more palatable to the monkeys than was a dry ration. The ration was offered in small amounts, about 50 g, 3 times daily. Control animals received the 18% casein basal(7).

In the first experiment, a group of 9 monkeys was placed on the basal ration containing 2% 6-MT and 0.05% dl-tryptophan for 7 days; and on the 8th, 9th, and 10th days this group and a control group of 9 monkeys were fed a total of 120 ml of 10% virus suspension (about 1,200,000 intracerebral LD_{50}) in 6 doses of 20 ml each. Temperatures were recorded daily and the animals were observed for signs of disease twice daily. Three weeks after the first virus feeding the surviving animals were placed on the 18% casein basal diet. The results are recorded in Table I.

Exp. 2 and 3 (Table I) were similar except that 1) the 2% 6-MT ration was not supplemented with tryptophan and 2) the monkeys received two feedings of 20 ml of a 1:400 dilution (approximately 10,000 intracerebral LD_{50}) from the same pool of Wisconsin '45 virus on the eighth day of the experiment. The surviving animals of all 3 experiments were challenged intracerebrally with 500 LD_{50} Wisconsin '45 virus 6-9 weeks after their oral exposure.

In Exp. 1, there was a suggestion of protection against the virus in the 6-MT group, *i.e.*, incubation periods were longer, febrile prodromata less pronounced and two animals escaped paralytic infection. These 2 animals, No. 3 and 4, apparently did experience an inapparent infection for they resisted a large intracerebral challenge 9 weeks later. In the second and third experiments, the smaller inoculum resulted in a lower incidence of paralytic infection in the controls, 8/10 and 4/9 respectively, while only one of the 20 animals receiving 6-MT became paralyzed. The surviving animals in the latter experiment succumbed to intracerebral challenge.

Summary. These experiments are obviously of a preliminary nature but they do show that the susceptibility of a primate to poliomyelitis via a natural portal of entry may be altered and they suggest that this approach to the modification of susceptibility to poliomyelitis should be explored further.

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Effect of Adrenocortical Steroids on Duration of Pentothal Anesthesia in Adrenalectomized Mice. (20628)

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It is well known that the adrenalectomized animal becomes less resistant to various types of damaging agents and more sensitive to various drugs, including anesthetics. And recently, much has been published on the central action of adrenocortical steroids. It is supposed from these that the adrenal gland may play an important role in anesthesia. The present experiment was performed to confirm this.

Materials and methods. The animals used in this study were 60 female albino mice weighing about 20 g, divided into 6 groups of 10 mice each, as shown in Table I. The

TABLE I. Duration of Pentothal Anesthesia in 6 Groups of Albino Mice.

Operation	Treatment	Mean waking time (min.)	Index of waking time	P (%)
Intact	—	25	1.0	
Adrex	—	110	4.4	$P < 0.5$
"	Cortisone acetate	33	1.3	$25 < P < 50$
"	Hydrocortisone acetate	28	1.1	$50 < P$
"	DOCA	100	4.0	$P = 0.5$
"	Saline	83	3.3	$P < 0.5$

anesthetic employed was pentothal sodium (Ravonal Tanabe). Cortisone acetate and hydrocortisone acetate, both in saline suspension (Cortone and Hydrocortone* Merck), and DOCA in sesame oil (Cortate Schering) were administered subcutaneously. The dose of the steroids was 0.5 mg daily for 2 days; 0.3 mg/10 g of pentothal sodium (3 mg/ml in isotonic saline) was administered intraperitoneally about 18 hours after the last injection of the steroids (about 48 hours after adrenalectomy). The anesthesia time was determined by Winter's method(1) on the same number of animals from each group at one time to avoid day to day variation. By Snedecor's t-test, the difference in mean anesthesia time between groups was determined as significant if p was smaller than 5%.

Results. As shown in Table I and Fig. 1, the mean duration of pentothal anesthesia in the adrenalectomized group is 4.4 times longer than in the intact group ($p < 0.5\%$).

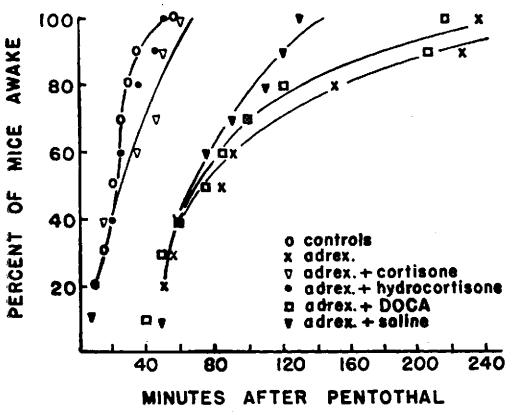

FIG. 1. Effect of adrenocortical steroids upon the waking response curve of adrenalectomized mice treated with pentothal sodium.

