TABLE II.
Effect of Vitamin E on Incidence of "Experimental" Arterial Lesions.

Diet	Weeks*	No. with lesions	
		No. in group	% positive
Table scrap + C.L.O.†	8-15	22/25	88.0
Table scrap + C.L.O. + Vitamin E	8-14	1/8	12.5

* Weeks of dietary feeding before production of renal insufficiency.
† C.L.O. = Cod Liver Oil.

Summary. 1. Twenty-two of 25 dogs fed a kennel diet of selected table scraps to which was added each day for 8 weeks or longer 3.0 cc/kg of cod liver oil developed typical arterial lesions when they died in uremia 7 to 35 days after the experimental production of renal insufficiency.

2. When vitamin E (mixed natural tocopherols, 2.5 to 4.0 mg/kg/day by mouth) was administered along with the diet, none of 4 dogs subjected to the same experimental procedures developed any arterial lesions.

3. Vitamin E was equally effective in preventing or retarding the arterial lesions when its administration by mouth was started 0, 1, and 2 days after renal insufficiency was induced.

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Cataract Development in Animals with Delayed Supplementation of Tryptophane.

A. J. SCHAEFFER* AND E. GEIGER.

From the Van Camp Laboratories, Terminal Island, California.

In several recent papers[1-4] demonstrating the production of lesions in the eyes of experimental animals on inadequate diets, corneal vascularization was encountered more frequently and as a more general response to deficiencies than cataract formation. The latter lesion was observed only rarely, chiefly in rats fed on tryptophane or histidine deficient diets. The development of cataract might be a result of disturbed protein synthesis due to the absence of the essential amino acids; however, since cataract did not develop in animals on diets deficient in other essential amino acids such as lysine, leucine, threonine, valine, isoleucine, arginine, etc., the possibility of a specific role of tryptophane and histidine in the metabolism of the lens must be considered. It was the intention of the present study to investigate this possibility by the method of delayed supplementation of tryptophane. One of the authors[5] has shown that infantile rats, on a diet of incomplete amino acid mixtures, did not grow if the missing amino acid was fed separate from the rest of the diet several hours later. These experiments indicated that amino acids are not stored in the body, and that protein synthesis can proceed only when all essential amino acids are simultaneously present in the tissues.

If the delayed supplementation of tryptophane would prevent cataract formation with-

* 4041 Wilshire Blvd., Los Angeles, Calif.

1 Chan, T. T., Chang, C. Y., and Luo, T. H., *Chinese J. Physiol.*, 1941, **16**, 241.

2 Totter, R. J., and Day, P. L., *J. Nutrition*, 1942, **24**, 159.

3 Albanese, A. A., and Buschke, W., *Science*, 1942, **95**, 584.

4 Sydenstricker, V. P., Schmidt, H. L., Jr., and Hall, W. K., PROC. SOC. EXP. BIOL. AND MED., 1947, **64**, 59.

5 Geiger, E., *J. Nutrition*, 1947, **34**, 97.

out promoting growth, we might conclude that this amino acid exerts its protective action independent of protein synthesis. Such a possibility has been suggested by Cahill and Kohalik[6] who found that abrine (1-methyltryptophane), which is not as efficient as tryptophane in promoting growth, nevertheless prevents cataract in animals on tryptophane free diets.

The experiments were performed on infant rats (35-45 g body weight) in 2 groups of 6 each. The first group was fed the tryptophane deficient Diet I for 12 hours, and Diet II, containing tryptophane, for the next 12 hours. The second group received Diet I for 2 hours and Diet II for 2 hours; all food was withdrawn for the duration of one hour when diets were changed in this group. For further technical details see Geiger.[5]

Composition of Diet I — 30 g acidhydrolysed fish protein—70 g basal diet.

Composition of Diet II — 6 g l(−) tryptophane — 94 g basal diet.

The basal diet contained: corn starch, 3050 g (78.3%); rice bran concentrate, 400 g (10.26%); cottonseed oil, 200 g (5.13%); U.S.P. salt mixture, 200 g (5.13%); fish oil (one g contains 2,000 I.U. vitamin A and 400 I.U. vitamin D) 50 g (1.27%); riboflavin, 75 mg; Ca pantothenate, 150 mg; choline chloride, 2.5 g.

The results regarding growth were essentially the same as those reported in the previous study. In spite of an adequate supply of tryptophane derived from the two diets consumed in 24 hours, the rats failed to show normal growth; this again demonstrated that tryptophane fed apart from the other amino acids could not be utilized for protein synthesis. We did not observe the alopecia or hyperexcitability described by Totter and Day.[2] These symptoms probably resulted from other nutritional lack (deficiency or certain vitamins, perhaps) of the diet employed by these authors.

