than that obtained with the uric acid alone. The reaction was not noted in normal controls. At that time we stated that we did not know whether phenol was the cause of this reaction.

That the so-called phenol reagent (phosphotungstate-phosphomolybdic reagent) first described by Folin and Denis[1] was not specific was shown by Tisdale,[2] Gortner and Holmes[3] and others. They showed that there were other substances which might be present in the blood that gave the same color reaction, hence interfering with the purpose of the original test. A deep blue color is also given by lactic acid, by indol, indol derivatives, protein derivatives and many other substances.

In order to eliminate at least one group of these substances, we determined the amino-acid nitrogen and the peptid nitrogen in some of these cases, using the method described by Van Slyke and Whipple.[4] Although the urea was high in some of these cases the amino-nitrogen and the peptid-nitrogen values were normal, or slightly above normal. One must remember, however, that there may be some toxic protein derivative products or amino acids present which are very toxic in small amounts, although not sufficient to perceptibly increase the amino or peptid nitrogen in the blood.

Hospital No.	Mgs. per 100 c.c.			Remarks.
	Amino N.	Peptid N.	Urea N.	
212138.....	12	14	19.6	Recovered
212031.....	12	14	15.	Died
212352.....	14	18	40.	Died
212338.....	14	17	19.6	Died
212410.....	15.4	17.1	14.2	Recovered

151 (1898)

The influence of sodium citrate on peristalsis.

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The Trendelenburg method was used for the study of intestinal movements in anesthetized animals. Doses of 30–60 mgs. sodium

[1] Folin, O. and Denis, W., *J. Biol. Chem.*, 1912, xii, 239.

[2] Tisdale, F. F., *J. Biol. Chem.*, 1920, xliv, 409.

[3] Gortner, R. A., and Holmes, G. E., *J. Am. Chem. Soc.*, 1920, lxii, 1678.

[4] Whipple, G. H., and Van Slyke, D. D., *J. Exp. Med.*, 1918, xxviii, 213.

citrate per kilo given intravenously stimulated the contractions of the small as well as of the large intestine, the effect lasting several minutes. Tonus and the rhythmic contractions were increased, but in dogs the effect on tonus predominated. Repetition of dose usually produced greater effects in cats and dogs, but in the rabbit it caused relaxation of the intestine and abolition of the rhythmic movements. This depression was followed, however, by gradual recovery. After intramuscular injections of very large doses of sodium citrate marked and prolonged stimulation occurred; both tonus and rhythmic movements were greatly augmented.

The action of citrate was also tested after the division of both vagi. The effect was usually the same as when the salt was given to animals with both vagi intact. Sodium citrate injected after atropine failed to stimulate the intestine, but when pilocarpine was injected after atropine, the subsequent administration of citrate produced temporary inhibition of the intestinal movements and marked decrease of tonus. If more pilocarpine is given and a sufficient interval of time allowed to elapse after the atropine, the usual effect upon the intestine may occur after the injection of citrate.

152 (1899)

The action of sodium citrate on the central nervous system.

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Maxwell[1] reported experiments in which sodium citrate applied to the cortex of the cerebrum in rabbits was without any effect, but when injected into the white matter underneath produced some of the symptoms of citrate poisoning. These observations were extended later by Robertson and Burnett[2] who made similar studies on the cerebellum. They likewise found that a reaction was obtained only when the solution of sodium citrate

[1] Maxwell, *Jour. Biol. Chem.*, 1906, ii, 183.

[2] Robertson, *Journ. Pharm. Exp. Therap.*, 1911–12, iii, 635.