tion, genetically homogeneous mice of the dba strain (line 1) were used. These are the result of more than 50 generations of brother × sister matings and are known to have a quantitatively predictable susceptibility to sound-induced seizures at a given age, when subjected for 2 min to a noise produced by a doorbell mounted on a circular galvanized iron washtub (*2, 3, 5*). The stimulus intensity is adjusted to approximately 100 decibels with a transformer. When a neutral solution of L-glutamic acid is administered to these animals for 8 to 14 days prior to exposure to audio stimulation, a statistically significant decrease in fatalities resulting from the audiogenic seizures occurs. Under standardized conditions, the magnitude of this decrease may be used to compare the relative effectiveness of various doses alone or with other substances or combinations of substances.

The glutamic acid was administered by subcutaneous injection at a concentration of 200 mg/ml. Each animal received 0.1 ml per 10 g body weight. Solutions were prepared in the manner described by Marx (*7*). The mice were tested for seizure susceptibility once daily, beginning with the 30th day of age. On these days, the glutamic acid was injected 30 to 45 min prior to exposure to the seizure-producing situation. Thirty-three animals (31 males and 2 females) were used in the control series. Fifty animals (21 males and 29 females) were used in the series receiving glutamic acid. The animals were fed a diet of Fox Chow Checkers and had access to food at all times. The data were tabulated as cumulative percentages for four trials.

A comparison of the treated and control groups reveals that the seizure incidence is not affected by the administration of glutamic acid, but the proportion of fatalities in the treated group is decreased by 18% ($t = 2.95$). This effect occurs primarily in males, where the decrease in fatalities below that found in the controls is 26% ($t = 3.53$). That for the females is 11%, but is not statistically significant ($t = 1.78$). Previous work has shown that untreated males are approximately 10% more susceptible to sound-induced seizures than are females (*3*). We therefore recommend that dba line 1 males should be used as the standard test animals.

These and other data will be presented and evaluated in greater detail in a forthcoming publication (*4*).

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The Recombination Coefficient for the F Layer[1]

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It seems probable that the time rate of change of maximum electron density does not give a true picture of the actual variations in ionization occurring in the F layer (*4*). We carried through an investigation to determine whether or not the total electron content of an ionospheric layer can be used for the calculation of α_T, the recombination coefficient for the region as a whole.

The equation

$$\frac{dN}{dt} = q - \alpha N^2 \qquad (1)$$

states that the rate of change of the electron density at any level in the layer is equal to the number of electrons per cubic centimeter produced by any means less the number lost by recombination processes. We must look into the equation when $\frac{dN}{dt}$ is the rate of change of total electron content in a vertical column of unit cross-sectional area of height τ of the layer, and q is the total production of electrons in this volume. It has been shown that total electron content N_T of the layer below the level of maximum electron density can be computed (*3*) from:

$$N_T = 2/3\tau N_M \qquad (2)$$

As far as q is concerned, we must assume for the present that the sun's ultraviolet light is the only agency responsible for the production of electrons in the layer. Considering only the F_2 layer, the number of electrons recombining with positive ions is proportional to the possible number of collisions of an electron and a positive ion, or

$$R \sim N_e N_+ \qquad (3)$$

and α can be thought of as a proportionality factor which indicates what proportion of possible recombinations will probably take place. Since we lack precise knowledge of existing conditions, it is convenient to consider that the number of electrons is equal to the number of positive ions, or $R = \alpha N^2$.

For a layer in which the electron density increases with height according to a parabolic law, we have the equation given by Appleton (*1, 2*),

$$N_h = N_M \left[1 - \left(\frac{h_M - h}{h_M - h_m}\right)^2\right] \qquad (4)$$

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where N_h is the electron density at any height h within the parabolic layer, N_M is the maximum electron density at height h_M, and h_m is the true height of the bottom of the layer. The possible number of combinations of electrons with positive ions at any height within the layer (assuming equal number of electrons and positive ions) is

$$N_h^2 = N_M^2\left[1 - 2\left(\frac{h_M - h}{h_M - h_m}\right)^2 + \left(\frac{h_M - h}{h_M - h_m}\right)^4\right] \qquad (5)$$

This must be summed for the half layer from the bottom of the layer to the point where N is maximum, that is, from h_m to h_M. Hence, we have

$$\int_{h_m}^{h_M} N_h^2 dh = \int_{h_m}^{h_M} N_M^2\left[1 - 2\left(\frac{h_M - h}{h_M - h_m}\right)^2 + \left(\frac{h_M - h}{h_M - h_m}\right)^4\right] dh = \frac{8}{15}\,\tau N_M^2 \qquad (6)$$

remembering that $h_M - h_m = \tau$.

The original equation takes the form

$$\frac{dN_T}{dt} = q_T - \alpha N_M^2 \frac{8\tau}{15} \qquad (7)$$

The units of α are $cm^3\ sec^{-1}$ and are the same as with the more simple form of the equation. The α given by this equation is not an α at any particular height but rather an α that describes recombination for the region as a whole. The semithickness τ of the ionospheric layer can be reduced from the original records (*4*) by the method of Booker and Seaton (*3*).

TABLE 1

	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Mean
$\alpha_M \times 10^{10}$ ...	3.24	4.87	7.00	5.99	3.48	1.97	4.42
$\alpha_T \times 10^{10}$ ...	4.54	6.77	11.93	8.37	5.46	2.37	6.57

The recombination coefficients at the level of maximum electron density, α_M, and for the layer as a whole, α_T, were computed from night ionospheric data observed at College, Alaska. During the night q vanished from the equation. The monthly mean data for the winter months (October through March) of 1948–49 were used for the calculations. The mean results are tabulated in Table 1.

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Lipid Interrelationship in Health and in Coronary Artery Disease

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It has been demonstrated that the serum cholesterol level is elevated in atherosclerosis and coronary artery disease (*4, 9*). Recently it has been suggested that an inverse correlation exists between serum cholesterol and time of appearance of atherosclerosis (*8*). Thus, individuals experiencing such diseases at an early age would be expected to show highly elevated serum cholesterol levels (over 300 mg %). This relationship has been under investigation for the past three years by the Coronary Research Project at the Massachusetts General Hospital.

In keeping with other reports, the present study found that serum cholesterol was considerably higher in males who had experienced myocardial infarction prior to the age of 40 than it was in healthy, active males of comparable age, the means being 286 ± 6.6 mg/100 ml blood and 224 ± 3.5 mg/100 ml blood, respectively, the difference exceeding one standard deviation of the normal group (*6*). Even though individual thresholds may exist, there was no evidence of a threshold "value" of serum cholesterol in the coronary disease group, the distribution being essentially continuous.

On further analysis of other serum lipids, it was found that the normal interrelationships of these lipids were altered in coronary artery disease as reported recently (*1, 7*). Since these observations also indicate that relationships rather than absolute serum levels are important, this communication includes a study of such relationships.

In this study, blood samples were taken from 243 individuals; 97 were males who had experienced myocardial infarction prior to the age of 40, and 146 were healthy, active working males comparable in age and other variables. Serum cholesterol determinations were made using the method of Bloor (*2*), while serum phospholipid determinations were made by the Fiske and Subbarow method (*5*). The two groups of individuals are referred to as the coronary artery disease group and the control group hereafter.

Results giving the mean values, standard deviations, and standard errors of the two lipids, and their ratios are summarized in Table 1.

The serum cholesterol and serum phospholipids means

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