TABLE II.
Infective Titer and Total Nitrogen Content of Virus Infected Tissue Suspensions Before and After Passage Through Ion Exchange Resin.

		Infective titer		Total nitrogen		
		Log dilution		Mg/ml		
	pH of resin supernatant	Before	After	Before	After	% reduction
	From chick embryo					
Japanese encephalitis—20%	7.8	6.6	6.6	0.96	.17	82
" " "	7.8	7.3	6.5	1.15	.25	78
" " "	7.8	6.4	6.5	0.80	.10	88
" " "	7.8	6.6	6.8	1.17	.24	79
" " "	7.8	7.5	7.4	1.10	.22	80
" " "	8.8	6.6	6.5	1.17	.34	71
Eastern equine encephalomyelitis—20%	8.8	9.1	8.5	1.24	.38	69
" " " "	7.6	9.0	8.6	1.17	.59	50
" " " "	7.6	9.6	9.5	1.12	.49	56
" " " "	7.6	8.6	8.5	1.28	.31	76
	From mouse brain					
Japanese encephalitis—10%	8.8	7.4	7.5	1.12	.15	87
Eastern equine encephalomyelitis—20%	7.6	9.2	7.9	2.22	.16	93
Western equine encephalomyelitis—10%	7.6	9.0	7.8	1.27	.47	63
Rabies—5%	7.0	6.8	5.6	0.65	.17	74
" 10%	6.8	7.2	6.6	1.26	.25	80

nitrogen content. These differences may have been a result of variations in the flow rate through the columns.

Summary. Ion exchange resin techniques offer a simple and effective method for the partial purification of the neurotropic viruses which have been studied.

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Immunological Studies of Embryo-propagated Hamster-adapted Newcastle Virus in Chickens. (17640)

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A California strain of Newcastle disease virus, No. 11,914, adapted to the Syrian hamster(1), has proved effective in the form of hamster brain suspension in immunizing chickens against the virulent unmodified virus of this California strain(2), and against virulent Connecticut and Colorado strains(3). These successful immunizations prompted further experiments to test the value of this hamster-adapted strain after one passage in 8-day-old embryonated eggs. Vaccine used for this experiment was prepared from the 73rd hamster passage. Fifteen 5-week-old Syrian hamsters (average weight 40 g) were injected intracerebrally with 0.05 cc of a 10%

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TABLE I.
Summary of Results Obtained with Living Hamster-Egg Newcastle Virus.

Vaccination 7/12/49					Symptoms & deaths		Challenge		Results	
Group	No. of birds	Vaccine	Amt in cc	Site	Typical	Atypical	Birds	Date	Died	Survived
1	60	Hamster-egg	.25	Base of wattle	5	4	51	8/8/49	0	51
1 RC	15	—	—	—	1	0	14	"	11	3
2	60	Hamster-egg	.01	Web of wing	2	0	58	"	2	56
2 RC	15	—	—	—	0	0	15	"	9	6

RC = Room control unvaccinated contact chickens.

brain suspension from infected hamsters of the above passage. Hamsters were sacrificed when typical symptoms of central nervous system involvement appeared in 48 to 72 hours. The brains of these hamsters were removed aseptically, ground with alundum, and diluted to a 10% suspension with physiological saline. Fifteen-hundredths cc of this suspension was injected into the allantoic sac of each of 124 8-day-old embryonated White Leghorn eggs. All eggs were incubated at 37.5° C. Fourteen eggs, dead after 24 hours incubation, were discarded. The allantoic and amniotic fluid were harvested and pooled from eggs that were dead in 48 to 96 hours. These eggs represented 69.09% of those alive after 24 hours. Later experiments showed that an increased volume of inoculum would cause a higher percentage of deaths at the proper interval. This harvested material titered $10^{-4.66}$ in eggs by the Reed and Muench calculation(4) and was used as the vaccine.

