the 6th and 7th anodally-migrating bands of the naphthyl acetate zymogram are, then, not specific for multiple sclerosis. However, relative diminution in activity of band 9 of the 9-10 doublet and the loss from plaque tissue of those enzyme activities against alpha-naphthyl propionate which are characteristic of normal white matter, are, to date, specific findings and seem of special interest. Because of the variations that occur in normal specimens, the significance of observed changes in cholinesterase zymograms of multiple sclerosis brain is difficult to evaluate.

No increase in esterase activity of multiple sclerosis tissue has been detected by densitometric analysis of zymograms or by quantitative assays of tissue homogenates(10). Experiments employing long-chain compounds (laurate and myristate esters of alpha- and beta-naphthol) have not disclosed the presence of a lipase activity(10). Presumably, if the abnormalities found in the esterase population of multiple sclerosis tissue have basic importance in the genesis of the disease they are related, in some way, to a defect in the normal metabolism of myelin.

Summary. Vertical starch-gel electrophoresis has been used for preparation of esterase zymograms of postmortem specimens of human brain. The specimens derived from 36 cases without a history of significant neurologic disability and 13 cases of multiple sclerosis. Certain esterases hydrolyzing alpha-naphthyl propionate were consistently demonstrable in, and characteristic of, zymograms of white matter from normal specimens. These were absent from zymograms of multiple sclerosis plaques. Alpha-naphthyl acetate zymograms of plaques and macroscopically-normal white matter from multiple sclerosis specimens showed frequently loss of, or diminution in activity of, the 6th, 7th and 9th anodally-migrating esterase bands, but alteration in bands 6 and 7 was not specific for multiple sclerosis. The cholinesterase activity of plaque tissue appeared to be diminished in comparison with that of macroscopically-uninvolved white matter from the same case, and one or more bands were absent in the plaque. The electrophoretic mobility of esterases of the plaque, and sometimes of grossly uninvolved multiple sclerosis white matter, appeared to be greater than that of normal specimens examined under identical conditions of electrophoresis.

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Myocardial Catecholamine Contents in Vitamin E and Sulfur-Deficient Chickens.* (28416)

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Changes in myocardial function have been reported in chickens made dystrophic through Vit. E deficient diets(1). The basis for this cardiac dysfunction is not clear. Since alterations in catecholamine content of the heart have been implicated in the pathogenesis of

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TABLE I. Dystrophy Producing Diet.*

	% by wt
Ingredients:	
Assay protein C-1	15.0
Gelatin	15.0
Chick salts A†	6.0
Tallow‡	10.0
dl-tryptophan	.30
dl-phenylalanine	.20
Choline chloride	.20
Vitamin mix S-35†	.50
" A (50,000 I.U./g)	.05
" D_3 (7,500 I.U./g)	.03
Alpha cel	1.00
Procaine penicillin G (50%)	.004
NF 180 (22% of active furazolidone)	.05
Glucose monohydrate	q.s. 100
Supplements:	
l-cystine	.40
Methylhydroxy analog of thiobutyrate	.30
dl-alpha tocopherol acetate	.01
Santoquin	.02

* Prepared by L. J. Machlin, Monsanto Chemical Corp., St. Louis, Mo.
† Ref. 3.
‡ Stabilized by addition of 0.2% Tenox 6.

other cardiac lesions(2), it seemed pertinent to compare the catecholamine content in the heart of dystrophic birds with the catecholamine content in the heart of normal birds.

Methods. Nine normal roosters, 9 normal hens, 6 dystrophic hens and 11 dystrophic roosters were used in this study. To develop the dystrophy in these birds a Vit. E and sulfur-deficient diet was prepared. The components of this diet are presented in Table I. A supplement of Vit. E, cystine and antioxidants was added to the same diet and fed to the normal birds. Quantities of the supplements are also presented in Table I. The dietary regimen was carried out for 8 weeks. At termination of the experiment the animals were anesthetized with thiopental sodium (20 mg/kg), the heart was excised, blotted free of blood and immediately placed on dry ice. Catecholamines were measured fluorometrically according to the method of Crout(4). Oxidations were carried out at pH 4.9 in order to express results as norepinephrine equivalents.

Results. The catecholamine content of the hearts of normal roosters ranged from .22 to .57 μg/g and averaged 0.40 $\pm$ .05 μg/g. The hearts of normal hens yielded contents ranging from 0.12 to 0.39 μg/g and averaged 0.22 $\pm$ .04 μg/g.

