The two animals in which anæmia was produced were pale and weak, but icterus was not ascertained at any time. During the following two to three weeks, the hæmoglobin percentage and erythrocyte count rose again to normal values. In all the animals the fall in hæmoglobin was followed by a transitory increase in reticulocytes in the peripheral blood. Thus, in the two animals with pronounced fall in hæmoglobin, the reticulocyte count rose respectively from 0·2 and 0·4 per cent before the injection of enzyme to 17·8 and 19·0 per cent nine days after.

For the two controls the increase in the reticulocyte count was from 0·6 and 0·0 to 2·4 and 3·0, respectively.

The experiments described above will be reported in detail elsewhere.

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Isolation of Mucic Acid from Fruits

Mucic acid has only been recorded twice as occurring in natural products, namely, in putrified blood[1] and in the diffusion juice from sugar beet[2]. Pure mucic acid has now been isolated from sound ripe peaches and pears by methods which make it appear certain that the acid was present in the free state in the fresh fruit.

The extraction and separation of the acids was carried out as described previously[3], except that the solution of acid and sugars (after removal of the bases and amino-acids with a cation-exchanger) was run straight on to a column of a strong-base anion-exchanger in the acetate form. This column was placed above two or more smaller columns of the same resin in the acetate form, and the acids were displaced with 0·1 *N* hydrochloric acid. Paper chromatograms of the fraction(s) between the malic and citric acid bands contained an acid with $R_F = 0$ in methyl ethyl ketone – cineole – aqueous formic acid (53 per cent w/v) (50 : 50 : 36 v/v)[3], and $R_{malic} = 0{\cdot}9$ in alcohol – ammonia – water (80 : 5 : 15)[4]. The isolated acid and mucic acid behaved identically on paper chromatograms and ion-exchange columns. The acids were revealed by rapidly pulling the paper through a solution of silver nitrate in acetone, followed by (*a*) heating the paper for a few minutes at about 100° C., and then (*b*) spraying it with alcoholic sodium hydroxide (cf. ref. 5). The chromatogram was then fixed with sodium thiosulphate (5 per cent). After treatment (*a*), all acids gave a white spot; after (*b*) acids having an α,β-glycol or an α-keto group gave an intense black spot on a grey background.

When the fractions containing mucic acid were allowed to stand at + 1°, crystals separated. Recrystallization from sodium hydroxide – hydrochloric acid yielded an optically inactive acid of melting point 215° (decomp.)[6], not depressed by authentic mucic acid. The diphenylhydrazide had melting point 242°[6]. The acid from peaches gave on analysis C, 34·2; H, 4·8; O, 61·0; that from pears C, 34·1; H, 4·9; calc. for $C_6H_{10}O_8$: C, 34·3; H, 4·8; O, 60·9 per cent. About 100 mgm. of mucic acid was isolated from 2 kgm. of ripe peaches or pears. Apricots, passion fruit and blackberries probably contain mucic acid in variable amounts, since crystalline precipitates, melting point 214–215°, were isolated from them by the same method.

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Effect of Silver Ions on Mitochondrial Adenosine Triphosphatase

Abood, Gerard and Ochs[1] claim that the respiration of cell-free dispersions and mitochondria of brain tissue was increased by the passage of electrical impulses between silver electrodes in the suspension medium. In general, increase in the respiratory rate of well-prepared mitochondria is consequent upon increase in the rate of removal of adenosine triphosphate, by addition of systems which catalyse its dephosphorylation, or through activation of the mitochondrial adenosine triphosphatase[2]. We were thus led to test the effect of electrical impulses on the adenosine triphosphatase activity of mitochondria, obtained from rabbit cerebral cortex, using the medium which had enabled the preparation of mitochondria of low initial adenosine triphosphatase activity from pigeon breast muscle[3]. For the adenosine triphosphatase assays the particles were incubated for 10 min. at 20° in a medium of the following approximate composition: 0·1 *M* potassium chloride, 0·05 *M* amino*tris*hydroxymethylmethane-hydrochloride buffer *p*H 7·4, 0·005 *M* magnesium sulphate, 0·005–0·0075 *M* sodium adenosine triphosphate, and 5×10^{-5} *M* ethylenediaminetetraacetate. With 0·0075 *M* adenosine triphosphate, mitochondria from 60 mgm. of tissue liberated about 20 μgm. of inorganic phosphate under these conditions, and the activity was increased three-fold on addition of 10^{-4} *M* 2 : 4-dinitrophenol.

Electrical impulses (2–3 V., 25–75 m.amp., 50 c./sec.) were applied during the incubation through dipping electrodes (type *G* of Ayres and McIlwain[4]). With gold and molybdenum electrodes, there was no effect on the adenosine triphosphatase activity; but with silver electrodes the rate of phosphate liberation increased two- to three-fold. However, the mere presence of the silver electrode, without passage of current, or addition of low concentrations (10^{-6} to 10^{-5} *M*) of silver nitrate, led to similar increases in adenosine triphosphatase activity.

In a paper[5] which appeared after the conclusion of these experiments, Narayanaswami and McIlwain state that pulses from molybdenum electrodes did not affect adenosine triphosphatase activity of dispersions of cerebral tissue, and, indeed, caused only a small increase in the respiration of brain mitochondria. Our experiments, however, disclosed the stimulation of mitochondrial adenosine triphosphatase by silver ions; and, since dinitrophenol also has this effect, it was of interest to determine whether silver ions behave similarly to dinitrophenol in other