gan to yield an increasing number of colonies which, when examined, indicated the presence of gas-producing organisms. There was a lack of correlation between changes in colony form and gas production. Colonies atypical of the inoculated variant might or might not yield a gas-producing culture. The same was true for the colonies of the type inoculated.

Conclusion. The evidence presented seems to be in accord with the concept that bacterial properties may vary independently of each other.

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A Reducing Substance in the Urine of Cats Under Nembutal Anesthesia.

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A series of 15 cats were given intraperitoneal injections of nembutal (sodium iso-amytal, penta-barbital), the average dose being 60 mg. per kilo. Urine samples were collected at intervals after the injection and later analyzed for reducing substances by Sumner's method.[1] In certain instances the results thus obtained were checked by the use of Benedict's quantitative reagent. Simultaneously blood samples were taken and later analyzed for "blood sugar" by the Randle-Grigg modification of the Folin-Wu Micro Method.[2] These observations were made in the course of experiments to determine the effect of stimulation of the *tuber cinereum* upon carbohydrate metabolism. Rather extensive surgical procedures under deep nembutal anesthesia were required in the experiments. The bladder was exposed and emptied and the urethra cannulated from one to 2 hours after the injection of nembutal. The first sample of urine was taken about an hour after the urethra was cannulated and from 2 to 3 hours after the administration of nembutal. This sample always showed the presence of an abnormal quantity of reducing substance, usually the maximum obtained during the experiment, the concentration decreasing progressively in subsequent samples. This maximum reduction where the lowest maximum was obtained, was equivalent to that produced by 240 mg. % of glucose. In the experiment where the largest maximum was found, the reducing sub-

[1] Sumner, *J. Biol. Chem.*, 1925, **65**, 383.

[2] Randles, F. S., and Grigg, W. K., *J. Am. Med. Assn.*, 1924, **82**, 684.

stance was equivalent to that produced by 1575 mg. % of glucose. An average maximum value, equivalent to that produced by 1045 mg. % of glucose, was obtained.

The amount of reducing substance in the urine was definitely related to the time interval following the injection of nembutal and not to the "blood sugar" concentration. Often in the later stages of the experiment the reducing substance would be decreasing in amount while the blood sugar was increasing.

In Cat C the blood sugar remained at approximately a constant level of 150 mg. % throughout the experiment. Two hours after the initial injection of nembutal, the urinary concentration of reducing substance was found to be 961 mg. %. Forty-five minutes later a second injection of nembutal was given equivalent to 10 mg. per kilo body weight. Another sample of urine was taken 45 minutes later and the concentration of reducing substance had risen to 1428 mg. %. The third urinary sample was taken an hour later and the concentration of reducing substance had fallen to 735 mg. %, falling to 657 and 471 mg. % in the following 2 intervals of an hour and an hour and 20 minutes respectively.

These results suggest that the reducing substance may be due to the injected nembutal. Swanson and Shonle[3] state that the exact mode of elimination of this drug is not known, that examination of the urine failed to reveal any trace of the drug administered. We have found that nembutal when added to normal urine does not reduce the standard reagents used for the detection of reducing substance in the urine. It is, of course, unlikely that nembutal increases the permeability of the kidney to sugar, and subsequent fermentations of urine of nembutalized cats failed to show the presence of more than a negligible amount of sugar. The possibility, however, is not excluded that the reducing substance in the urine may be a product of the break-down of nembutal in the body.

[3] Swanson, E. E., and Shonle, H. A., *J. Lab. and Clin. Med.*, 1931, **16**.