The catecholamine content of hearts of dystrophic roosters ranged from .31 μg/g to 1.18 μg/g and averaged .67 $\pm$ .09 μg/g; content of dystrophic hens ranged from .19 to .55 and averaged .32 $\pm$ .02 μg/g.

The difference between the mean values of the catecholamine contents of hearts of normal roosters and normal hens is statistically significant ($P < .02$). The difference between the means of the values obtained from dystrophic birds as compared to normal birds was statistically significant ($P < .05$). The data are summarized in Table II.

Discussion. Muscular dystrophy can be developed in chickens by dietary restrictions of sulfur and Vit. E(5). Such dystrophy in chickens is associated with a significant increase in catecholamine content of the heart as indicated by the above data. However, on the basis of this study it is not clear whether cystine deficiency, Vit. E deficiency or both are responsible for this increase. The relationship of this change to observed alterations in myocardial function has not been established. Premature ventricular contractions and elevations of S-T segments have been recorded in Vit. E deficiency(1). However, these changes are non-specific and may be demonstrated in many disease states.

The increase in the myocardial store of catecholamine need not necessarily reflect increase in activity of the sympathetic nervous system. Variation in binding at storage sites and impairment of release of the transmitter may underlie such changes.

Decrease in phosphorylase activity has been associated with Vit. E deficiency in the chicken (6), and in hereditary muscular dystrophy in the mouse(7). The alteration in activity is not associated with a change in ratio of active to inactive phosphorylase. This suggests that

TABLE II. Catecholamine Content of Hearts of Normal and Dystrophic Chickens (μg/g).

			Range	Mean $\pm$ S.E.
Normal	♂	(9)	.22– .57	.40 $\pm$ .05
	♀	(9)	.12– .39	.22 $\pm$ .04
Dystrophic	♂	(11)	.31–1.18	.67 $\pm$ .09
	♀	(6)	.19– .55	.35 $\pm$ .02

No. of chickens in parentheses.

the phosphorylase activation system is not impaired. Our data are consistent with this view, since the available activator, catecholamine, increased rather than decreased.

The significantly higher concentration of catecholamine content in the normal male of this species is also noteworthy. This is consistent with observations that cardiac output is greater in the rooster than in the hen under a variety of environmental circumstances(8). The predominant catecholamine in the heart of the normal chicken is norepinephrine, epinephrine constituting one-third of the total, or less(9).

Summary. Dystrophy in chickens due to Vit. E and sulfur deficiency is associated with an elevation in myocardial catecholamines. The catecholamine content of the heart of the normal rooster is significantly greater than that of the normal hen.

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Influence of Thyroid State on Positive Inotropic Effect of Ouabain on Isolated Rat Ventricle Strips.* (28417)

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While it is a common belief that heart failure accompanying disorders of thyroid gland function is relatively refractory to the effects of cardiac glycosides(1), there have been few systematic evaluations of this concept. Frye and Braunwald(2) observed an increased requirement for acetylstrophanthidin or digoxin in patients with chronic atrial fibrillation when treated with triiodothyronine, using slowing of the ventricular rate as an index of glycoside action.

Rosén and Moran(3) and Morrow *et al.* (4), have studied the effects of cardiac glycosides in hyper- and hypothyroid dogs. Both groups found ouabain to augment cardiac contractile force in all animals. Rosén and Moran noted a lesser effect of small doses of ouabain in hypothyroid dogs than in controls whereas in hyperthyroid dogs the response to low doses was similar to that of euthyroid dogs but the effect of high doses was depressed. Morrow *et al.*, observed a greater effect of ouabain on contractile force of hypothyroid dogs compared with normal and hyperthyroid animals.

The following experiments were designed in an attempt to evaluate the influence of altered levels of thyroid activity on the positive inotropic effect of ouabain on isolated ventricle strips of rats.

Methods. Male, Sprague-Dawley, albino rats (170-200 g) were divided into 3 groups. Group I (11 rats) were thyroidectomized (with concomitant removal of parathyroid glands). Group II (8 rats) received daily subcutaneous injections of sodium *dl*-thyroxine (20 μg/100 g). Group III consisted of 18 normal rats. Calcium lactate was added to the drinking water (1% w/v) of the thyroidectomized rats to prevent hypocalcemic tetany. Three weeks after thyroidectomy propylthiouracil was added to the diet (0.1% w/w). All animals were given water and food *ad libitum*.

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