in the force of the contractions which was sometimes produced by small amounts of mercury and subnormal amounts of calcium never occurred after treatment with the same concentration of mercury when the calcium content was increased beyond 0.012%. The difference in the effect was even more pronounced in the experiments in which the calcium chloride content was increased to 0.02%. In one experiment the heart, though weak, was still contracting 50 minutes after treatment with mercury 1:100,000. The same amount of mercury in the other experiments produced considerable slowing, but no change in the strength of the contractions could be noticed, and at the end of 21 hours feeble beats were still observed. The antagonistic effect of calcium was less pronounced, however, when the concentration of mercuric chloride was increased. Solutions of 1:50,000 were almost equally potent when the amount of calcium chloride was 0.012 or 0.2%. Heart action continued 15 to 20 minutes, and in one experiment with mercury and normal Ringer the heart was still beating 45 minutes after the introduction of mercury. But when the amount of calcium was subnormal mercury 1:50,000 promptly brought the heart to a standstill.

The results presented above justify the conclusion that deficient amounts of calcium greatly increase the resistance of the frog heart to mercury. While excess calcium may be antagonistic to mercury, this is less certain than the injurious effects produced when the heart is exposed to mercury in the presence of subnormal amounts of calcium.

5009

Vitamin Requirements of Nursing Young. VIII. Effect of Vitamin B* Deficiency on Alkaline Reserve of Blood of Nursing Young of Albino Rat.†

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In order to determine the influence of uncomplicated vitamin B deficiency on the alkaline reserve of the blood of nursing young of

* Unless stated otherwise, vitamin B deficiency refers to the uncomplicated polyneuritis produced by a deficiency of only the antineuritic factor. An abundance of the antipellagric factor was provided by an ample supply of autoclaved yeast in the maternal diet.

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the albino rat, 84 nursing young, whose mothers received stock diet No. 6‡ were sacrificed during the later part of the lactation period, and the carbon dioxide volume per cent determined on blood secured from the carotid artery. We used the manometric method of Van Slyke and Neill[2] on samples of 0.1 cc. of plasma.

Since in the pathological nursling there has been, because of stunting of growth, a considerable prolongation of the nursing period, 6 additional animals on maternal stock diet No. 6, 32 days of age, were taken during the post-lactation period. The range of carbon dioxide volume per cent found was 37 to 65.

The 72 pathological animals examined were in a condition of prolonged maintenance, or in a condition of prolonged maintenance accompanied by incipient polyneuritis. The avitaminosis of the nurslings was produced on maternal diet 1438,[3] adequate in every respect with the exception of vitamin B. Only 4 pathological animals showed a deviation from the normal which occurred during the last stages of polyneuritis. These results are in agreement with the findings of Sure and Smith[4] on growing and adult rats.

5010

Vitamin Requirements of Nursing Young. IX. Effect of Vitamin B Deficiency on Glycogen Content of Liver of Nursing Young of Albino Rat.*

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In this study we have employed 50 nurslings, 29 control and 21 pathological. The animals were sacrificed by cutting both carotid arteries with a sharp scissors. The vagus nerves were also severed and death occurred almost instantaneously. The chemical method used for determination of glycogen of the livers was one kindly furnished us by Dr. C. F. Cori, which is a modification of the

‡ Stock diet No. 6 is a modification of our stock diet No. 1 (1) in which 5% of the corn is replaced by an equivalent amount of rice polishings.

1 Sure, B., *J. Biol. Chem.*, 1926, lxix, 65.

2 Van Slyke, D. D., and Neill, J. M., *J. Biol. Chem.*, 1924, lxi, 523.

3 Sure, B., and Smith, M. E., *J. Nutr.*, 1929, i, 537.

4 Sure, B., and Smith, M. E., *J. Biol. Chem.*, 1929, lxxxiv, 727.

* Research paper No. 143, Journal series, University of Arkansas.