Remarkably, in this case, the high LDH activity was accompanied by shifts in the isoenzyme pattern in similar directions for both serum and BALF. The fact that this shift in serum resembles that in BALF suggests that the major source of the serum LDH was the lung. The source of the LDH activity in the lung may be inflammatory cells, such as alveolar macrophages rapidly recruited to rid the lung of the phospholipid[9]. This case report highlights the promising role of serum LDH activity — a simple potential marker of disease activity — in monitoring and follow-up of drug-induced pneumomtls caused by amiodarone. Moreover, this determinant seems to be a sensitive marker to predict such reaction. An isolated increase of serum LDH indicates the necessity to discontinue amiodarone.

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Arginine consumption in coronary disease

We wish to make the following comments on the issue of the significance of arginine consumption in preventing coronary disease.

The importance of diet in the secondary prevention of coronary disease was recently reiterated by de Lorgeril and coworkers[3]. In their study, coronary patients of Lyon ate the Mediterranean diet consumed by the rural population of Crete as determined by the seven countries study[2]. The 76% reduction in clinical complications of coronary disease reported by the authors exceeds the benefit accrued by the most aggressive pharmacological lipid lowering intervention and suggests additional contributing factors. By using tables on amino acid content of foods[3] we determined the daily arginine intake of the study group prior to the diet administration and following its application and arrived at 3-5 g and 7 g respectively.

We believe that by doubling arginine consumption, nitric oxide availability may have increased and endothelial dysfunction may have been corrected in these coronary patients. This would facilitate vasodilatation and fibrinolysis and mitigate blood coagulation, adhesion of leucocytes and platelets as well as smooth muscle cell proliferation.

Further more the work of Kromhout and coworkers enables us to examine whether arginine has a role to play in primary prevention of heart disease. Kromhout in a number of publications[5-7] correlated several dietary factors consumed by the original 16 cohorts of the seven countries study with their 25 years coronary heart disease mortality. Extending this correlation to arginine by using the tables on amino acid content in food[3] would show whether consumption of arginine by healthy middle-aged men is related to their long-term coronary heart disease mortality.

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An association of an antibody against *Chlamydia pneumoniae* and coronary heart disease observed in Japan

*Chlamydia pneumoniae* is an important cause of acute respiratory illness, including pharyngitis, bronchitis and pneumonia[1]. However, there has been accumulating evidence implicating *C. pneumoniae* in atherosclerosis[2-9]. Saikku et al.[2] first reported on an antibody against *C. pneumoniae* and coronary heart disease (CHD) in 1988. Subsequently, Short et al.[3] and Kuo et al.[4] detected *C. pneumoniae* in coronary artery atherosclerotic plaques by immunocytochemistry, polymerase chain reaction and electron microscopy. Their findings have been confirmed by other investigators worldwide. We have also investigated the association of *C. pneumoniae* antibody and angiographically diagnosed CHD in Japan.

The study was conducted in four separate hospitals in Okayama, Osaka and Shizuoka, Japan between April 1993 and December 1994. There were 160 patients with CHD (34-81 years of age, mean 60-0 years; 115 males and 45 females). Cases were defined as patients who had at least one coronary artery lesion occupying at least 50% of the luminal diameter by angiography. One hundred and fourteen patients had myocardial infarction as defined by ECG and angiography. Controls who were matched for age and sex were enrolled from the patients attending the same hospitals. The criteria for inclusion were absence of signs and symptoms of CHD, as judged by a negative history and a normal resting ECG. Informed consent was obtained from all subjects. *C. pneumoniae* IgG and IgA antibodies were measured by the microimmunofluorescence (MIF) test[1] using a Japanese isolate KK-pn15 as antigen. The serologic criteria for a positive test was a titre of greater than or equal to 1:16 for IgG or 1:8 for IgA. Logistic regression was used for statistical analysis.

The odds ratios (ORs) were 2.1 (95% confidence interval [CI], 1.2 to 3.9) for IgG and 2.5 (95% CI, 1.7 to 4.3) for IgA. After adjustment for other cardiovascular risk factors of age, hypertension, diabetes, cigarette smoking and serum cholesterol, the ORs were essentially unchanged at 2.2 (95% CI, 1.2 to 4.1) for IgG and 2.7 (95% CI, 1.7 to 4.4) for IgA. The adjusted ORs were greater for patients with IgG titres of greater than or equal to 1:64 and IgA titres of greater than or equal to 1:32, i.e., 4.5 (95% CI, 2.2 to 9.1) and 6.1 (95% CI, 2.4 to 15.7), respectively. The geometric mean titres of IgG and IgA were significantly higher in patients with CHD than controls (39.2 vs 20.9 for IgG, *P* = 0.0001 and 12.6 vs 6.2 for IgA, *P* = 0.0001) by the Mann-Whitney U tests.

This study confirmed the observations of an association between antibody against *C. pneumoniae* and CHD in Western nations is also present in Japan. Our results are comparable to the previous seroepidemiological studies reporting ORs of 2.0 or greater[2,5-9].

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