TABLE I.

Patient	Diet Na	Urine vol, ml/day O	Urine vol, ml/day A	Urine Na, meq/day O	Urine Na, meq/day A	Urine K, meq/day O	Urine K, meq/day A	Feces wt, g/day O	Feces wt, g/day A	Fecal Na, meq/day O	Fecal Na, meq/day A	Fecal K, meq/day O	Fecal K, meq/day A
K.	500	638	1077	29.5	22.6	37.5	37.9	81	151	1.5	5.4	7.4	14.9
	1500	1071	1424	21.6	57.7	46.1	50.3	102	164	2.2	10.2	5	14.6
P.	500	2307	2332	87.1	48.7	82.1	51.8	166	161	4.9	2.2	21.7	27.4
	1500	2560	2237	96.3	88.5	101.1	87.7	151	248	2.2	9.2	19.5	36
N.	500	1489	2017	89.8	48.4	102.6	65.9	76	135	1.9	2.9	7.9	21.8
Z.*	500	2074	2252	53.2	24.7	74	45.5	119	185	3	6.1	10.6	12.7
	1500	1727	2050	69.1	38.2	72.7	50.9	233	349	6.7	15.8	26.3	43.4

* Z (control patient).
O = control period; A = alginic acid period.

fact that his regular mercurial medication was temporarily increased. Despite this, his fecal excretion of sodium significantly increased. The large increase in urinary sodium excretion in this instance was felt to be associated with the marked diuresis of this cirrhotic patient.

The results of this study indicate that the adsorption of sodium by alginic acid is approximately one-fifth to one-sixth of the values reported for cation exchange resins in clinical use(6). The reasons for the discrepancy between the *in vitro* cation adsorption capacity of alginic acid and the results reported here are not clear. It may be that partial digestion of alginic acid occurs, as has been reported in animals(3), or that an insoluble calcium alginate is formed in the stomach.

In view of the definite sodium adsorbing capacity of alginic acid, although admittedly small in comparison with cation exchange resins, and because of its favorable tolerance by patients and lack of marked undesirable side effects, it is felt that further trial and clinical investigation may be warranted. Possible chemical modification of alginic acid to increase its *in vivo* adsorption capacity may be worthy of study.

Conclusions. 1. Alginic acid is well tolerated in humans when ingested in quantities of 45 g per day and is mildly laxative. 2. Alginic acid increases fecal sodium and potassium excretion in humans to the extent of approximately one-fifth to one-sixth that of the cation exchange resins in clinical use.

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Vitamin E in Pituitary Gland Function of Fowls. (19406)

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The necessity of vit. E for proper function of the anterior pituitary gland has been a controversial problem for a number of years. Verzar(1), in early studies, stated that vit. E acted like anterior hypophyseal hormone in

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inducing precocious sexual maturity in young rats, and that vit. E might be necessary for the formation of hypophyseal hormone. Diakov and Krizenecky(2) and Mattill(3) believed that vit. E did not exhibit gonadotropic effects, and that chorionic gonadotropin and corpus luteum extracts did not maintain the testes of rats on an E-free diet. Drummond, Noble, and Wright(4) found that vit. E had no effect on the reproductive organs of immature or hypophysectomized rats. They also concluded that hypophysectomy of vit. E-deficient rats caused further decline in weight of the testes and that the pituitary glands of the E-deficient rats contained an increased amount of gonadotropic hormone. The appearance of castration cells in the pituitaries of animals on an E-free diet is of particular significance. Mason(5), Evans and Burr(6), Nelson(7), and Mattill(3) found castrate cells in the pituitary glands of male rats that had been on an E-free diet for 120 days. They did not, however, find castration cells in female rats that were on an E-free diet. Severinghaus(8) stated that castration cells do not develop in the pituitary glands of guinea pigs while on a diet deficient in vit. E. The work of Van Wagenen(9), Nelson(7), and Mattill(3) disclosed, however, that castration cells in the pituitary developed during a deficiency in vit. E. Rowland and Singer (10) concluded that when there is a deficiency of vit. E, the pituitary gland does not have the capacity to produce an ovulation-producing substance which will cause ovulation in the oestrus rabbit. Müller and Müller(11) kept female rats on a vit. E-free diet for 137-240 days with no morphological changes in the pituitary gland. After 280 days on the diet, however, the pituitaries showed castration cells. Stein(12) found no notable pituitary changes from an E-free diet in rats. Nelson(7) found that vit. E is necessary for normal pituitary and testis growth. Biddulph and Meyer(13) found that after rats had been on a diet deficient in vit. E the testes showed reduction of about 50% in size but that pituitary glands from these animals were not altered in gonadotropic potency. They further report an increase in size of the basophilic cells after the animals had been on the E-deficient diet for some 6 months.

