

A Conversation with Seth Baum, MD New Member of the ANA Medical Advisory Board

Seth Baum, MD, FACC, a cardiologist in Boca Raton, Florida was recently appointed to the ANA Medical Advisory Board.

Barry Fox, PhD, JANA Associate Editor, conducted the following interview with Dr. Baum.

Q: Dr. Baum, how did you come to practice intervention cardiology?

My initial training and practice were quite conventional. I graduated from the Columbia College of Physicians and Surgeons, took a Residency in Internal Medicine at NYU Medical Center, then completed a Fellowship in Cardiology, Interventional Cardiology, and Electrophysiology at New York Medical College. In 1991, I opened a private practice in Boca Raton, Florida, specializing in clinical/interventional cardiology.

For the first several years, I primarily did tertiary care. But after seeing patients come back for repeat procedures, I began wondering if there wasn't another approach. Instead of repeatedly performing invasive techniques on people who had already developed significant cardiovascular disease, was it possible to help prevent the cardiovascular problems from arising in the first place? Or, at least, to reduce the risk?

I began searching for ways to prevent disease, and to intervene in existing disease, without using catheters and invasive means. One of the first areas I looked into was naturopathy. I read many of their texts, did distance training in nutrition and supplementation, and over the next several years introduced alternative health strategies into my practice. Overall, the results were positive and I felt I was offering my patients the best of both worlds: alternative methods to help them reduce the risk of developing cardiovascular disease, and standard medical interventional cardiology and electrophysiology for those who already had advanced disease.

From 2000 to 2003, I served as Medical Director for the John W. Henry Center for Integrative Medicine at the Boca Raton Community Hospital, as well as Medical Director of the Mind/Body Medical Institute at Beth Israel Deaconess Hospital, Boca Raton Division. Today, I continue to have one foot in the world of standard medicine and one in the world of alternative medicine, working with my patients to reduce the risk of cardiovascular disease, as well as lecturing and writing on Preventive Cardiology. I'm also Board Certified

in Clinical Lipidology and have achieved level 3 verification in Coronary CT Angiography. My dream is to integrate the best of standard and alternative approaches, and develop a center for preventive cardiology, a concept that is unfortunately not as widespread as it should be in this country.

Q: As a cardiologist practicing in integrative medicine, what do you think people should be doing to reduce their risk of developing heart disease?

I think it's a good idea to eat a healthful diet, take appropriate nutritional supplements, maintain an ideal body weight, exercise every day, learn to manage stress, and have their blood lipids checked regularly. I believe that, in addition, people should ask their physicians to delve deeper into their lipids and begin to look, for example, not just at the LDL cholesterol level, but also at the number of LDL particles or LDL-P.

Q: Which brings us right to the next issue. Most people are familiar with the standard "cholesterol numbers" – total cholesterol, LDL "bad" cholesterol and HDL "good" cholesterol – and know that an elevated LDL is a risk factor for cardiovascular heart disease. However, research conducted over the past ten or so years has suggested that simply keeping LDL cholesterol under control may not be the best way to attack cardiovascular disease.

That's true. This concept dates back to the 1960s, when Dr. Friedrickson, one of the fathers of lipidology, said that our focus should be on the assessment of lipoproteins, on their number, size and characteristics, rather than simply on the total amount of cholesterol carried by the LDL population. Unfortunately, back then we did not have the commercial capacity to measure lipoproteins, so a surrogate marker, LDL cholesterol, was chosen instead.

Let me step back a bit and remind you that cholesterol is transported through the body in different "vehicles," including LDL – low density lipoprotein particles. LDL



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particles come in different sizes and carry differing amounts of cholesterol, but we didn't have an easy way of measuring the number and sizes of those particles. What we could do was measure the amount of cholesterol carried in the population of LDL particles, so we did.

LDL-C, or the amount of cholesterol the population of LDL particles contained, became the gold standard measurement by default, not by choice. It was assumed that there was a direct relationship between LCL-C and the number of LDL particles in the blood. All of the major trials were designed around measuring LDL-C. Reducing LDL-C became a major focus of treatment, and medicines were designed to do that.

Unfortunately, we've found that no matter how low we drive the LDL-C, we're still missing a lot of people. We're only decreasing the risk by about 30%, which means we're missing some 70% of cardiovascular events. Here's another alarming statistic: about 50% of the people who have heart attacks have normal LDL-C levels.