Healthy 6-week-old New Hampshire Red chickens, identified by wing bands, were used in the experiment. Hemagglutination-inhibition tests when performed with serum collected before vaccination were negative. Sixty chickens were injected in the base of the wattle with 0.25 cc of a 10% dilution of the virus-bearing allantoic fluid in physiological saline. Fifteen unvaccinated chickens were placed with the vaccinated birds as room contact controls. These 75 birds comprised Group I.

A second series of 60 chickens was vaccinated by the stick or wing-web method with concentrated allantoic fluid. Fifteen unvaccinated chickens were placed with these vaccinated birds as room contact controls. These 75 birds were designated as Group II. Careful observations of both groups were made daily. Results of the 27-day observation period before challenging are shown in Table I.

After 27 days birds of both groups were challenged in the base of the wattle with 0.5 cc of a 10^{-1} dilution of allantoic and amniotic fluid from a virulent California strain (No. 11,914) of Newcastle disease virus, which had been carried through 43 passages in embryonated chicken eggs. Normal susceptible birds of corresponding age were challenged at the same site of inoculation with the same material. The results obtained after challenge are shown in Table I. The challenge material titered 10^{-5} in 7-day-old embryonated chicken eggs and $10^{-4.47}$ in 6-week-old chickens as calculated by the method of Reed and Muench(4).

Discussion. The allantoic fluid from the hamster-egg passage, used as a vaccine, was pathogenic for chickens to the extent of 8.33% by subcutaneous inoculation, and 3.33% by the stick method. Of the normal room controls added to the vaccinated chickens before challenging, 3.33% contracted Newcastle disease from vaccine contact; and 68.96% of those remaining became infected when challenged with Newcastle disease virus. On challenge, no chickens of the group vaccinated subcutaneously showed evidence of Newcastle disease, while 3.44% of chickens in the stick method vaccinated group became infected.

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Summary. The immunizing value of a living vaccine prepared from the first hamster-egg passage was of a high order against challenge by injection. Although a slightly lower degree of protection was obtained, the vaccine is considerably less pathogenic when given by the stick or wing-web method of vaccination.

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Eosinopenia Due to Glucose Administration.* (17641)

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In recent years the physiology of the adrenal cortex has received enormous consideration because of its importance in maintaining homeostatic equilibrium under normal conditions as well as during periods of stress. It is generally considered that the activity of the adrenal cortex is under the influence of adrenocorticotrophic hormone which is liberated by the anterior pituitary. Forsham *et al.*(1,2) believe that the depression of the circulating eosinophiles of the blood is a sensitive physiological criterion of increased adrenal cortical activity. They have shown that 11-oxygenated corticosteroids, ACTH, and epinephrine(3) will decrease the eosinophile count in subjects with normally active adrenal cortical tissue, but that the response is absent when this tissue is functionally deficient. The eosinopenia, which is maximum about 4 hours after adrenal cortical stimulation, is accompanied by an increased renal excretion of sodium, chloride, potassium, uric acid, 17-ketosteroids, and 11-oxysteroids, which is further indication of increased adrenal cortical activity. Pincus *et al.*(4) have shown that oral administration of glucose in the rat produced a lymphopenia which was synchronous with the rise in the blood sugar. This was interpreted as evidence of increased adrenal cortical discharge. A significant correlation was noted between the magnitude of the hyperglycemia and the lymphopenic response. Jailer(5), using comparable doses of glucose in normal man, found that there was no such correlation and that the changes in the lymphocyte levels were within the limits of experimental error. Forsham *et al.*(1) have also cast doubt upon the validity of lymphopenia as an index of adrenal cortical discharge, since they demonstrated an overlap in this response in Addisonian and non-Addisonian patients following ACTH administration. The recent observations of Steeples(6) that adrenal cholesterol increased within 30 minutes following the oral administration of glucose in the rat, was interpreted as indicative of adrenal cortical inhibition. The variance in these results motivated us to evaluate the action of hyperglycemia in terms

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