Prior to 1944 a series of 4 experiments were conducted in this laboratory to test the gonadotropic activity of the pituitary glands of chickens that were on a diet deficient in vit. E. An abstract was published(14). Recently this work was repeated but the results were so uniform in all groups of animals and both series of tests, the data are combined in those places in which experimental conditions were parallel.

Materials and methods. In 6 different groups 164 male chickens, one-half of which received vit. E and the other half deprived of vit. E, were studied. They were 6 weeks of age when the E-free diet was started and they were kept on it for 8 weeks. The basic diet used in all experiments consisted of: dry casein 18%, salt mixture 4%, corn starch 36%, rice 20%, dried brewers yeast 10%, lard 5%, fiber (ground cane fiber or ground oat hulls) 5%, and fish oil extracts 2% (or equivalent levels of vit. A and D). To one-half of the birds, vit. E was supplied in the form of mixed tocopherols. The diets were available to the birds at all times. At the termination of each experiment the birds were killed and their pituitary glands removed. The pituitary glands from 21 of the birds that received vit. E and a like number that did not receive the vit. E, were fixed in Heidenhain's Susa fixative for histological study. All others from both groups were prepared for the assay of gonadotropic hormones.

Observations. The pituitary glands prepared for histological study were stained with Heidenhain's Azan triple stain, a modification of Mallory's triple stain. Glands from the birds fed an E-free diet showed many basophils that were similar to those described by Severinghaus(8) as being in the later stages of secretion. This is also in agreement with results described by Barrie(15). The nuclei were highly pyknotic and the cytoplasm was much reduced in quantity. Counts of types of cells indicated that the glands from birds supplied with vit. E contained close to 100% more active basophils than those from birds given no vit. E. No castrate cells were found in the birds deprived of vit. E. In these experiments the birds were on the E-free diet a

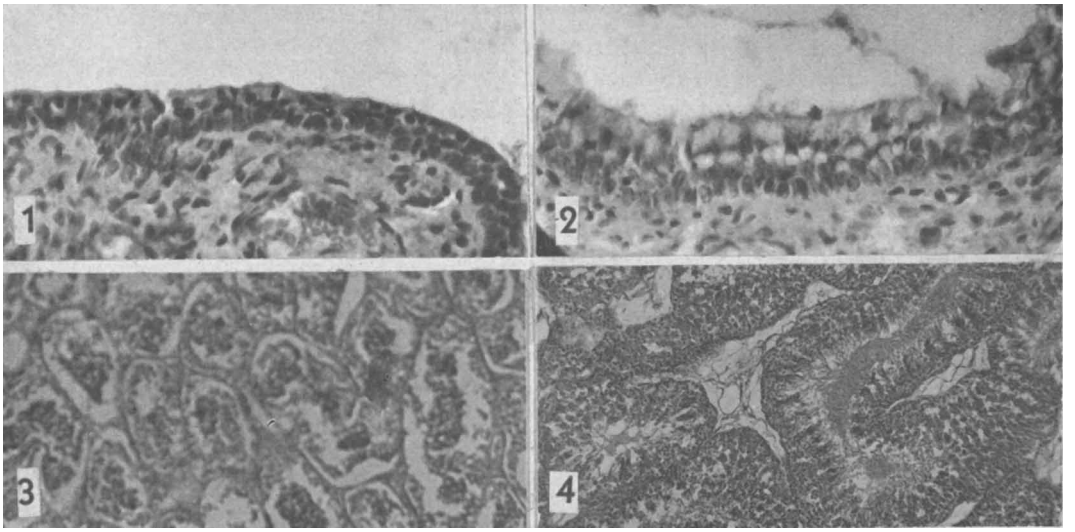

FIG. 1. Photograph of section of vaginal epithelium from assay rat that had received pituitary substance from animals deficient in vit. E.