Clearly, looking only at the LDL-C is not enough. It's as if a traffic engineer were only counting the number of people on the road, rather than the numbers of cars they're sitting in. There might be 1,000 people on a road right now, but the more important issue is how many cars they're sitting in. If each person is in his own car, there are 1,000 cars on the road and you have a traffic problem. But if those 1,000 people are in 500 cars, traffic isn't so bad. And if they're sitting four to a car, there are only 250 cars on the road, so there's absolutely no traffic at all. If you were a traffic engineer trying to ensure that traffic flows smoothly, it would certainly help to know the number of people on the road. But it would be far better to know the number of vehicles on the road.

A number of researchers have looked into the LDL cholesterol versus LDL particle number issue. Major studies that have looked at LDL particle information include the Cardiovascular Health Study (*Arterioscler Thromb Vasc Biol* 2002), the Women's Health Study (*Circulation* 2002), the Healthy Women's Study (*Am J Cardiol* 2002), the Veterans Affairs HDL Intervention Trial (VA-HIT) (*Am Heart Assoc* 2002, *Circulation* 2006), the Pravastatin Limitation of Atherosclerosis in Coronary Arteries (PLAC-1) (*Am J Cardiol* 2002), and the Framingham Offspring Study (*Am Heart Assoc* 2004). These and other studies have found that the LDL particle number is a vastly superior measure of cardiovascular risk and future events.

LDL particle number is a better measure of risk than LDL cholesterol because the particles come in a variety of sizes and vary in cholesterol content. This explains why cholesterol content and particle number fail to correlate. The more particles you have, the greater the chance that one – or more – will penetrate an arterial wall and begin the atherosclerotic process.

Q: How does the LDL cholesterol versus LDL particle number issue play out in your practice?

This issue arises in up to 70% of my patients. They've been receiving treatment and their LDL cholesterol's are

only moderately elevated, or perhaps even in the safe range. This suggests that their treatment has been successful, but when you measure their LDL particle number, you realize that they are still at risk of cardiovascular disease and need more therapy.

Q: Is the size of the LDL particles important? Should physicians be measuring this as well?

The verdict is still out on particle size, but the number of particles is clearly much more important than their size.

Q: You said earlier that it had been difficult to measure LDL particle number and size. Is that still a problem?

No. LipoScience Labs of Raleigh, North Carolina, offers the NMR LipoProfile, which uses nuclear magnetic resonance spectroscopy to measure LDL particle concentration (or number) and size, as well as the LDL, HDL and VLDL subclass levels. The test is available nationwide through LabCorp. By way of disclosure, I should tell you that I'm a consultant to LipoScience Labs.

Other labs, including Atherotech of Birmingham, Alabama, measure qualitative size and cholesterol content, although they do not count particles.

Q: How are LDL particle results measured, and what are the various levels?

LDL particle results are given as nmol/L. Anything below 1,000 is considered optimal, from 1,000 to 1,300 is near optimal, from 1,300 to 1,600 is borderline, above 1,600 is high and over 2,000 is very high risk.

Q: How do you reduce an elevated LDL particle number?

The approach to reducing an elevated LDL particle count is the same as for reducing elevated LDL cholesterol. The statin drugs can be helpful. Other drugs such as Zetia, which inhibits cholesterol absorption, and Niaspan, which can enlarge particle size and lower the particle number, oftentimes without affecting LDL cholesterol, are also helpful.

Non-pharmacologically, exercise can help lower the particle number. Dietary interventions are also helpful. Transfats are the biggest dietary offenders. Saturated fats should also be significantly limited, but not entirely eliminated.

Q: Not every researcher agrees that the LDL particle number should routinely be measured in cardiology patients. Can you comment on the conflicting ideas?

It's not unusual for doctors to disagree about the best approach to patient care; that happens in all areas of medicine.

A great deal of research has demonstrated that measuring the LDL particle number is better than simply looking at the LDL cholesterol for identifying and managing patients' lipid abnormalities. Some researchers have argued that it's not cost-effective to measure the LDL particle number in large numbers of people over many years. I doubt that this is true, but we practicing physicians treat our patients individually – we care about the Mr. Jones or the Ms. Smith who is in the office now, and deserves the best possible care. I want to be able to look all of my patients in the eye and tell them that I have given them the best possible care.

