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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TABLE 1

SERUM CHOLESTEROL, SERUM PHOSPHOLIPIDS, AND CHOLESTEROL/PHOSPHOLIPID RATIO IN THE CONTROL GROUP AND THE CORONARY ARTERY DISEASE GROUP

	Serum cholesterol*		Serum phospholipids*		Ratio: $\frac{\text{cholesterol}}{\text{phospholipids}}$	
	control group	coronary disease group	control group	coronary disease group	control	coronary
Number	146	97	146	61	146	60
Range	148–332	167–490	215–415	195–414	52.0–104.0	60.4–110.8
Mean ± S.E.†	224.4 ± 3.5	286.5 ± 6.6	299.3 ± 3.3	316.4 ± 6.6	75.1 ± .92	89.4 ± 2.04
S.D.‡	42.6	64.9	40.2	52.2	10.9	15.9

* Figures represent mg/100 ml blood. Serum phospholipids are expressed as licithin (25 × lipid phosphorous).
† S.E. = Standard error.
‡ S.D. = Standard deviation.

were both significantly higher in the coronary disease group, while the ratio of cholesterol/phospholipids was also significantly higher in the coronary disease group. Proportionately, the cholesterol level had risen higher in the coronary artery disease group than in the control group. It is reasonable to assume that if serum phospholipids were to rise proportionally to the serum cholesterol, the ratio would remain unchanged. Thus, it is obvious that the ratio is increased in the coronary disease group due to the lack of a proportional rise in serum phospholipids.

The correlations between the two lipids and between age and each of the lipids were determined for both the coronary disease group and the control group, as shown in Table 2.

Thus there is, in the normal control group, a moderate correlation between the two lipids, and low to moderate correlations between age and each of the two lipids studied. However, in all three correlations the coronary disease group is significantly lower, with the correlations between age and cholesterol and age and phospholipids no longer significantly different from zero. This is further proof that the interrelationship between the lipids

TABLE 2

INTERCORRELATIONS BETWEEN CHOLESTEROL, PHOSPHOLIPIDS, AND AGE

Coefficient of correlation between:	Controls	Coronary disease group
Cholesterol and phospholipids	+.66 ± .05	+.51 ± .09
Age and cholesterol	+.30 ± .08	+.16 ± .10*
Age and phospholipids	+.43 ± .07	+.20 ± .12*

* Not significantly different from zero.

is disturbed in coronary artery disease, while the normal age trend is largely masked if not disrupted.

Because of the number of intercorrelating variables at work in the two groups, partial correlations were calculated (Table 3) in order to study the effects of two intercorrelating variables, with the third held constant. Accordingly, each of the three variables was eliminated in turn.

With age constant, the correlation between cholesterol and phospholipids remains moderately high in both groups, although it is lower in the coronary disease group. The serum phospholipids continue to show an age correlation in the normal group, but it is an insignificant one in the coronary disease group. Serum cholesterol, on the other hand, failed to show age changes with serum phospholipids held constant in both groups. Thus, again the normal age increments are absent in the coronary disease group, while phospholipids seem to mediate the normal cholesterol-age relationship in some way in the normal.

It is therefore reasonable to suggest that the phospholipids play a role in the normal age changes in serum cholesterol; the failure to find such cholesterol changes in coronary artery disease may merely reflect the basic difference in amount and in proportions of the phospholipids in this disease group.

Peters and Man (*10*) suggested that the interrelationships of the serum lipids are far more important than the consideration of any single lipid. The protective ac-

TABLE 3

PARTIAL CORRELATIONS: AGE, CHOLESTEROL, AND PHOSPHOLIPIDS

Partial correlation	Control group	Coronary disease group
Cholesterol and phospholipids, age constant	+.62 ± .05	+.50 ± .10
Cholesterol and age, phospholipids constant	+.05 ± .08*	+.07 ± .10*
Phospholipids and age, cholesterol constant	+.32 ± .07	+.14 ± .13†

* Not significantly different from zero.
† Not significant.

tion of serum phospholipids in experimentally produced atherosclerosis has recently been demonstrated, and its method of protection has been speculatively described as being in the nature of a colloid stabilizer (*1, 7*). This is in keeping with Browder's earlier observation of the antagonistic effect of serum cholesterol and serum phospholipids in biological reactions (*3*).

From the results of this general study, it is known

that the serum cholesterol in individuals with coronary artery disease reaches inordinately high levels in many (but not all) instances. The serum phospholipids, on the other hand, do not keep pace with this rise in serum cholesterol. Hence it is believed that one of the factors favoring the deposition of cholesterol in the intima is enhanced because of the lack of a colloid stabilizer which may be reflected by the proportion of phospholipids in the serum. Conversely, in the normal individual it may be suggested that the colloid stability of cholesterol is unchanged because the rise of serum phospholipids is proportional to the rise in serum cholesterol.

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Inhibition of Anaphylaxis in Guinea Pigs by D-Catechin

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The use of antihistaminic agents has proved to be an effective adjunct in the treatment of various allergenic reactions by virtue of their antagonistic activity toward preformed histamine. Recently, Martin *et al.* (*4*) demonstrated *in vitro* the inhibitory effects of vitamin P compounds in histidine decarboxylase. This enzyme, present in animal tissues, is capable of forming histamine from histidine (*3*, *6*, *7*). Preliminary tests *in vivo* (*1*) also indicated that these compounds are active. Their activity might be directed toward inhibition of the formation of histamine. Inhibition of histamine formation in the body seems a rational approach to the treatment of allergies.

In this study, 14 guinea pigs were sensitized in the manner described by Raiman *et al.* (*5*). Half of the animals received 2 mg of D-catechin, an aglycone flavonoid, intraperitoneally daily for 19 days. The remaining animals were not treated and served as controls. At the end of the 19-day period each animal was shocked by an intracardial injection of 0.1–0.5 ml of fresh normal horse serum.

The animals receiving D-catechin exhibited no anaphylactic reactions. The control animals exhibited typical reactions followed by extreme dyspnea and finally death due to asphyxia. The complete reaction lasted approximately 5 min.

Four additional guinea pigs, which had received daily doses of D-catechin for 1 week, were injected intracardially with 0.1 mg of histamine diphosphate. These animals died several minutes later with typical shock symptoms.

The dead control animals and the animals from the histamine group were autopsied. No significant difference in gross pathology could be observed. The predominating characteristic in both groups of animals was the constriction of the bronchiolar muscles. Each animal showed varying degrees of pulmonary edema and hyperemia.

These studies show that D-catechin protects guinea pigs from anaphylactic reactions but not from histamine shock. It appears reasonable to believe that this protective activity might be attributed to an actual inhibition of histidine decarboxylase. This reaction would tend to prevent the formation of histamine, which is an important factor in the anaphylactic syndrome (*2*).

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Regeneration of the Shoot Apex of *Lupinus albus* after Operations upon the Central Initials

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In attempting to transplant the central portion of the shoot apex, it was noted that the uninjured portions of the original meristem regenerated into one or two normal apices. This regeneration was similar to that described by Linsbauer (*2*) and Pilkington (*3*) after different operations. The heavy black line in Fig. 1 shows the position of the cuts made in the shoot apex. The sector (*S*) was either transplanted to another apex, replaced in the same or reversed orientation in the original apex, or excised. Usually the sector died when it was left in an apex (Figs. 2, 3, 4, 5), and its shrunken remains marked the site of the operation. When the sectors were re-