is, however, of the same magnitude.

The reason for the greater delay of the first sound in basal as compared to apical ectopic beats remains speculative. When the ectopic focus is in the base of the ventricle, the impulse probably must proceed through the ventricular wall toward the apex and then through the wall of the other ventricle. However, if the ectopic focus is at the apex, it could travel simultaneously through both ventricular walls. In other words, the wave of depolarization would require a longer time to activate sufficient ventricular muscle mass to inaugurate contraction in basal ectopic beats. When auricular systole is absent the valves must be closed from the open position by ventricular contraction(3). The greater delay in the beginning of ventricular contraction in basal ectopic beats could thus explain the greater delay in onset of the first sound.

Summary. 1. Simultaneous electrocardiograms and phonocardiograms were recorded from 5 human subjects having ectopic ventricular systoles. Similar tracings were obtained from 5 dogs in which ectopic systoles were produced by induction shocks to the exposed heart during vagal arrest. The time interval between beginning of ventricular depolarization and onset of the first heart sound was measured for normal and for ectopic beats. The data were subjected to statistical analysis. 2. The beginning of the first heart sound was significantly delayed in ectopic ventricular contractions. The first sound occurred significantly later in ectopic beats arising from the ventricular base than in those from the apex. The probable reasons for these findings are discussed.

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Toxicity of Choline and Dimethylethanolamine in the Guinea Pig and the Rat.* (20978)

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The toxicity of choline in the rat has been established(1-3). The metabolism of choline in the liver is related to the activity of choline oxidase(4,5). This enzyme system has a very low activity or is entirely lacking in the liver of the guinea pig and thus choline deficiency cannot be produced by dietary means (6). Dimethylethanolamine, a precursor of choline(7), is not oxidized by liver choline oxidase(8). In view of these observations it was of interest to determine if there was a difference in the toxicity of choline or dimethylethanolamine in the guinea pig and rat.

Methods. Male albino rats (100-110 g) and guinea pigs (200-300 g) were divided into two groups. Group 1 received intraperitoneally 45 mg of dimethylethanolamine or choline (base) per 100 g of body weight in 1 cc and Group 2 received 60 mg. Animals which lived longer than 30 minutes in most cases survived.

Results. From the results in Table I it was apparent that there was no difference in the toxicity of choline in the guinea pig or

TABLE I. Toxicity of Choline and Dimethylethanolamine in the Guinea Pig and Rat.

mg (base) /100 g of body wt		Rats		Guinea pigs	
		No. of animals	% mortality	No. of animals	% mortality
45	Choline	14	29	45	20
	DME*	24	75	28	71
60	Choline	20	60	39	74
	DME	25	88	22	77

* Dimethylethanolamine.

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rat. The absence of choline oxidase in the liver of the guinea pig does not increase the toxicity of choline. Dimethylethanolamine, which is not metabolized by choline oxidase in the liver, appears to be more toxic than choline in both animals. Dimethylethanolamine is metabolized much slower than choline, for it can be detected in the tissues of rats following the administration of large quantities(8). On the other hand, after similar doses of choline were given, neither free nor combined dimethylethanolamine could be detected in the tissues of the rat. This difference in metabolism of dimethylethanolamine may help account for its increased toxicity.

Summary. 1. The toxicity of choline and dimethylethanolamine in the guinea pig and the rat has been compared. 2. There is very little difference in the toxicity of choline or dimethylethanolamine in the guinea pigs and rats. However, dimethylethanolamine appears to be the more toxic.

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Effect of ACTH on Distribution of Plasma Proteins of Patients with Pulmonary Tuberculosis. (20979)

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(Introduced by Smith Freeman.)

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Vaughan and coworkers(1) have shown that both ACTH and cortisone therapy depress fibrinogen and gamma globulin levels in the blood of patients with active rheumatoid arthritis and scleroderma. Reiner(2) likewise has shown that these agents increase albumin concentration, but decrease gamma globulin in sera of patients with *disseminated lupus erythematosus*. Studies reported in this communication describe the changes which occur in the electrophoretic pattern of the plasma proteins of tuberculous patients following administration of ACTH. The results will be shown to be similar to that described in(1,2).

Material and methods. Clinical material. Heparinized plasma samples were obtained from 11 patients hospitalized for pulmonary tuberculosis. These samples were generally obtained before, at 2-week intervals during, and after treatment with ACTH. ACTH was generally administered in divided doses intramuscularly each 6 hours. Seventy-five to 100 mg were generally given at onset of treatment, and then the amount was gradually tapered to 20 mg daily before being discontinued.

Electrophoretic fractionation. Diagrams were obtained under nearly the same experimental conditions in order to make comparisons possible. The plasma was diluted 1-6 with veronal buffer solution, pH 8.6, ionic strength 0.1; electrophoresis was conducted in the standard 11 ml cells of the Klett apparatus. Ten milliamperes of current were passed until separations were satisfactory; this was generally about 3 hours. Enlarged tracings were made of anode pattern photographs and per cent of total plasma proteins deter-