Preventive Cardiology, Our Greatest Hope for Eradicating Heart Disease

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We physicians struggle daily to do what is best for our patients. We assiduously endeavor to stay current with the ever-expanding volumes of medical literature, simultaneously responding to our patients' very real and immediate health issues. Journals multiply like viruses and studies fill these journals. How do we digest all the data? How do we sift through divergent results and conclusions, and arrive at an approach that is reasonable and effective? How do we practice evidence-based medicine when the evidence is so ephemeral? On one hand, doctors tend to resist change, clutching their views like life preservers in a raging storm at sea. On the other hand, we can be fickle, latching on to a single trial that jibes with our oft-times preconceived notions and personal belief systems, the end result being a dismissal of something that may be of immense value. A perfect example of this latter phenomenon relates to Homocysteine. Elevated levels of Homocysteine have clearly been associated with cardiovascular events – not to mention Alzheimer's, osteoporotic fractures, macular degeneration and stroke – yet a single negative trial in the realm of heart disease serves as a battle axe to the Homocysteine naysayers, which they wield to dismantle all prior literature. An example of the former phenomenon relates to cholesterol. The powers that be, the doctors who establish guidelines such as NCEP and ATP 3, have held tight to cholesterol as the answer to the cardiovascular

plague that afflicts the western world. Yet a thorough review of the major Statin trials (that utilize LDL-C, the cholesterol contained within LDL particles) teaches us that controlling LDL-C eliminates only thirty or thirty-five percent of cardiovascular risk. What of the remaining sixty-five or seventy percent of events? If controlling LDL-C is THE answer, how do we account for this massive remaining unmanaged risk? Clearly there is far more to this story. And this brings us to the concept of Prevention, an approach to patient care that demands open-mindedness and at times, great inner strength.

The practice of Preventive Medicine, though a concept thousands of years old, has recently enjoyed a resurgence. Perhaps it is the increasingly impersonal medical system or the over-abundance of technologically-oriented aspects of medicine that has brought prevention to the fore in many circles. Perhaps it is our patients' clamoring for solutions that reach beyond the patchwork effects of medications and surgery. Whatever the reason, prevention seems to be here to stay. And when we look at some statistics as they relate to Cardiovascular Prevention – my particular area of interest – they reveal why prevention is so essential. Forty-two percent of Americans still die from cardiovascular disease. That's one American every thirty-three seconds. One and a half million heart attacks still occur annually in the United States. Sixty-four percent of us are overweight, while a solid thirty-three percent are, by definition, obese. A third of our country's youth is now overweight and consequently, "Adult Onset" Diabetes Mellitus is becoming commonplace in children. Without a concrete preventive approach, we will surely continue on the road to disaster. In the remainder of this article, I will address three tools that can be incorporated in a clinical practice of Preventive Cardiology – LDL particle information, Coronary CT Angiography (CCTA), and assessment of the Carotid

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Intima-Media Thickness (CIMT). Please do not infer that the absence of a discussion on Therapeutic Lifestyle Changes (TLC) means that I undervalue exercise, dietary interventions, and stress modification. In fact, the opposite is true. I believe wholeheartedly that TLC is the cornerstone of disease prevention. I also believe that in doing everything humanly possible to try to stave off the number one killer in the western world, we physicians should examine and treat other important lipid components – HDL-C and Triglyceride – as well as the “emerging cardiovascular risk factors” such as CRP, Lp-PLA2, and Lp(a). My focus on the three aforementioned tools stems from an issue of space; it is just impractical to discuss all preventive techniques in this article. Also, follow-up case reports in the next few issues of *JANA* will focus on the merits of these three tools. And so, let me now introduce these techniques and their clinical applicability.