FIG. 2. Photograph of section of vaginal epithelium from assay rat that had received pituitary substance from animals supplied with vit. E.

FIG. 3. Photograph of section of testis tissue from chicken that had received no vit. E for 8 weeks.

FIG. 4. Photograph of section of testis tissue from chicken fed the same diet as in Fig. 3, except that vit. E had been added to the diet.

shorter period of time than that reported by others in which castrate cells were observed. In the experiments reported here, however, the testes showed marked degenerative changes (Fig. 3).

In the birds that received vit. E the testes appeared normal in size and structure and averaged 9.71 g each in weight (Fig. 4). The thickness of the walls of the seminiferous tubules measured 44.29 μ. In the birds deprived of vit. E, the testes averaged 6.56 g in weight and the walls of the seminiferous tubules measured 27.10 μ in thickness. The relative degree of activity of the testes was reflected in the size of the combs. In 2 experimental groups that were weighed, the combs of the E-fed group weighed 44 g, whereas those from the E-deficient group averaged 20 g each.

Procedure and results of assay. All pituitary glands that were removed for assay purposes were ground to a paste and then combined with distilled water, one ml for each gland. The suspension was injected into sexually immature female mice or rats once daily for 3 days, a total of 0.3 ml being given at each injection. Following the series of injections the test animals were killed and the vaginal tracts were removed, fixed and sectioned. As a means of evaluating the degree of gonadotropic activity the thickness of the vaginal epithelium was measured. Care was exercised to choose regions of the vagina for measurement that were at the same distance from the cervix and four measurements were made for each test animal. The mean of the measurements from the E-fed groups, in which rats were used for the assay, was 58.25 μ whereas the mean of the E-deficient groups was 38.78 μ (Fig. 1 and 2). An analysis of all measurements within the groups (Snedecor, p. 80, 16) reveals a statistically significant difference between these two means ($t = 5.21$ and $P = <.001$). The measurements in which mice were used as the assay animals were not incorporated here since they were in a different range of magnitude although their comparative values were essentially the same as those reported here for the rats. It is concluded from these results that pituitary glands of male chickens contain more gonadotropic hormone if vit. E is supplied in the

diet than if it is not supplied.

Summary. The testes of male chickens kept on a diet deficient in vit. E for two months were much reduced in size, and the seminiferous tubules showed considerable degeneration. The combs, likewise, were smaller in the group deprived of vit. E. The assays of pituitary glands revealed that more gonad stimulating substance was produced by those of fowls which had received vit. E in their diet. The pituitary glands from the birds on the E-deficient diet had approximately one-half as many active basophilic cells as those from the birds with the E supplied.

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Pulmonary Infarction from Cardiac Catheterization.* (19407)

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The major complications that have been reported as a result of cardiac catheterization have been reviewed by Zimdahl(1) and have been of two general types: First are a variety of cardiac arrhythmias developing largely as a result of the introduction of the catheter into the right auricle and ventricle. These have consisted of ventricular premature beats, ventricular tachycardia, ventricular flutter, ventricular fibrillation, right bundle branch block, and heart block. Auricular premature beats, auricular flutter, paroxysmal auricular tachycardia, and auricular fibrillation also have occurred when the tip of the catheter has been in the right auricle. The second type of complication has been thrombosis occurring in the veins at the venotomy site and sometimes propagating centrally. In one reported case (2), in whom the catheter was introduced into the heart by way of a leg vein, a thrombosis propagating centrally to the heart resulted in death several weeks later.

We wish to report the production of pulmonary thrombosis and infarction in a series of patients with pulmonary congestion in whom a branch of the pulmonary artery has been occluded in order to record the pulmonary "capillary" pressure.

Methods. To measure the pulmonary "capillary" pressure, the catheter is introduced by the usual fashion into the pulmonary artery and then advanced further until it is wedged into one of the distal branches of this vessel(3). On the basis of about 100 ex-

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