Article 3*

The Non-Medicated Life:
A New Laboratory Test to Assess Heart Attack Risk

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This is the third in a series on optimal diet and lifestyle to help prevent and treat heart disease. Subsequent articles will include information on national guideline recommendations, the newest lab tests to assess risk, specific dietary practices shown to reduce risk, as well as the evidence for the use of specific vitamins and supplements. Any planned change in diet, exercise or treatment should be discussed with and approved by your personal physician before implementation. Consultation with a registered dietitian is strongly advised.

Optimal diet and lifestyle may naturally accomplish for most individuals many, if not most, of the benefits of medication. As the May Health column described, individuals may determine the proper mix of medicine, diet and lifestyle by first knowing the national guideline cholesterol targets for heart attack and stroke reduction and then empirically trying to reach those targets with the mix that makes most sense to them. Beyond the usual testing, there is a new inexpensive blood test which may further aid individuals in determining their risk for heart attack. It is called high sensitivity C reactive protein (hs-CRP). Moreover, optimal diet and lifestyle may reduce risk identified by this new test.

To understand the benefits of the new test one should first have some understanding of what causes heart attacks and strokes. Over the last several years, there has been increasing evidence that heart attacks and strokes may result from inflammation in arteries caused, in part, by cholesterol deposits in the walls of the arteries. Researchers believe that the mechanism of a heart attack or stroke is the disruption of a cholesterol deposit or plaque by the inflammatory response the body mounts against the plaque.

In this scenario, arteries may be likened to tubes carrying oxygenated blood and nutrients to all areas of the heart or brain. The walls of these tubes are lined on their inner most aspect by thin tile like cells called endothelial cells which abut one another forming a protective barrier very much like a sheet of tiles in a bathroom shower stall. The endothelial cells are in contact with the flowing blood and are the first level of protection against the bad cholesterol (LDL).

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When excess saturated fat is consumed, the body metabolizes it to LDL. The excess LDL then may penetrate the endothelial cells and form a deposit or plaque immediately underneath them. This plaque may enlarge and actually push the overlying endothelial cells into the hollow space in the center of the artery where blood flows and narrow that space. The result may slow or limit flow downstream from a plaque and may actually cause transient chest pain with exertion if the oxygen and nutrients the artery supplies cannot meet the demands of heart muscle downstream from the plaque.

Of even greater concern, the LDL in the plaque may oxidize like rust in a pipe setting up a very unfortunate chain of events. Oxidized LDL is viewed by the body as a foreign substance. White blood cells, which are the body's sentries against invaders, will actually attack the plaque because it is viewed as a foreign invader much like a bacteria. Thus, white blood cells begin attacking the plaque in our arteries and this is

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the inflammatory response which sets the stage for a heart attack.

Plaques which become inflamed are more likely to become disrupted or crack. This cracking of the plaque will tear the overlying endothelial cell layer. Small corpuscles in the blood called platelets whose role is to plug holes in arteries after trauma become fooled into thinking that an actual hole has opened in the artery wall. The platelets stick to the torn endothelial cells and form a clot. It is the clot on top of the disrupted plaque which stops the flow of blood, oxygen and nutrients downstream from the plaque and results in the death of heart muscle, a heart attack or brain tissue, a stroke.

If inflammation in artery walls leads to plaque instability and heart attacks and strokes, then a measure of this inflammation may help predict a heart attack waiting to happen. The new test hs-CRP appears, indeed, to help do this. In fact, it appears to double the predictive value of the usual cholesterol blood test measurements.

Dr. Paul Ridker of Harvard has pioneered work with hs-CRP He has shown that in population studies, in which individuals are followed over time until they have a heart attack or stroke, the measurement of hs-CRP provides additional useful information and may help predict an event 4-6 years before it occurs. In the Women's Health study, apparently healthy women with hs-CRP levels greater than 1.5 milligrams per liter (mg/L) were 3 to 7 times the risk for a heart attack or stroke than woman under 1.5. Moreover, the test was predictive even in the absence of other traditional risk factors such as high blood pressure, family history, smoking and even high cholesterol. In men, hs-CRP levels greater than 2.1 mg/L predicts 3 times the risk for heart attack and 2 times the risk for stroke.

More interestingly, when stored blood samples from already completed clinical trials of cholesterol lowering medicines were tested for hs-CRP, the test appeared to help predict heart attacks and strokes independent of the cholesterol level. This was some of the first evidence that a group of cholesterol lowering drugs called "statins" could lower the risk of heart attack and stroke by a mechanism independent of the cholesterol lowering effect of the drug. Statins appear to lower hs-CRP by about 20 percent. Very safe levels of hs-CRP are generally thought to be below 0.6 mg/L.

What about a non-medicated approach to hs-CRP? It must be emphasized that while higher hs-CRP levels seems to predict heart attacks and stokes, researchers are not absolutely sure that lowering it will be protective. Certainly the already completed clinical trial data would suggest lower is better. But prospective clinical studies which test a hypothesis going forward in time rather than retrospectively or after the fact are not yet completed. Prospective studies are the gold standard of medical research. Despite this, most researchers feel that lowering hs-CRP is the way to go. While one may lower it with drugs like stains or aspirin, one may also lower it with exercise, smoking cessation, and most powerfully with weight loss. Especially in obese or overweight individuals with even slightly high blood sugars, lowering weight by 20 pounds will decrease hs-CRP by 30 percent.

In summary, a new blood test called hs-CRP, which may be ordered easily by all clinicians, may significantly improve the ability of cholesterol tests to predict the risk for heart attack and stroke in men and even more so in women. While an elevated value may be lowered by drugs, optimal diet and lifestyle again may be shown to achieve for most individuals a similar result. Without underemphasizing the proven benefits of today's drugs to reduce death and disability, the non-medicated and minimally medicated life remain a viable alternative to an over reliance on bottles of pills to treat all our health care problems.

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