Blocked Arteries: Clean Them Out Naturally

The Role of Nitric Oxide in Preventing Chest Pain

A chemical in the blood called nitric oxide is essential to the ability of blood vessels to relax. Not to be confused with nitrous oxide (“laughing gas”), nitric oxide is a critical relaxing factor that is made by the endothelial cells lining our blood vessels. However, when cholesterol levels rise, the blood vessels do not relax sufficiently in response to nitric oxide. Changes in blood cholesterol levels can make a significant difference in the amount of relaxation in a short period of time. Recently, researchers caused patients' cholesterol levels to rise by stopping their cholesterol-lowering drugs. Within two weeks there was a significant drop in responsiveness to the effects of nitric oxide, reducing relaxation.

Why would high cholesterol levels interfere with the effects of nitric oxide? Recent lab work provides one reason for this association. Researchers have now documented that high blood cholesterol levels cause the destruction of nitric oxide. However, due to the rapid destruction of nitric oxide, those cells generally run out of L-arginine, a crucial raw material that is essential for making nitric oxide. A recent study demonstrated that a significant cholesterol drop will improve stress EKG test scores (showing improved blood flow to the heart) in four weeks. In harmony with these relationships, scientists have found that giving additional L-arginine helps restore normal artery relaxation even if someone has high cholesterol levels. L-arginine just happens to be found in abundance in a vegetarian diet, but is sparse in meat and dairy products. Tabulation of the arginine content of foods is shown in Figure 17: Arginine Content of Food.

Note that meat and dairy products fall far short of arginine in comparison to beans, nuts, and seeds, which have 3 to 35 times as much.

Consequently, the rapid angina improvement in the Lifestyle Heart Trial may be due to at least two separate effects. First, reduced cholesterol levels may help to restore normal blood vessel relaxation mechanisms. Second, an increase in dietary arginine provided by the vegetarian diet will further help to relax arteries from the outset, likely even before blood cholesterol levels have dropped significantly.

Reduction of Red Blood Cell "Stickiness" Reduces Chest Pain

There is a third explanation for the rather dramatic short-term angina reduction occurring with cholesterol-lowering diets. A reduction in the tendency for red blood cells to stick together, or blood "fluidity," was observed in a study by German researchers. This is another study of the impact of lifestyle changes on heart disease progression. Dr. Gerhard Schuler and his associates made some
of the most detailed measurements of any of the studies regarding the regression of artery blockages.\textsuperscript{41} Using regular physical exercise and a diet that averaged 26 percent fat and 135 mg of cholesterol, (better than the best "national cholesterol education diet") they helped 30 percent of 56 study participants attain regression. Only four percent of a second group achieved regression without the lifestyle changes.

Of interest to the angina question, the researchers measured the tendency for red blood cells to stick together (called the "erythrocyte aggregation rate"). Stickier red blood cells tend to clump and interfere with optimal blood flow to the heart muscle, which can increase angina. Even on this less-than-optimal lifestyle program, the red blood cells' tendency to stick together decreased by a highly significant percentage. This evidence thus provides another reason why we would expect angina to decrease on a better lifestyle program. One only wonders how profound the changes would have been if a zero cholesterol, total vegetarian diet had been adopted.

A summary of the three possible reasons for rapid angina reductions that occur with cholesterol-lowering diets is shown in Figure 18: Reasons for Rapid Angina Reductions with Lifestyle Changes.

References

37 Harrison DG, Ohara Y. Physiologic consequences of increased vascular oxidant stresses in hypercholesterolemia and atherosclerosis: implications for impaired vasomotion. Am J Cardiol 1995 Feb 23;75(6):75B-81B.


