The story of Juan…
a heart rendering account of child abuse

8 Questions to ask the doc before knee replacement

Is a mammogram still the best choice for cancer check

TV co-star, Megyn Price has a message for your teens

Can you chew your way to a healthy smile?
I have often used the analogy of a bicycle wheel in discussing risk modification with my patients.

As a Preventive Cardiologist I have a singular goal - to avert heart attacks and strokes. At first blush this may seem to be an easy task. After all, everyone knows the major risks for vascular disease – smoking, diabetes, high blood pressure, elevated cholesterol, unfavorable genetics, age, and gender. Understanding and identifying the causes of heart disease should make the mission of prevention rather effortless. For example, a patient presents to my office with high cholesterol and ongoing tobacco abuse. All I have to do is inform her that unless we get her cholesterol down and she quits smoking she will be at risk for a heart attack and stroke. She accepts the cholesterol lowering medicine, quits smoking, and lives happily ever after. Sadly, this storybook fantasy is just that - pure fiction. Rarely is the task of modifying a patient’s risk factors so seamless. Let’s use this simple example as a springboard to discuss the major problems inherent in risk assessment and cardiovascular prevention. First, however, we should define the modern day usage of “prevention.”

Levels of prevention

Prevention presents in three levels: primary, secondary and tertiary. Primary prevention refers to helping people avoid dangerous habits. As it relates to cardiology, the avoidance of smoking cigarettes would be the best example. Other non-cardiac modes of primary prevention include getting vaccinations, wearing seat belts, and practicing safe sex. Secondary prevention refers to a method whereby we physicians try to prevent an already established disease process from reaching its undesired culmination. For cardiovascular disease this would mean halting the progression of established but “sub-clinical” (asymptomatic) coronary artery or cerebrovascular disease so that a patient will never experience a “hard outcome” such as heart attack, stroke, or sudden cardiac death. In the world of non-cardiovascular medicine, screening tests such as mammograms and bone densitometries are excellent illustrations of secondary prevention. Tertiary prevention comes into play when we see patients who have already experienced a hard outcome; now we must preempt another event. The practice of cardiovascular prevention is built upon a foundation of solid
risk assessment at all three levels, with subsequent strategic correction of these risks.

Our first obstacle to confront and hopefully overcome is the fact that all risk-scoring systems possess inherent flaws in their ability to discriminate those individuals who will develop an event from those who will not. The most treacherous example of such a defect is the clear underestimation of risk in young people. In one popular scoring system a forty-year-old male smoker with a systolic blood pressure of 180 mm Hg has a ten-year risk of only four percent. At sixty-five, this same hypertensive smoker would have a ten-year risk of 47 percent! In this illustration, the clinician is left with the unenviable task of trying to convince the forty-year-old of the slippery slope upon which he is unwittingly floundering. One way to contend with this problem is to reclassify the young smoker. Inform him that although his four percent risk may seem low, it is comparable to that of a healthy sixty-five year-old. In other words, his “risk age” is sixty-five; he’s basically bearing twenty-five extra “life years.”

Another shortcoming inherent in all risk classification systems resides in their inability to consider all cardiovascular risks. It is not a far cry from the truth when I say that nearly every week someone publishes a paper revealing a new risk for heart disease. In the realm of genetics, abnormalities in specific genes clearly put people at higher risk for heart attacks yet none of these variants has been included in a risk scoring scheme. Then there are the inflammatory markers that are unquestionably associated with heart disease. Only one, CRP, has made it into our scoring systems. How about the omega-3 fatty acids DHA and EPA? Although there is now solid evidence that the amount of these fats contained in our red blood cell membranes can predict the risk of sudden cardiac death (90 percent reduction in risk with scores over eight percent!) no scoring system has yet included the HS-Omega-3 Index® as a variable to consider. You may be asking yourself why it is that all known risks have not been included in these scoring systems. The answer is that it is simply too difficult to do so. Not only must complex statistical assessments be made, but any revised calculator must