We have known for many decades that cholesterol plays a pivotal role in the genesis of atherosclerosis. In 1913, Anitshkow showed that cholesterol fed rabbits developed aortic atherosclerotic plaques, whereas sunflower oil fed rabbits did not. In the mid 1960s, Fredrickson, one of the fathers of Lipidology, emphasized that Lipoproteins (LDL, VLDL, HDL, IDL, and Chylomicrons), not the cholesterol contained within them, should be the focus of our attention. Although he proclaimed that the preferred way to manage our patients would be to directly count these lipoproteins, the lack of a commercial means to do so led to the adoption of LDL-C as a surrogate marker for LDL-P (the number of Low Density Lipoprotein Particles), and thus a Gold Standard by default. Earlier I alluded to the inability of LDL-C to adequately predict CV events. Many studies have shown us this. Even the famed Framingham Trial found that half of all heart attack victims have normal LDL-C levels, and eighty percent of premature CV events occur in individuals with an LDL-C under 125 mg/dl. From where does this LDL-C shortcoming emanate, and why is LDL-P such a superior predictor? The answer to these questions lies in the nature of LDL (again, the particles that carry cholesterol).

It turns out that LDL particles vary greatly in both their size and the amount of cholesterol they contain. Consequently, there cannot possibly be a consistent and direct relationship between LDL-C and LDL-P. One is unable to glean from LDL-C how many particles exist, and the opposite holds true as well. This reality would be inconsequential were it not for the fact that LDL particles are what penetrate arterial walls to cause vascular disease. These particles do not dump their cholesterol in the blood; they enter the intima-media as holoparticles and once inside the arterial wall, they do their damage. In this circumstance, as in many other aspects of medicine, gradients come into play. The more particles there are in the bloodstream, the more likely they will be to invade the arteries. How much cholesterol they carry is not the issue; it's how many LDL particles there are that counts. This is why study after study has shown that LDL-P is far more effective at predicting CV events than LDL-C. It is a better clinical tool by which we can manage our patients' lipid abnormalities and prevent events. Though anecdotal, my management of

LDL-P has resulted in a dramatic decline in the number of acute myocardial infarctions I see in my clinical practice. In fact, I cannot recall the last patient I saw who had an acute event with an optimally controlled LDL-P.

Coronary CT Angiography is a technology that fits well in both medical camps – Preventive and Therapeutic. Our ability to image coronary arteries non-invasively through high speed CT scanners has enabled us to detect early disease in the vessel walls. The therapeutic implications abound, but for now I'll limit myself to the Preventive applications. By seeing coronary artery disease directly, unequivocally, and far sooner than either stress testing or cardiac catheterization would enable us to do, doctors can now aggressively attack risk factors before the disease has progressed to a symptomatic stage. We are all familiar with patients who resist our attempts to manage their CV risks. The old adage, “A picture is worth a thousand words” can be understood with crystal clarity when we present these resistant patients with images of their own diseased arteries. It is an unparalleled motivator to see your own vessels clogging with plaque. Once this alarming plaque accumulation is actually witnessed and the ramifications of these findings fully explained (i.e., the very real risk and possibly imminent danger of stroke, MI, etc.), it is rare for patients to oppose making the appropriate healthful changes in their lives.

Carotid Intima-Media Thickness can be utilized in a fashion similar to the CCTA. By measuring the intima-media thickness, we can predict future cardiovascular and cerebrovascular events. In fact, in 2000, the AHA recognized IMT as an independent predictor of CV events, and in 2006, the Screening for Heart Attack Prevention and Education (SHAPE) Task Force recommended the use of IMT in Low and Intermediate risk patients for improved risk categorization. The downside of CIMT is that it is only a surrogate marker for coronary artery disease. Unlike CCTA, it does not tell us with absolute certainty whether or not a patient has CAD. However, because of the systemic nature of atherosclerosis, 70% of people with this disorder have both coronary and carotid artery disease. This makes CIMT a very accurate predictor of the presence of coincident coronary artery disease. The upside of CIMT is that it does not introduce radiation exposure or require the administration of contrast. CIMT can be used not only as a means of establishing a patient's current CV risk (at times upgrading low or intermediate risk patients to high risk), but also as a guide in managing our patients' risk factors – when intima-media thickness increases by more than 0.033 mm annually, we are alerted to the fact that much more needs to be done from a preventive standpoint.

This article has been a brief introduction to three relatively recent advances in the world of preventive cardiology – LDL-P, CCTA, and CIMT. The next few issues of *JANA* will include case reports that elucidate how internists, family practitioners, and cardiologists can utilize these tools to help prevent CV events. I am confident that by possessing greater knowledge about our patients' CV status, we will be more likely to make real and permanent changes in our patients' lives and by so doing, decidedly diminish their risks of succumbing to a CV event.