The technic of the method is as follows: To 8 c.c. of water in a 50 c.c. centrifuge tube is added 1 c.c. of the extract to be examined, and the solution warmed to just 40° C. in a water bath with thermostat attachment. One c.c. of one per cent. cane sugar is now added and incubation carried out for 30 minutes. The solution is rapidly cooled in cold water and 0.5 to 1.0 gram of dry picric acid added, thoroughly mixed, centrifuged and filtered. The inverted sugar is now estimated colorimetrically in 3 c.c. portions as already described.[1]

One must not let the sucrose stand for a long time in contact with the picric acid, owing to the hydrolytic action of the picric acid.[2] For this reason it might be of advantage to employ sodium picrate, as recently recommended by Benedict.[3]

Utilizing the above method we have been unable to satisfactorily demonstrate sucrase in human blood or that of rabbits. With one per cent. yeast extract there was 60 per cent. inversion in one-half hour, *i. e.*, 6 of the 10 mg. were inverted, while a one per cent. yeast extract in human blood gave a 54 per cent. inversion. With a 10 per cent. extract of the mucous membrane of rabbit's intestine figures of 13.2 and 12.8 per cent. were obtained. Controls were all negative.

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The phosphate and calcium content of serum in the condition of guanidine tetany.

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In the altered metabolism in parathyroidectomized dogs, Greenwald[4] found a marked decrease in the elimination of phosphate in the urine together with a distinct retention in the blood.

[1] Myers and Bailey, *Jour. Biol. Chem.*, 1916, XXIV, 147.

[2] Rose, these PROCEEDINGS, 1917, XX, 16.

[3] Benedict, *Jour. Biol. Chem.*, 1918, XXXIV, 203.

[4] Greenwald, I., *Am. Jour. Physiol.*, 1911, XXVIII, 103.
Greenwald, I., *J. Biol. Chem.*, 1913, XIV, 363; Ibid., 1913, XIV, 369.

It has been established that the lack of parathyroid is accompanied by increased elimination of ammonia, decreased acid excretion and the lowering of the hydrogen ion concentration. The same conditions are manifested after the administration of guanidine hydrochloride.[1] The increase in guanidine nitrogen was found by Koch[2] and Burns and Sharpe[3] in experimental parathyroid tetany and also in idiopathic tetany. In a recent communication[4] we reported that the above-mentioned phenomena were an accompaniment of the acidosis produced by guanidine administration and also suggested that phosphates might be retained in the body to neutralize the acids formed by the muscular activity.

Another fact that in parathyroid tetany, the calcium content of the tissues was decreased was reported by Sabbatani[5] and Quest[6] and by many other investigators. In this country MacCallum and Voegtlin[7] confirmed the above statements and also reported a marked reduction in the blood calcium caused by the increased output in the urine and feces. It, therefore, seemed advisable to inquire into the phosphate and calcium changes in the blood in guanidine tetany and to compare these changes with those occurring in parathyroid tetany in order to decide whether the increase in guanidine nitrogen is the only cause of this phenomenon or whether other factors are involved in the production of parathyroid tetany.

Marriott and Howland's methods were employed in this investigation. Rabbits were used and two samples of blood were drawn from the jugular vein; one before the subcutaneous administration of a sublethal dose of guanidine hydrochloride and the other after—about 10 to 48 hours after the injection of the drug. It is necessary to wait for the second sample of blood until the acidosis is well developed, though most animals do not live long after this condition becomes severe. Since animals usually died at midnight or early in the morning, the second blood sample

[1] Watanabe, C. K., *J. Biol. Chem.*, 1918, XXXIV, 51.
[2] Koch, W. F., *J. Biol. Chem.*, 1912, XII, 313; Ibid., 1913, XV, 43.
[3] Burns, D. and Sharpe, J. S., *Quart. J. Exp. Physiol.*, 1916, X, 345.
[4] Watanabe, C. K., *J. Biol. Chem.*, 1918, XXIV, 65.
[5] Sabbatani, "Rivist. sperim. di frematria," 1901 (quoted by Quest).
[6] Quest, R., *Jahrb. f. Kindelheinlkunde*, 1905, LXI, 114.
[7] MacCallum, W. G. and Voegtlin, C. J., *Exp. Med.*, 1909, XI, 118.

could not be taken and thus comparisons of the normal with the tetanic samples were impossible.

From the comparison in the result it is clear that after guanidine administration the phosphorus is markedly increased, in some cases five times above the normal. The calcium shows no marked change, though there is a tendency for it to decrease as the phosphate increases. In normal conditions, rabbits with albumin in the urine show a rather higher content of phosphate than those which have no albumin.

In consideration of the above experiments we may say that the phosphate content of the serum in guanidine tetany is markedly increased, but that the reduction of calcium is rather doubtful. The small number of experiments does not warrant a decision on this important phenomenon. The experiments are being continued and further results will be reported later.

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An oxidation product of creatine.

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Mercuric acetate in watery solution oxidizes creatine to α-methylguanidoglyoxylic acid ($NH_2.C(:NH).N(CH_3)CO.COOH$). This compound, which was isolated in pure form, evidently precedes methylguanidine oxalate which was obtained by Dessaignes[1] many years ago by heating creatine with mercuric oxide. Dakin[2] obtained glyoxylic acid upon oxidizing creatine with hydrogen peroxide. The stages of oxidation of creatine may, therefore, be expressed as follows:

$$NH_2.C(:NH).N(CH_3).CH_2COOH \xrightarrow{O} NH_2.C(:NH).N(CH_3).CHOH.COOH \xrightarrow{O}$$

$$NH_2.C(:NH).N(CH_3).CO.COOH \xrightarrow{H_2O} NH_2C(:NH).NH(CH_3).COOH.COOH$$

[1] Dessaignes, M., *Compt. rend. Acad.*, 1854, XXXVIII, 839.

[2] Dakin, H. D., *J. Biol. Chem.*, 1905–06, I, 271.