rabbit serum and typhus serum.

4. A positive agglutination in 1:400 serum dilution is significant, when the OX-19 strain supplied by the National Institute of Health is utilized. The X-19 strain must not be used even when the suspensions are treated with phenol and with alcohol, because it gives apparently non-specific agglutination in relatively high dilution with the sera of many healthy persons.

5. The serum of a large proportion of persons agglutinated proteus OXK organisms in 1:25 dilution. It was observed that the agglutination was strong, usually 3 plus, in 1:25 dilution and completely negative or very weak in 1:50 dilution in the majority of the sera.

6. The vast majority of the X-2 cultures tested (20 out of 23) failed to agglutinate with high titer typhus serum. This is not in accordance with the statement frequently encountered in the literature and in textbooks that X-2 organisms are agglutinated by typhus serum.

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Influence of Fat Mobilization on Acetone Body Production.

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The administration of estrogens to male or female birds results in a rapid increase in the blood lipids,[1-3] which is associated with the infiltration of large amounts of fat into the liver. When the administration of the estrogens is discontinued, there is a rapid return of the blood lipids to the normal concentration.

In view of these facts, it became of interest to study the influence of the rapid mobilization of fat on the rate of ketogenesis. Two groups of ducks were fasted for 3 days, and then one group was given daily a subcutaneous injection of 4 mg diethyl stilbesterol suspended in sesame oil, while the other group, used for control purposes, received a daily injection of 0.8 cc peanut oil. Every third day thereafter, until the 18th day after the beginning of the fast, blood samples were drawn for the determination of the concentration of fat[4] and total acetone bodies.[5]

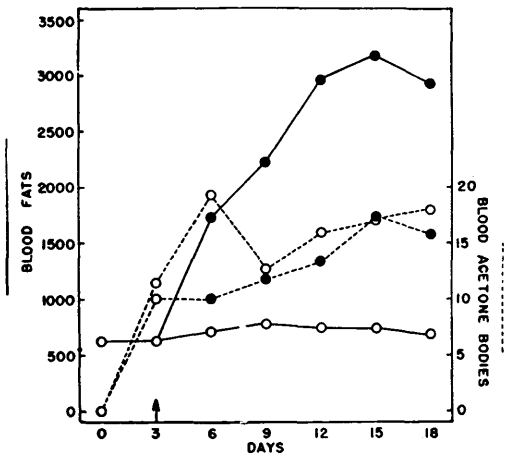

FIG. 1.

The influence of stilbesterol on blood total lipids and total acetone bodies. The open circles refer to animals receiving peanut oil and the black circles refer to animals receiving stilbesterol. The injections began on the third day after the beginning of the fast. The dotted lines depict the blood total acetone bodies, the solid lines the blood total lipids.

The results are depicted in Fig. 1, where each point represents the average findings from 6 ducks. Although a rapid lipemia ensued in consequence of the administration of stilbesterol, there was no significant effect on the rate of acetone body accumulation in

1 Zondek, B., and Marx, L., *Nature*, London, 1939, **143**, 378.

2 Lorenz, F. W., Chaikoff, I. L., and Entenman, C., *J. Biol. Chem.*, 1938, **126**, 763.

3 Flock, E. V., and Bollman, J. L., *Proc. Staff Meetings of the Mayo Clinic*, 1941, **16**, 783.

4 Street, H. R., *J. Biol. Chem.*, 1936, **116**, 25.

5 Mirsky, I. A., Nelson, N., and Grayman, I., *J. Biol. Chem.*, 1939, **130**, 179.

the blood. If anything, the stilbesterol-treated ducks showed a lower rate of acetone body accumulation than did the control ducks.

It is now recognized by many that the acetone bodies are the products of normal fat oxidation in the liver.[6,7] It has been demonstrated also that an infiltration of fat in the liver is associated with the lipemia produced by stilbesterol injections,[2,3] presumably in consequence of an extensive mobilization of fat from the depots to the blood stream. Accordingly, if the availability of lipid substrate were a factor in the oxidation of fat in the liver, an increase in the rate of acetone body production would ensue in consequence of stilbesterol administration. Our data indicate that the latter does not occur. Hence, they support the hypothesis that the availability of fats and their oxidation in the liver are two independent phenomena and that an increase in the former does not influence the latter.

Summary. The administration of stilbesterol produces a marked lipemia but does not influence the rate of acetone body production.

6 Mirsky, I. A., *J. A. M. A.*, 1942, **118**, 690.

7 MacKay, E., *Proc. Am. Diab. Assn.*, 1942, **2**, 135.

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Changes in Blood and Urine after Intravenous Amino Acid Mixture in Patients with Liver Disease.*

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Recent reports of studies on patients show that mixtures of the essential amino acids, as obtained by enzymatic digestion of casein, can be used to maintain nitrogen balance when given intravenously.[1-6] The statement has been made, however, that in patients with liver disease the method is contraindicated. Our clinical experience does not support this conclusion and, although the question requires further study, the data acquired in the present experiments seem to have some relevancy.

Experimental Method. The solution of amino acids used ("Amigen") was supplied by Dr. Warren M. Cox, Jr. of Mead Johnson and Company, and consisted of a sterilized aqueous nonpyrogenic 10% solution of an enzymatic hydrolysate of purified casein and pork pancreas. The nitrogen of the solution is present chiefly as nitrogen of amino acids and polypeptides, and the material is not allergenic. The solution contains the amino acids essential for maintenance and growth as shown by animal experiments. The hydrogen ion concentration of the solution was adjusted to a pH value of 6.5.

Amino acid concentration in whole blood and plasma was determined by the ninhydrin-CO_2 method recently developed by Van Slyke, MacFadyen and Hamilton.[7] Amino acid concentration in urine was likewise determined by the ninhydrin-CO_2 technic devised by the same investigators but as yet unpublished.[8] Standard methods were

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1 Elman, R., and Weiner, D. O., *J. A. M. A.*, 1939, **112**, 797.

2 Shohl, A. T., Butler, A. M., Blackfan, K. D., and MacLachlan, E., *J. Pediat.*, 1939, **15**, 469.

3 Farr, L. E., and MacFadyen, D. A., PROC. SOC. EXP. BIOL. AND MED., 1939, **42**, 444.

4 Elman, R., *Ann. Surg.*, 1940, **112**, 594.

5 Brunschwig, A., Clark, D. E., and Corbin, N., *Ann. Surg.*, 1942, **115**, 1091.

6 Landesman, R., and Weinstein, V. A., *Surg., Gyn. and Obstet.*, 1942, **75**, 300.

7 Van Slyke, D. D., MacFadyen, D. A., and Hamilton, P., *Fed. Proc.*, 1942, **1**, 139.