and is accompanied by a rapid disappearance of glycogen from the liver and a hypoglycemia. In view of the profound effects upon the carbohydrate metabolism produced by the ligation of the hepatic arteries, such a procedure cannot be employed with safety for the purpose of studying the effects of perfusion of the pancreas.

On the other hand, the effect of the ligation of the pancreatico-duodenal vessel is not so well known. I therefore wish to record the influence of such a procedure upon carbohydrate metabolism.

These protocols are typical of the reaction which follows the ligation of the pancreatico-duodenal artery, as determined on 8 cats. Constriction of the vessels for a few minutes causes a hyperglycemia which in most instances is accompanied by a glycosuria.

It is clear, therefore, that the effect which Collens ascribes to the perfusion of the hepatic artery after ligation of the pancreatico-duodenal artery, can at present be interpreted only as the result of the ligation of the pancreatico-duodenal artery.

Summary. 1. Application of a ligature to the pancreatico-duodenal artery produces *per se* a marked hyperglycemia and glycosuria. 2. The hyperglycemia and glycosuria following ligation of the pancreatico-duodenal artery and perfusion of the hepatic cannot be interpreted as resulting from the latter procedure alone. This is a preliminary report.

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Acetylation.

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Acetylation is a process sometimes used by the body as a method of detoxication. When, for example, *p*-amino-benzoic acid is fed either to man or to rabbit, it is partly excreted as *p*-acetyl-amino-benzoic acid. It has seemed to us that this reaction might find an

application in studies dealing with intermediary metabolism. That acetaldehyde and acetic acid are two products formed in the degradation of foodstuffs is generally believed. The evidence for their formation, like the evidence for the formation of other substances which are supposed to represent the intermediary steps in the metabolism of foodstuffs, is, as a rule, indirect rather than direct. By using such a substance as *p*-amino-benzoic acid, we are enabled to *fix*, as it were, the acetyl group. Since the acetylated compound formed can be recovered in the urine, and its amount determined, a measure of the extent to which acetylation occurs in the body may be obtained.

An important question now arises, to what extent will acetylation be affected if, in addition to the *p*-amino-benzoic acid, we feed the rabbit various substances which we have reason to believe influence the amount of acetaldehyde or acetic acid formed in the body? The results might give us more direct, more concrete evidence as to whether substances do or do not pass through the acetic aldehyde-acetic acid stage in the course of their degradation in the body.

Using rabbits as our test animals, we first fed them *p*-amino-benzoic acid for 4 days, then, in addition, the substance under examination for 4 days, and, finally, the *p*-amino-benzoic acid alone for 4 more days. The 24 hour samples of urine were collected, and the acetylated compound recovered and weighed. The percentage increase in the acetylated product as a result of the addition to the diet of the substance under examination, could now be calculated. The results are as follows, the numbers representing the percentage increases: alcohol 207, tyrosine 188, ethyl ester of acetoacetic acid 176, sodium acetate 158, β-oxy-butyric acid 133, pyruvic acid 126, glyceric aldehyde 118, histidine 102, glycerol 93, acetic aldehyde 81, olive oil 52, serine 49, *n*-leucine 42, fructose 40, glycocoll 28, alanine 27, lactic acid 22, glucose 16, tryptophane 15, isoleucine 12, sucrose 0.

The results are not altogether in accordance with views generally held. We would have anticipated, for example, that alanine, lactic acid, and even glucose, would be high up on the list; and that acetic aldehyde would not fall so considerably below sodium acetate. We realize, of course, that the numbers given represent quantitative differences to an approximate extent only; but the difference between pyruvic acid and lactic acid, for example, or even between sodium acetate and acetic aldehyde, is so considerable, that experimental errors involved in the method used cannot entirely explain

the results. We, therefore, question whether the generally accepted views regarding intermediary metabolism are not, in a number of instances, now open to revision. This is a preliminary report.

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The Distribution of Electrolytes in Dogs Following Ligation of Both Ureters.

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A study has been made of the electrolyte distribution in the blood of 5 dogs with experimental uremia, produced by ligation of the ureters. A comparison of the blood removed before operation and just before death showed very striking changes in the electrolytes. These alterations for serum are averaged as follows:

Total base = +0.1 m. Eq.
Protein anion = —1.1 m. Eq.

Chloride	= —17.9 m. Eq.	Phosphate	= + 9.4 m. Eq.
Carbonate	= — 5.5 m. Eq.	Sulphate	= +12.9 m. Eq.
Total	= —23.4 m. Eq.	Total	= +22.3 m. Eq.

There is an equimolar supplanting of chloride and carbonate by phosphate and sulphate with no consistent change in total base. Analysis of skin, muscle and brain for chloride before and after operation indicated that these tissues tend to follow the changes in the blood. Of 7 pairs of analyses (1 on brain, 3 on muscle and 3 on skin) only one failed to show a decrease in chloride after the onset of uremia. Vomitus contained insignificant amounts of chloride. The determinations on whole blood showed that the cell electrolytes vary in parallel fashion to those of the serum.

The acidosis of uremia in dogs with renal insufficiency is caused by retention of sulphate and phosphate. The base available for these acids is supplied by carbonate and chloride. It is important to note that in average figures chloride supplies three times as much base as carbonate. This is a preliminary report.