approximately 900 calories per day. The basic experimental diet consisted of 170 g of rice, 340 cc of fruit juice, and 60 g of sugar. It contained approximately 13 g of protein and 212 g of carbohydrate with a total value of 900 calories. Daily supplements of 6000 I.U. of vitamin A, 1600 I.U. of vitamin D, 25 mg of nicotinamide, 1.6 mg of thiamine chloride, and .45 g of ferrous sulfate were administered. Since the animals often refused part of the diet offered, the basic diet represents the maximum possible intake. One dog refused the diet entirely and died before any significant observations were made.

TABLE I.
Blood Pressure Before and After Kempner Diets.

Dog No.	Mean arterial pressure, mm Hg.				Weight		
	Before induction of hypertension	Before beginning diet	After 8 wk of diet	Change	Initial, kg	Final, kg	% change
1.	105	220	138	—82	12.8	10.4	—18.7
2.	120	200	132	—88	16.4	14.2	—13.4
3.	132	210	154	—56	12.6	9.4	—22.2
4.	120	192	152	—40	12.4	8.6	—30.6
5.	120	168	126	—42	13.5	9.8	—27.4
6.	120	165	142	—23	10.2	9.3	— 8.1
7.	130	162	144	—18	12.4	8.6	—30.6
8.	120	160	124	—36	10.3	8.2	—18.4
9.	130	160	120	—40	8.2	5.8	—29.2
10.	120	150	148	— 2	10.2	9.3	— 8.9
11.	110	140	120	—20	9.4	9.7	+ 3.1
Avg	120.3	181.6	138	—43.6	12.1	9.7	—19.7

1 Fishberg, A. M., *Hypertension and Nephritis*, p. 704, Lea and Febiger, Philadelphia, 1939.

2 Kempner, W., *North Carolina M. J.*, 1945, **6**, 61; *ibid.*, 1945, **6**, 117.

3 Grollman, A., Harrison, T. R., Mason, M. F., Baxter, J., Crampton, J., and Reichmann, F., *J. A. M. A.*, 1945, **129**, 533.

4 Dick, G. F., *Arch. Path.*, 1945, **39**, 81.

As noted in Table I a significant reduction in arterial pressure occurred in 10 of the 11 dogs. The greatest fall in pressure was found in the animals with the highest initial levels. Although weight loss was evident, no direct quantitative correlation could be established between the amount of weight loss and the extent of fall in blood pressure.

The average blood nonprotein nitrogen was 23.6 mg per 100 cc before the diet and after 8 weeks had decreased to 18 mg per 100 cc. The average total plasma protein was 6.20 g per 100 cc initially and 6.22 g per 100 cc after 8 weeks.

It appears that the Kempner regime is capable of causing significant lowering of the arterial blood pressure of dogs made hypertensive through the induction of nephrosclerosis. The role of weight loss, salt restriction, and nitrogen balance in this result requires further study.

Summary. In 11 dogs with experimental hypertension the blood pressure fell in every animal following Kempner diets.

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Hypoglycemic Effect of Intraspinal Glucose Injection.

LUIGI MARINELLI AND VALENTINO GIUNTI. (Introduced by Harry Sobotka.)
From the Instituto di Clinica Medica Generale e Terapia Medica, University of Perugia, Italy.

We wish to report our experimental observations on variations in the glucose content of the blood produced by the introduction of glucose into the spinal fluid.

In diabetes the level of glucose in the spinal fluid is subject to considerable variation. Moreover some regions in the brain can be supplied with nutritive substances only through the cerebrospinal fluid. Not only may nutritional influences be exerted but stimulation of the central nervous mechanism may also take place, through variation of the chemical composition of the fluid.

The first series of experiments were performed on dogs. Blood and spinal fluid glucose levels were determined on the fasting animal and 0.1 g glucose was then injected directly into the "cisterna magna" after an equivalent amount of spinal fluid had been removed. The concentration of the injected glucose solution was calculated to increase the glucose content of the fluid to approximately twice the initial values. Blood and spinal fluid sugar levels were determined at frequent intervals by the Hagedorn-Jensen[1] method, since this requires only 0.1 ml of material. In previous control experiments it was established that (1) the blood glucose content of normal dogs showed practically no variation (± 3 —8 mg %); (2) neither the suboccipital tap alone, nor the introduction of isotonic salt solution, influenced the level of the blood sugar; (3) introduction into the spinal space of redistilled water had no effect upon the blood glucose level. This last control test was made in order to eliminate the possibility that the introduction of the glucose solution would produce an effect upon the nervous center by changing the ionic equilibrium.

After the introduction of the glucose solution, there was a steep rise in the level of the spinal fluid glucose followed by a rapid fall to the base level. The blood sugar reached its lowest level in 15 minutes and returned to normal after 2 hours (Table I).

The experiments were repeated in the same way in man. After fasting for 12 hours, 0.2 g of glucose was introduced into the spinal cavity to approximately double the initial level of glucose. Controls similar to those on the dog were repeated on man with the same negative results. Table II shows the results of these experiments.

[1] Hagedorn, H. C., and Jensen, B. N., *Biochem. Z.*, 1923, **137**, 92.