upset by epinephrine. May we not have a mechanism analogous to secretin which controls the glycogenic function?

The subjects for our sugar experiments were healthy students (men and women). All were familiar with the technique of venapuncture, both as operators and as subjects. The blood samples were taken from an arm vein by skilled operators, and there was not the slightest ground for suspecting psychic effects on blood sugar in any of the experiments reported here.

ABSTRACTS OF THE COMMUNICATIONS.

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The diagnostic value of phosphate metabolism in experimental rickets.

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These results are based on a study of 150 rats, taken from the mothers at the age of three weeks when they weighed about 30 gm. and placed on experimental diets in small separate cages shielded from ultraviolet rays of sunlight. They were not given cod-liver oil or butter fat, but they had sufficient vitamine-A for varying degrees of growth. All of them were x-rayed at the end of this period and turned over to Dr. C. M. Jackson for morphological study. His observations have confirmed my diagnoses.

In order to diagnose rickets in rats the average P metabolism and growth per day were determined (weights being expressed in milligrams) and the following empirical formula was used. Rachitic index = *RI*.

RI = increase in body weight $\times$ 0.0022 $-$ (P retention $-$ 2).

If *RI* is positive the rat has rickets and if negative the rat is normal. For example, if a rat increases in weight 2,000 mg. per day, 2,000 × 0.0022 = 4.4. If its P retention is 4 mg. per day, 4 − 2 = 2 and *RI* = 4.4 − 2 = 2.4, therefore, this rat has rickets. If the P retention is 7 mg. *RI* = — 0.6 and this rat is normal The magnitude of *RI*, if it is positive, denotes the severity of rickets provided the metabolism has been determined over long enough periods and with sufficient accuracy, but the skeleton grows on a "maintenance" diet and this tends to disturb the applicability of the formula. Rats usually retain $\frac{1}{5}$ to $\frac{1}{3}$ of the P of the diet. Since a rat's body contains about 0.5 per cent. P and the normal rate of growth is about 2 grams per day they should retain about 10 mg. P per day or 5 mg. per gm. increase in body weight, and this is found in normal rats of the age studied. As pointed out by McCollum, the rate of growth influences the degree of rickets. I found that during loss of weight, 0.12 per cent. P in the diet prevented rickets whereas during normal growth 0.4 per cent. in the diet may be insufficient to prevent rickets. Every growth promoting substance has a rachitic influence when it promotes growth, therefore, every antirachitic substance that promotes growth may have two opposing actions. In my rachitic rats *RI* varies from 0 to 4 and in my normal rats *RI* varies from − 1 to − 5 due to storage of P in excess of the "minimum" requirement. The use of *RI* in diagnosing rickets has the advantage that it can be done without killing or injuring the animal.

The Ca retention is low in rickets as well as in osteoporosis. If the P intake is lowered the P retention is lowered and the Ca retention is lowered (rickets). If the Ca intake is lowered the Ca retention is lowered but the P retention remains normal (osteoporosis). This was found true for levels of P content of the diet varying from 0.2 per cent. to 0.4 per cent. The Ca content was reduced from 0.8 per cent. in the diet to 0.2 per cent. and the Ca retention became less than $\frac{1}{2}$ the normal. At the end of a month on this diet, the thickness of the bones and the thickness of their walls was reduced. The total volume of the wall substance of the shafts of the long bones was reduced to 53.7 per cent. of the normal.