later, the rats on the lysine-deficient ration had shown a slight weight loss, while those receiving l-lysine were gaining weight. The largest weight losses were recorded by the rats receiving the synthetic racemic compound (Table 1). One of the rats in this group died on the 11th day, and the experiment was discontinued. The animals were sacrificed and the kidneys were dissected and weighed. Kidneys of rats receiving the α-amino-ε-hydroxy caproic acid were slightly heavier, but histological examination[1] failed to reveal any differences between groups.

TABLE 1

WEIGHT CHANGES (GRAMS) IN RATS RECEIVING α-AMINO-ε-HYDROXY CAPROIC ACID AND IN CONTROL GROUPS

	Zt	Ztl	Ztl-OH
No. of rats per group	4	6	6
Wt. changes during last 10 days of preliminary period (24 days)	−4	−3	−2
Range	−3, −6	−1, −4	−2, −3
Wt. changes during experimental period (11 days)	−2	+5	−8
Range	−1, −3	+2, +9	−6, −10

It is concluded that α-amino-ε-hydroxy caproic acid cannot replace lysine in the diet and that it may have some toxic effect.

Mechanism of Renin Tachyphylaxis—Restoration of Responsiveness by Tetraethyl Ammonium Ion

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The pressor responses to repeated intravenous injections of renin in intact animals diminish progressively. This phenomenon is known as tachyphylaxis. The action of renin is dependent on the presence of plasma renin-substrate, the exhaustion of which might account for tachyphylaxis. But administration of large amounts of α_2 globulin, the fraction of plasma protein containing substrate, does restore the response in tachyphylactic animals (*6*).

Renin through its effector agent, angiotonin, acts directly on peripheral blood vessels. Nevertheless, sectioning of the brain at certain levels causes the pressor response to be reversed to depressor (*3*), a phenomenon which has been further localized by Euler and Sjöstrand (*2*). Widespread direct injury to the central nervous system also temporarily abolishes the pressor response to angiotonin (*4*), as does severe shock produced by a variety of means (*5*). Thus, the nervous system influences vascular responsiveness to angiotonin.

One of the difficulties of accepting the renal-vasopressor system as a cause of renal hypertension has been the phenomenon of renin tachyphylaxis.

Acheson and Pereira have shown that injection of tetraethyl ammonium chloride (*1*) blocks sympathetic ganglia. We have found that repeated (1–4 times) intravenous injections of 100 mg. into normal dogs anesthetized with pentobarbital completely overcame tachyphylaxis induced by repeated injection of renin. The response to angiotonin itself was also augmented. Even more striking was the manyfold increase in the response to adrenalin at a time when the response to nicotine was wholly abolished. The responses to histamine, mecholyl, and barium chloride were usually also augmented. Without anesthesia the same phenomena occur, but to a lesser degree. Adrenalectomy or nephrectomy in brief experiments did not alter these responses.

If the caudal spinal cord was destroyed after section at C_8 and the animal tested the next day after complete recovery from anesthesia, the responses to renin were augmented. Further, 8 injections of renin did not lead to appreciable tachyphylaxis. Administration of tetraethyl ammonium ion now did not further augment the already large response.

[1] Kindly done by C. Auger, of the Pathology Department.

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Absence of a Macrocytic Anemia in Dogs Fed Choline or Choline Plus Fat

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Several groups of investigators in this university have been interested in choline since this substance was established as a dietary factor by the work of Best and Huntsman (*1*). While no detailed study of the effect of choline on the blood picture had been made here, a number of hemoglobin and red cell estimations in dogs fed large amounts of choline had failed to reveal any signs of anemia. A series of papers by Davis (*2*), in which it was reported that the administration of choline or choline plus fat produced in dogs a macrocytic anemia, came, therefore, as a surprise. An additional stimulus to the investigation of Davis' claims was provided by the fact that choline or its precursor, methionine, is now being used in many clinics in an effort to determine its role in the prevention of fat deposition or cirrhotic changes in the liver of patients.

A series of four normal male dogs was carefully observed by one of us (M. F. C.), and after the somewhat extended period necessary to obtain satisfactorily constant basal values for the red cell counts and the hemoglobin levels, choline or choline plus fat was added to the stock diet of the animals, which consisted of Canada Packers Frozen Dog Chum, sucrose, and bone meal. Dried yeast and cod-liver oil were provided twice each week and tomato juice once a week. All four dogs were on this diet for at least three months before the feeding of choline or choline plus fat. During this period they all gained weight, and their coats improved in texture.