The ocular findings were as follows. Lenticular involvement and corneal vascularisation was noted in 10 of the 12 experimental animals. The 2 animals with no sign of cataract had been on the experimental diet for only 6 weeks. In all cases the lenticular opacities showed the character of a posterior cortical cataract. The opacities were first noted subcapsularly, but gradually involved most of the posterior cortex, and in 2 cases progressed to the anterior cortex, the nucleus, and finally the entire lens.

The shape of the opacities showed considerable variation. In one case, it involved the central portion of the posterior cortex, forming a flower shaped figure, while the peripheral region remained clear. In other cases, the peripheral margin developed denser opacities, while the central area remained clear. The suture lines became visible; dehiscense of the lines, fluid clefts and vacuoles became more and more noticeable. In a few cases the anterior cortex became involved, principally in the equatorial region. In 2 cases, this development was followed by progressive intensification of the milky, diffusely opaque appearance of the whole lens.

The period of development also showed marked variations. In most cases, some definite sign of cataract formation could be observed by the sixth week of the experiment. In the 2 cases where cataract development proceeded furthest, some incipient signs had been noted by the fourth week of the experiment with a rapid subsequent course. By the twelfth week the lenses appeared whitish and opaque to the unaided eye in these animals, while in other animals only posterior cortical opacities were observed.

These results are similar to, if not identical with, the growth and eye manifestations noted in the studies of rats on tryptophane deficient diets. There are no clinical differences between animals completely deprived of tryptophane and those receiving delayed supplementation with respect to corneal vascularization and cataract formation.

In these experiments, ocular symptoms developed in spite of the fact that the rats ingested relatively large quantities of tryptophane (0.1 to 0.2 g per day). Prevention of cataract, like promotion of growth, can be achieved only if tryptophane is supplied

[6] Cahill, W. M., and Kotalik, G. C., *J. Nutrition*, 1943, **26**, 471.

simultaneously with the other essential amino acids. This suggests a possible causal relationship between disturbed protein synthesis and cataract formation. Further investigations as to why disturbance of protein synthesis by the lack of essential amino acids other than tryptophane and histidine does not result in cataract formation is required.

Summary. Experiments with delayed supplementation of tryptophane show that cataract formation in rats occurs even after consumption of relatively large quantities of tryptophane. It seems that like growth promotion, cataract prevention by this amino acid can be accomplished only when it is supplied simultaneous with the other essential amino acids.

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Simulated Auricular Flutter in the Electrocardiogram of the Dog.

A. SURTSHIN. (Introduced by L. N. Katz.)

From the Cardiovascular Department, Research Institute, Michael Reese Hospital, Chicago.*

In the course of studies with epinephrine in the unanesthetized dog, undulations of the baseline at a rate of the order seen in auricular flutter developed in the electrocardiographic tracing. At first sight, the mechanism appeared to be auricular flutter. The coincident occurrence of tachypnea and retching during the recording of these electrical phenomena suggested the possibility that these undulatory waves might be extracardiac, caused by retching and tachypnea.

The possibility exists that similar records in man might, on occasion, be misinterpreted as auricular flutter. It thus appears advisable to present these records for publication.

In Fig. 1A are shown two sections of a continuous recording of lead 2 obtained in the dog after the intravenous injection of one mg epinephrine. In the first few cardiac cycles of the top strip regular undulations in the baseline at a rate of 356 per minute resemble the "F" waves of auricular flutter. This was associated with tachypnea of the same rate. The ventricular rate is 65 per minute. In the middle of the tracing during a period of transitory apnea, the undulations are absent, no P waves are seen, and the contour and rate of the ventricular complexes are unchanged (the QRS and T waves are huge and upright). Toward the end of the top strip the phenomenon in the first part of the tracing recurs and continues during the first part of the second strip which was recorded 2 minutes later. In the middle of the second strip it is possible to identify P waves even though they are partially obscured by the undulations. Some of these are labeled. The ventricular rate at this time is 88 per minute and irregular. We are dealing here with a nodal rhythm followed by a sinus arrhythmia at a faster rate and the record is obscured by extracardiac artefacts simulating impure auricular flutter.

Fig. 1B is a tracing of the femoral arterial pressure in the same dog recorded with a Hamilton manometer simultaneously with a pneumogram obtained with a pneumograph strapped around the animal's chest. Below is a baseline recording time in seconds. The pneumogram shows post-epinephrine tachypnea at a rate of 306 per minute. In some instances, not seen here, these respiratory waves may even appear as noticeable fluctuations of the blood pressure within a single cardiac cycle.

Fig. 2 is a continuous electrocardiographic tracing (lead 2) obtained under similar circumstances in another dog. At the beginning

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