

ARTERIOSCLEROSIS

ATHEROSCLEROSIS

by Ronald L. Myers, CNC

I choose to group these together because over my career in this field I have observed many doctors using these terms almost "interchangeably". This is not hard to understand, as these conditions tend to occur simultaneously. In fact, current diagnostic thinking considers one (atherosclerosis) to be a form of the other (arteriosclerosis). From a nutritional standpoint, the treatment for both of these conditions is the same.

Before beginning any nutritional program in patients with these conditions, **ALWAYS** insure biliary stasis is **NOT** present. Clinically I have been able to demonstrate hypochlorhydria and biliary problems in most patients presenting with the above conditions. Resolving the patient's biliary dysfunction will, in most cases, speed the resolution of the blood vascular condition as well.

LABORATORY INDICATORS:

| | | |
|-----------------|----|-----------------------------|
| CHOLESTEROL | -- | INCREASED |
| TRIGLYCERIDES | -- | INCREASED (usually > chol.) |
| HDL | -- | DECREASED |
| SERUM URIC ACID | -- | INCREASED |
| HOMOCYSTEINE | -- | INCREASED (serum or urine) |

TREATMENT:

BILIARY DYSFUNCTION --

Hydrozyme 2 tablets with each meal. Provides HCl, pancreatin, B6.

Beta TCP 2 tablets before each meal. Provides betaine, Taurine, vit C, pancreatic lipase.

I **ALWAYS** recommend using Hydrozyme in the treatment of biliary problems because the G.I system is a pH regulated system. From a purely physical point of view, the beginning of that regulation is in the stomach. For the hormones of digestion (cholecystokinin, secretin) to function adequately, the chyme entering the pylorus must be pH 5.0 or less. This may not be the case if the patient is hypochlorhydric, which a high percentage of them are. Beta TCP will resolve biliary dysfunction, temporarily, but if we do not address the probable cause, we have applied a temporary band-aid. Hydrozyme will address the probable cause of the dysfunction. I encourage you not to stop there. Ordering a serum gastrin for the patient will allow you to assess the patient's potential to produce hydrochloric acid. If that number is less than 45, the patient is probably not going to produce HCl at optimum levels, indicating a need for HCl supplementation long term. If the serum gastrin number is between 50 and 100, the patient has the physiological ability to produce HCl at optimum levels, the question then is; why aren't they? At this point, we need to assess those factors that we know are essential for the patient to produce hydrochloric acid, they are WATER, sodium, *chloride*, thiamine and zinc. If the patient is presenting with hypochlorhydria, and their serum gastrin indicates the ability to produce adequate HCl; begin by assessing how much water they drink each day. Let's remember, it is **HYDROchloric acid**. Next, let's make sure they are not on a NO or LOW salt diet and that they are using **REAL** salt

(like Hains Solar Dried Sea Salt), not some form of sodium silico aluminate (Morton's salt). The Zinc Taste Test will allow you to assess zinc need; and the Anion Gap (inc.) and CO₂ (dec.) on blood chemistry will confirm or rule-out thiamine need.

ARTERIOSCLEROSIS/ATHEROSCLEROSIS--

PorphyraZyme 4 tablets 3 times daily on an empty stomach. Chelates divalent metals.

Intenzyme Forte 8 tablets on arising and at bedtime. Provides live proteolytic enzymes to facilitate plaque breakdown.

Flax Seed Oil 2 capsules with each meal. Provides essential fats as Omega 3, 6, and 9.

Phosphatidylcholine 3 capsules with each meal.

BioCardiozyme Forte 2 tablets with each meal.

The treatment of the above mentioned conditions would not be complete without an assessment of predisposing factors.

Genetic predisposition

Diet--high in refined carbos, hydrogenated oils, alcohol, red meat.

Endocrine dysfunction (thyroid hypo-function most common)

Homocystine (need for B6, B12, betaine [trimethylglycine], or folic acid).

Carbohydrate handling problem (diabetes, Syndrome X, etc).

Every year, more and more control studies are showing the effectiveness of nutritional means of treating this condition.

CITATIONS:

"Dietary Cholesterol and the Optimal Diet For Reducing Risk of Atherosclerosis", McNamara, Donald, J., Ph.D., Canadian Journal of Cardiology, October, 1995;(Suppl.G):113G-126G. In evaluating more than 30 years of 128 cholesterol-feeding studies in more than 2,750 patients, researchers found that, for the majority of individuals, modest changes in dietary cholesterol have little effect on plasma lipoprotein concentrations.

"Potential Role of Raising Dietary Protein Intake For Reducing Risk of Atherosclerosis", Wolfe, Bernard, M., et al, Canadian Journal of Cardiology, October, 1995;11(Suppl. G.):127G-130G. Increasing HDL cholesterol and lowering cholesterol and triglycerides is facilitated by increasing protein consumption at the expense of sugar and starch.

"Homocysteine and Coronary Atherosclerosis," Mayer, Ellen, L., M.D., et al, Journal of the American College of Cardiology, March 1, 1996;27(3):517-527. Elevated homocysteine may lead to atherosclerosis due to its effects on platelets, clotting factors and endothelium. Homocysteine degradation involves transsulfuration and re-methylation by the enzymes cystathionine-B-synthase and methylene tetrahydrofolate reductase. Folic acid, vitamin B6 and vitamin B12 are cofactors for these enzymes.

"Relationships Between Nutrient Intake and Progression/Regression of Coronary Atherosclerosis as Assessed by Serial Quantitative Angiography", Watts, Gerald, F., M.D., et al, Canadian Journal of Cardiology, 1995;11(Suppl. G.):110G-114G. The intake of trans fatty acids was directly related to the atherosclerotic progression.

"Tocotrienols Positively Affect Atherosclerosis," The Nutrition Report, February, 1996;14/"Antioxidant Effects of Tocotrienols in Patients With Hyperlipidemia and Carotid Stenosis," Tomeo, A., et al, Lipids, 1995;30(12):1179-1183.

"Trace Metal Analysis in the Aorta With and Without Atherosclerotic Lesions," Vijaya, I., et al, Trace Elements and Electrolytes, 1995;12(4):200-202.

"Homocysteine and Coronary Atherosclerosis," Mayer, Ellen, L., M.D., et al, Journal of the American College of Cardiology, 1996;27:517-527. This is an extensive review article on the role of homocysteine and the vitamins B6, B12 and folic acid and possibly betaine and choline in reducing homocysteine levels.

"Uric Acid and Coronary Heart Disease Risk: Evidence for a Role of Uric Acid in the Obesity-Insulin Resistance Syndrome: The Normative Aging Study", Lee, Jerry, et al, American Journal of Epidemiology, 1995;142(3):288-294. This data suggests that serum uric acid may be involved in the obesity-insulin resistant syndrome, which in turn, may explain the relationship of serum uric acid levels to coronary atherosclerosis.

"Oral Chelation is Here: New Oral Chelation Agents are Challenging Desferrioxamine", Kontoghiorghes, George J., British Medical Journal, November 23, 1991;303:1279-1280.

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