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.

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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.

wished to investigate the reactions by which cystine is apparently converted into cystein, and cystein into cystine in the body.

4. In studying the catabolism of cystine we decided to determine, if possible, the origin of the ethereal sulfates.

Lewis[3] has found that the sulfur of phenyluramino cystine is not to any great extent oxidized. He has shown that the compound is however apparently split into phenyluramino cystein, as he obtains a decided test with sodium nitroprusside, and ferric chloride, on the urine of rabbits after feeding the phenyluramino cystine.

Schmidt[4] has shown that taurine, $CH \cdot NH_2 \cdot CH_2 \cdot SO_3H$, is not catabolized in the body but passes out in the urine unchanged. Cysteic acid, $CO_2 \cdot HCH \cdot NH \cdot CH_2 \cdot SO_3H$, only undergoes hydrolytic deamination but not oxidation in the body.

We have fed to rabbits phenylacetyl cystine, phenyluramino cystine, phenylhydantoin cystine as well as cystine itself.

In these derivatives of cystine either the amino group alone or the amino and carboxyl group were both blocked, and our results have shown that a protecting of the amino group does not seem to entirely protect the sulfur, as considerable of it is excreted as sulfates.

We fed on each of three succeeding days phenylacetyl cystine to one rabbit and phenyluramino cystine to another rabbit in such amounts that each rabbit received 0.4 grams sulfur. Only a small amount of the phenylacetyl cystine could be traced, as most of it was completely lost. Enough of the original substance was recovered and isolated to determine the melting point and also some of it was reduced to the corresponding cystein compound as shown by the sodium nitroprusside test, but attempts to convert this phenylacetyl cystine into benzylphenylacetyl cystein, for its further identificaion were impossible.

In the case of the phenyluramino cystein, we were able not only to isolate some of the substance fed, but proved the presence of the corresponding cystein compound by extracting it from the urine and converting it into the benzyl derivative which

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[3]Lewis, H. B., and Root, L. E., *J. Biol. Chem.*, 1922, l, 303; Lewis, H. B., and McGinty, D. A., *Ibid*, 1922, liii, 349.

[4] Schmidt, C. L. A., Adelung, Von Aldelung, E., and Watson, T., *J. Biol. Chem.*, 1918, xxiii, 501; Schmidt, and Allen, E. G., *Ibid*, 1920, xlii, 55; Schmidt, and Clark, G. W., *Ibid*, 1922, liii, 193.

we had previous prepared. We next fed phenyluramino cystine which we had synthesized, to rabbits, to see whether this compound would be excreted or some of it changed into the cystine derivative. We found that much of it had been oxidized to the cystine compound.

This shows that the reaction

$$\text{Cystin} \rightleftarrows \text{Cystein}$$

may be a common metabolic reaction and easily as well as precisely controlled by the living cell. It is interesting in as much as it is closely allied with the finding of Hopkins[5] on his glutathione work and is strongly corroborative.

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A micro colorimetric method of estimating the hydrogen ion concentration of the blood.

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A discussion of the bicolorimetric principle was first presented to this Society in November, 1921,[1] at which time we had the present work in mind. The method described below is essentially an adaptation of the colorimetric method of Cullen[2] for the determination of the P_H of the blood plasma (or serum) to the bicolorimeter described by one of us.[3] As modified the final determination is carried out on 0.1 c.c. of plasma, and does not require more than 10 minutes after the blood has been obtained. The color comparison can be made with an accuracy of $\pm$ P_H 0.02.

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5 Hopkins, F. G., *Biochem. Jour.*, 1921, xv, 286.

* Medical Fellow of the National Research Council.

1 Myers, V. C., PROC. SOC. EXP. BIOL. AND MED., 1921, xix, 78.

2 Cullen, G. E., *J. Biol. Chem.*, 1922, lii, 501.

3 Myers, V. C., *J. Biol. Chem.*, 1922, liv, 675.