trical stimulation of either the hypothalamus or the cruciate cortex, other than that in the former case the animals which received more than 60 mg of morphine sulphate would cease their pseudo-affective behavior and relapse into stupor immediately after the cessation of the hypothalamic stimulus.

Conclusion. In the cat, the local injection of from 1 to 10 mg of morphine sulphate in Locke's solution or the intraperitoneal administration of from 30 to 100 mg of the drug does not affect the reactivity of either the cruciate cortex or the hypothalamus to faradic stimulation, except that in animals which receive more than 60 mg intraperitoneally the hypothalamic responses are limited to the duration of the stimulus.

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Effect of Sulfanilamide on the Isolated Frog Heart.

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A study was made of the effects of sulfanilamide on the isolated frog heart using the Straub-Fuehner method. The solutions of sulfanilamide used ranged from 0.05% to 0.50% and were made up in frog heart Ringer's. This solution was used between sulfanilamide perfusions to return the heart to normal.

All concentrations caused an increase in amplitude, more marked in the higher concentrations. In the weaker dilutions, the rate was only slightly increased, followed by depression, whereas the stronger solutions all caused a depression of rate. Further observations were made with stronger solutions of sulfanilamide, up to 0.8%, essentially with the same results. The heart always stopped in diastole. This was not due to the weight of the lever, since it also stopped in diastole when wholly disconnected from the lever.

In order to determine if any part of the action of sulfanilamide was caused by a change in pH, both the Ringer's solution and the 0.5% solution of sulfanilamide in Ringer's were tested. The sulfanilamide raised the pH very little so that the solution was still within the pH range permissible for frog heart Ringer's. Therefore, it was not a change in pH which affected the heart.

The effect of sulfanilamide on the isolated frog heart is stimulation

followed by depression. The stimulation increases the amplitude. The effect is transient, however, and often lasts only 10 minutes although, in some cases, it persists as long as 20 or 30 minutes. The maximum increase occurs with a 0.25% solution. The rate may also be slightly increased but it is the amplitude that shows the greatest effect of stimulation. On the other hand, the secondary depression appears as a decreased rate. In the lower concentrations (up to 0.2%) this is not evident in the first 10 minutes but in higher concentrations, may set in sooner. The depressive effect increases with the concentration of sulfanilamide. A 0.5% solution of sulfanilamide causes a 50% to 100% decrease in rate within 10 minutes after it is introduced into the heart. While some hearts stopped within the first 10 minutes, others maintained a very slow rate with some irregularity for 40-60 minutes.

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Reduction in Experimental Rat Caries by Fluorine.

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Dental research in the field of caries prevention received new impetus when Hoppert, Webber and Canniff[1] experimentally produced dental caries in 90 to 100% of their rats by feeding them a diet, which although adequate nutritionally, consisted mainly of coarse corn particles. Their hypothesis was that the corn particles became impacted in the teeth and produced decay. Lilly[2] obtained a 90% reduction in the incidence of dental decay by substituting commercial casein for the powdered whole milk (20% of the Hoppert, Webber and Canniff diet). The same year, Hodge, Luce-Clausen and Brown[3] found that the commercial casein used by them was contaminated with fluorine (0.2%). It occurred to us that fluorine might be the factor producing the caries reduction in Lilly's experiments. Two major findings in the literature support this contention: (1) clinical observations by Dean and others[4] give evidence of a reduction of caries in areas of endemic fluorisis (mottled enam-

1 Hoppert, C. A., Webber, P. A., and Canniff, T. L., *Science*, 1931, **74**, 77.

2 Lilly, C. A., PROC. SOC. EXP. BIOL. AND MED., 1938, **38**, 398.

3 Hodge, H. C., Luce-Clausen, E. M., and Brown, E. F., *J. Nutrition*, 1939, **17**, 35.

4 Dean, H. T., *Public Health Rep.*, 1938, **53**, 1443.