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Chemical changes in the blood in intestinal obstruction.

By J. F. CONNORS, J. A. KILLIAN and H. B. EISBERG.

[*From the Surgical Service of Harlem Hospital, Departments of Biochemistry, New York Post-Graduate Medical School and Hospital, and of Experimental Surgery, University and Bellevue Hospital Medical College.*]

For the past two years an intensive study has been made of the non-protein nitrogen of the blood and some of its components, in various pathological conditions. The results obtained in pneumonia and the toxemias of pregnancy have been described by one of us (K) [1, 2]. This communication is an extension of that study, presenting observations on similar blood changes in patients with intestinal obstruction and a duplication of the findings in experimental animals. The studies were completed in 1921. A number of articles have been contributed to the literature reporting somewhat analogous findings. In 1916 Whipple, Rodenbaugh and and Gilgore[3] isolated from closed intestinal

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1 Killian, J. A., *Amer. Jour. Obst. and Gyn.*, 1921, ii, 6.

2 Killian, J. A., *Proc. N. Y. Path. Soc.*, 1922, xxii, 72.

3 Whipple, G. H., Rodenbaugh, F. H., and Kilgore, A. R., *Exper. Med.*, 1916, xxiii, 123.

loops toxic compounds closely resembling primary proteoses in all their properties. Cooke, Rodenbaugh and Whipple[4] have shown that in cases of acute intestinal obstruction, whether produced experimentally in animals or occurring in human cases, is accompanied by a rapid rise in the non-protein nitrogen of the blood, from 3 to 10 times normal figure. However, in chronic cases there may be little or no increase in the blood non-protein nitrogen. These intoxications also show an increased blood content of urea and creatinine. The authors state that the kidneys in all of these cases are practically normal (anatomically) and that the protein or tissue destruction, rather than impaired renal function, is responsible for the rise in the blood nitrogen. Rabinowitch[5] reports a significant increase in the urea nitrogen of the blood in patients with intestinal obstruction. In many cases it rose to more than 100 mg. per 100 c.c With this rise in urea nitrogen the author noted a normal phenolsulphonephthalein excretion by the kidneys, and he ascribed the increased urea nitrogen to tissue destruction. Haden and Orr[6] noted after ligation of the duodenum, ligation of the duodenum with gastroenterostomy and a ligation of the upper half of the ileum in dogs, a rise in the non-protein and urea nitrogen and CO_2 combining power of the blood, but a fall in the chlorides. The uric acid, creatinine, amino acid nitrogen and sugar remained unchanged. Ligation of the ileum at the ileocecal valve is followed by but a slight increase in the nitrogen, and no change in the CO_2 combining power. These authors believe that the blood urea nitrogen is a good index of the protein destruction.

At present report we are including only data on non-protein nitrogenous compounds of the blood, the sugar and CO_2 c.p. Hiller and Van Slyke[7] in a recent paper have shown after a comprehensive study of effect of the various protein precipitants used in blood analysis upon the constituents of the non-protein nitrogen, that precipitation of the proteins with 2.5 per cent. trichloracetic acid is the preferable method in analyses concerned with the undetermined nitrogen. Our procedure has been to

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[4] Cooke, J. V., Rodenbaugh, F. H., and Whipple, G. H., *Exper. Med.*, 1916, xxiii, 717.

[5] Rabinowitch, I. M., *Canad. Med. Assoc. J.*, 1921, xi, 163.

[6] Haden, R. L., and Orr, T. G., *J. Exper. Med.*, 1923, xxxvii, 365 and 377.

[7] Hiller, A., and Van Slyke, D. D.; *Jour. Biol. Chem*, 1922, lii, 253.

dilute the blood 1-5 with water and after laking has been completed, to precipitate the proteins with an equal volume of 5 per cent. trichloracetic acid. The acid then has a concentration of 2.5 per cent. of the total volume.

In simple but complete experimental intestinal obstruction the first change noted in the blood was a rise in the non-protein nitrogen from 36-90 mg. This rise is more rapid, the nearer the obstruction is placed to the duodenum, where the toxemia is most severe, indicating a corresponding increase in the rest-nitrogen, Since the dogs are on a water diet, this nitrogen must be of endogenous origin. A similar rise in non-protein nitrogen is noted in the segmental type of obstruction. However, this rise is much greater and more rapid than simple obstruction occurring at the same level. In but one animal the urea nitrogen rose above normal limits, 21.5 milligrams. The non-protein nitrogen, however, was 47 milligrams which was more than twice the urea nitrogen.

All the animals showed a decrease in the alkaline reserve as indicated by the carbon dioxide combining power. In obstructions of the segmental type the drop in the carbon dioxide combining power is more marked.

In the clinical cases reported, an increase in the non-protein nitrogen from 36 to 83 milligrams occurred. This is in accord with the experimental findings. In many cases the urea nitrogen is above normal, but it forms less than fifty per cent. of the non-protein nitrogen. The uric acid is definitely increased above normal 4-11 mg. per 100 c.c. Following operation, with relief of obstruction in most instances there is a steady decrease in non-protein nitrogen. This decrease in non-protein nitrogen was found to be associated with a clinical improvement in the patient's condition.

In one case there was a rapid rise in non-protein nitrogen following operation from 51 to 83 mg. in 4 days. Following a stormy convalescence the patient recovered. In this instance several feet of gut with questionable viability were returned to the peritoneal cavity. The increase in urea, uric acid and creatinine of the blood appears to be subsequent to a rise in the non-protein nitrogen.

When there is an accumulation of the nitrogenous waste products they follow the order of retention characteristic of renal impairment, namely a successive rise in uric acid, urea nitrogen and creatinine.

CONCLUSIONS

The results obtained above indicate that the extent of the toxemia is related to the level of the non-protein rather than the urea nitrogen. The toxemia is dependent upon the location of the obstruction in relation to the duodenum and the type of the obstruction, whether simple or segmental. From the practical standpoint the chemical examination of the blood is of inestimable value in pre-operative diagnosis and prognosis.

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Cystine metabolism.

By G. J. SHIPLE, A. R. ROSE and C. P. SHERWIN.

[*From Fordham University, New York City.*]

In our study of cystine and cystein we have had in mind four different problems.

1. An attempt to synthesize cystine in the animal body either from endogenous nitrogen and sulfur or from these same elements when fed in different forms. This could not be accomplished as has already been shown.[1]

2. We prepared several compounds of cystine and cystein[2] where first, the amino group was blocked by some radical such as the phenylacetyl or phenyluramino,—then both the amino group and the carboxyl as in the phenylhydantoin derivative,—then a blocking of the S group with a benzyl radical followed by a blocking of both S-H and amino group, and lastly a blocking of these two and the carboxyl group.

3. We have fed these compounds in order to determine whether the blocking of one or more of these groups prevent the oxidation of the cystine or cystein molecule. Besides this we

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1 Muldoon, J. A., Shiple, G. J., and Sherwin, C. P., PROC. SOC. EXP. BIOL. AND MED., 1922, xx, 46.

2 Shiple, G. J., and Sherwin, C. P. (in press), *J. Biol. Chem.*, 1923, lv, 671.