

rine. As little as 0.2 μ g of arterenol per ml can produce this effect. It can be determined visually that the initial peak in curves 2 and 3 (Fig. 2) has the light green color of epinephrine fluorescence, whereas the secondary peak has the deeper green color of arterenol.

In the course of these studies various buffers were used. When small quantities of inorganic phosphate were combined with arterenol before the addition of hydroxide, the fluorescent maximum was increased by a factor of 6, and the time required to reach the maximum was reduced by 1/2 (Fig. 3). However, when the phosphate was combined with the hydroxide, and this mixture added to the arterenol, no phosphate effect was observed. When epinephrine and phosphate were combined, a diminution in fluorescent intensity occurred, and the time to reach the maximum was slightly reduced. A simple summation of these effects was found when arterenol, epinephrine, and phosphate were tested in solution together.

Although the explanation for the described action of arterenol on epinephrine is not at hand, it is suggested

that oxidation products of arterenol may act as oxidizing agents on epinephrine. Such an assumption finds support in observations made by Falk (2) which describe the increased rate of epinephrine oxidation in the presence of one of its oxidation products, adrenochrome. The influence of phosphate on arterenol cannot be explained in this manner. It is assumed that the fluorescent product of arterenol contains an indole ring, as does the fluorescent substance of epinephrine, and that ring formation is accelerated in the presence of phosphate.

The physiological significance of these interrelationships is under investigation.

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Biotin Deficiency as the Causative Agent of Induced Cryptorchidism in Albino Rats

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In 1948, the egg-white injury condition (1) was produced in four weanling male albino rats, by feeding an experimental diet in which spray-dried egg white at a 66% level was the sole source of protein. Four additional weanling males were maintained as controls on a commercial stock diet. The testes of all animals fed the experimental diet failed to descend. Autopsy revealed these organs to be abnormally small and located in the posterior abdominal region.

In an experiment just concluded, four 30-day-old rats, in which the testes had already descended, were placed on the same experimental diet. As a control group, four additional males of the same age were also placed on the same diet in which, however, the egg white was heat-treated at 121° C for 3 hr. By the 19th day, the testes of all animals fed the unheated egg white had

returned to the abdominal position. Autopsy produced the same findings as in the earlier test. Histological examination of the testis tissue showed incomplete spermatogenesis and abnormally small or completely closed tubule lumina (3).

That the egg-white injury condition is a biotin deficiency has been shown by Eakin *et al.* (2). In the advanced stages of the deficiency in this particular experiment (as in the 1948 test) there was, among other symptoms, a condition in which many of the muscles of the biotin-deficient animals were in a continuous state of tetany. This suggests that the retention of testes in the abdomen, in the one instance, and the return of these organs to the abdomen, in the other, are due to the contracture of the cremaster muscle. These data appear significant, since the growth and development of the control animals in both experiments were normal.

The results of the described exploratory tests merit further and more extensive investigation.

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