In an attempt to quantify cardiac risk, a number of scoring systems have been devised. They all are built upon the same premise - multiple cardiac risks are generally far worse than a solitary one. For example, an isolated but substantially elevated LDL-C (the amount of cholesterol carried in our LDL particles) is generally not as ominous as the combination of mildly elevated blood pressure, central obesity, and slightly elevated triglycerides. In other words, risk factors act synergistically; their whole is definitely greater than the sum of their parts. The most famous risk assessment system is known as the Framingham Risk Score. By inputting seven simple variables into this risk calculator – age, gender, total cholesterol, HDL cholesterol, smoking status, blood pressure, and the use of blood pressure medications – the clinician can gain a solid sense of a particular patient’s ten-year risk for hard cardiac events. We use this information to risk stratify our patients. They are seen as low, moderately high, high, or very high risk depending on their ten-year score. With this knowledge in hand, we can then inform our patients of their status and begin the work of appropriately chiseling away at their risk factors in order to help create a lower risk person. The concept is sound, but as with everything in life, it’s not perfect.

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be appropriately tested. Unfortunately, the calculators are valid only within the population tested, and as socioeconomic and genetic differences undoubtedly influence the manifestation of cardiac events, a superior scoring system would have to be tested in an enormous and heterogeneous population. Needless to say, this would be a Herculean task, one that is currently nearly impossible to perform. So where do we go from here? How can we physicians do the best for our patients?

First, it is my contention that although we utilize population-based studies to guide our management of patients, we must not lose sight of the fact that individual patients are indisputably unique. It is our job as clinicians to care for the patient who is sitting in front of us in our exam room. Our oath is to the patient, not the population. We must therefore stretch beyond the boundaries of population-generated guidelines and approach each patient with a fresh look.

**A myriad of tests are available**

When I evaluate a patient, I start with the basics. I utilize a risk assessment system such as Framingham in order to be able to know where a patient lies within our traditional guidelines. I then move beyond, looking at a host of newer risks such as LpPLA2, Lp(a), homocysteine, CRP, and the HS-Omega-3 Index. Based upon this evaluation, I reclassify my patient’s risk. If she is intermediate or high risk, I often go further, probing for the presence of sub-clinical atherosclerosis. Two tests are very helpful here: Carotid Intima Media Thickness (CIMT), and Coronary CT Angiography (CCTA). The CIMT measures the thickness of the carotid artery’s inner lining. Thickening is a sign of early disease and its presence raises an individual’s risk of future heart attack and stroke. This test can be a wonderful motivator; seeing nascent plaque in one’s carotid artery will often convert a Statin non-believer into a veritable zealot. CCTA is simply a CT scan of the arteries that feed the heart. New software has enabled this test to be performed with remarkably low radiation exposure, about one eighth the radiation of a nuclear stress test. This test can be not only very motivating (for the same reason as the CIMT) but it can also at times dramatically alter therapy. Recently, an asymptomatic surgeon with only a “moderate” risk score underwent a CCTA only to learn that he had imminently life-threatening coronary disease. Without his subsequent bypass surgery, within a short time he would most likely have become yet another sudden cardiac death statistic.

Today, there are many opportunities available to you and your physician to have a substantive impact on minimizing or even preventing serious heart disease sequelae. Family history, gender, and age are the only risks set in stone, but all other risk factors that can be modified, should be modified. I have often used the analogy of a bicycle wheel in discussing risk modification with my patients. The wheel represents heart disease while each spoke of the wheel represents a modifiable risk factor.

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**As founder of VitalRemedyMD, a medically directed nutritional supplement company, Dr. Seth J. Baum has bridged the gap between allopathic and complementary forms of cardiology. His Boca Raton practice, Integrative Heart Care is devoted to the prevention of cardiovascular disease. This article is also available at www.radthemag.com.**

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