* We thank Dr. C. A. Winter of Merck Institute, Rahway, N. J., for supplying us with Cortone and Hydrocortone.

In the adrenalectomized groups treated with cortisone acetate or hydrocortisone acetate, anesthesia is not significantly longer than in the intact group—1.3 times ($25\% < p < 50\%$) and 1.1 times ($50\% < p$), respectively, but is significantly shorter than in the adrenalectomized non-treated group ($0.5\% < p < 1\%$). The difference between the cortisone acetate and hydrocortisone acetate treated adrenalectomized groups is not significant ($25\% < p < 50\%$). The anesthesia time of the adrenalectomized DOCA treated group is 4.0 times longer than that of the intact group ($p = 0.5\%$), but not significantly different from that of the adrenalectomized non-treated group ($50\% < p$). That of the adrenalectomized saline treated is 3.3 times longer than that of the intact ($p < 0.5\%$), but not significantly different from that of the adrenalectomized non-treated ($25\% < p < 50\%$).

Discussion. Prolongation of the duration of pentothal anesthesia in adrenalectomized mice is considered to have been caused mainly by the deficiency of glucocorticoids, because the anesthesia time almost returned to the normal level when the animals were treated with cortisone acetate or hydrocortisone acetate, but not with DOCA.

Winter(2) reported that cortisone but not DOCA antagonizes the effect of hexobarbital (Evipal) in normal mice. By the present experiment, it is shown that these steroids act in the same manner upon the effect of pentothal sodium in adrenalectomized mice.

Testing the effect of adrenocortical steroids on the electro-shock seizure threshold (EST) of rats, Woodbury(3) demonstrated that cortisone acetate was more potent in central stimulating action than hydrocortisone acetate. In the present experiment, however, no

significant difference was found between these 2 steroids at the dose levels employed. Further, Woodbury(4) has shown that chronic administration of diphenylhydantoin or phenobarbital with DOCA pellets caused an increase in the EST greater than the sum of the increases produced by the 2 drugs given separately. And so, it might be expected that treatment with DOCA would also prolong the anesthesia time in adrenalectomized mice. However, such an effect was not demonstrated.

Summary. The effects of the administration of cortisone acetate, hydrocortisone acetate and DOCA on the duration of pentothal anesthesia in adrenalectomized female mice were ascertained. Adrenalectomy induced a highly significant prolongation in the duration of pentothal anesthesia. Pretreatment of cortisone acetate and hydrocortisone acetate returned the anesthesia time to normal level in adrenalectomized mice, while DOCA and saline had no effect.

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Sulfate Fixation by Embryonic Chick Lung in Tissue Culture.* (20629)

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The authors have studied the synthesis of protein, nucleic acid, and collagen in chick embryonic tissue by a quantitative roller tube method(1-3). In view of the relationship of chondroitin to collagen, it was considered worth while to study the uptake of S^{35}-labeled sulfate under these conditions. Layton(4-7) has found a high rate of sulfate fixation after 45 hours' incubation with embryonic chick heart, skeletal muscle, liver, and bone in Tyrode's solution containing labeled sulfate. He suggested that the labeled sulfate was bound in the chondroitin sulfate of the connective tissue ground substance; cortisone added to the medium inhibited sulfate fixation by heart and skeletal muscle.

Methods. Roller tube cultures of 15-day chick lung were prepared as previously described(1) using 20-40 mg of tissue per tube. Tyrode's solution and 25% chick embryo extract were used as media. The medium was changed 3 times weekly, and incubation of the cultures were continued in embryo extract for as long as 6 weeks at 37°C. At the termination of incubation, the medium was poured off, the tissue was washed with water, and the tissue fragments were rubbed from the walls of the tubes in 5% trichloroacetic acid. The contents of 4-5 tubes were pooled, dehydrated, and delipidized. Details of the procedures have been described previously (1). In order to determine the incorporation of labeled sulfate into the tissue, sulfate§ was added to the nutrient medium in quantities of 0.002 millicuries per ml of medium. Uptake of the labeled sulfate was recorded as counts per minute per milligram‖ of the lipid-free

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§ The radioactive sulfate used in this investigation was supplied by the Oak Ridge National Laboratory on allocation by the U. S. Atomic Energy Commission.