2005/05

Thursday, May 19, 2005 at 7:00pm

- Newsletter - read about the speaker and the talk
- Directions to the meeting
- Online discussion of this meeting
- Order a DVD video of this talk
- Previous SLF talk by Dr. Miller
- Speaker web site

- 2005/06 Next meeting
- 2005/04 Previous meeting
- Meetings

Discussion period Notes

Dr. Philip Lee Miller, M.D.

Heart Disease

Newsletter http://smartlifeforum.org/2005/05/newsletter.html

Copy of newsletter below

Phillip Lee Miller, MD

Cardio Vascular Disease and Heart Attacks

Cubberly Community Center 4000 Middlefield Road, Room H1, Palo Alto, California

May 19, 2005 at 7:00 PM

Future Speakers:

1. June 16, David Cowan, BA, CNC, CBT

Unlocking Wisdom of the Body via Electrophysiological Reactivity Using the QXCI or SCIO Biofeedback

1. July 21, To be Announced
2. August 18, Raymond Peat, PhD

Protecting and Restoring the Nervous System

1. September 15, Stan Field

Another Perspective on Heart Disease

1. October 20, (tentative) David Brownstein, MD

Iodine, Why You Need It, Why You Can't Live Without It

Opening Bio-Med 101 Presentation

Bernd Friedlander, DC, "Effect of Microwater on Free Radicals & Anti-Oxidant Enzyme Levels"

Attempts are under way to inhibit oxidation of blood by increasing anti-oxidant intake using electrolyzed alkaline water. Animal studies have shown that ORP 300 microwater significantly reduced serum peroxide levels and increased SOD enzyme levels.

Introduction

Born in San Francisco, Dr. Miller graduated in the Centennial class at UC Berkeley with a degree in Biochemistry. His senior advisor was the now famous Bruce Ames. He entered the UC San Diego School of Medicine in the charter class in 1968. Prior to graduating from UC San Diego he co-produced a 30 minute film entitled Waiting – a prescient documentary about evolving trends in health care delivery becoming more group and institutionally-based. Interestingly, one of the stars of that film was Dr. Dean Edell.

For 25 years he was involved in Emergency Medicine and Urgent care, punctuated by training in Neurology at UC Davis – Sacramento Medicine Center. It was here that he trained on the first generation of EMI CAT Scanners. In 1994 he met with Dr. Julian Whitaker and went on to work closely with him for one year at the Whitaker Wellness Institute in Newport Beach, California. In 1996 Dr. Miller founded the Los

Gatos Longevity Institute, one of the very first centers nationwide singularly focused on providing full service Anti-Aging and Longevity Programs. He now provides one of the most structured, comprehensive and time-tested programs available anywhere.

He is currently a charter member of the American Academy for Anti-Aging Medicine and has passed the first-ever Board Exams in Anti-Aging Medicine in December 1997 and December 1998 qualifying him as a Diplomat of the American Board on Anti-Aging Medicine. Additionally, he holds distinctive memberships in the European Academy for Quality of Life and Longevity (EAQUALL), the American College for the Advancement in Medicine (ACAM), past membership in the American Academy of Neurology (AAN), as well as the Santa Clara Medical Society, and the California Medical Association (CMA). Dr. Miller is a co-author of the LEF Revolution: the New Science of Growing Older without Aging. Other projects in the works including a new, distinct, and topical Longevity Newsletter. See him at www.antiaging.com.

Main Presentation

Remember in high school and in college physics courses how simple things were at the atomic level? Electrons, protons and neutrons. Fast forward to the last 15-20 years and there are now a rich array of leptons, bosons, baryons, color, charm and quarks. It's dizzying. Similarly, 30-40 years ago it was all so simple. High cholesterol from eating too much butter clogged up your arteries leading to coronary thrombosis. You suffer "the big one" – a heart attack. In technical terms a myocardial infarction – death of heart muscle cells and tissue. In the 1970's rarchers were looking at the etiology of coronary thrombosis vs. the possibility of undetected coronary artery vasospasm. These may still be operative, but a highly complex array of factors is now known to be at play.

Lipid research and clinical application has moved from simple cholesterol monitoring based on the flawed, and now fatal, "cholesterol theory." High cholesterol caused heart attacks. The 40 year old Framingham study showed that 50% of all heart attacks had absolutely normal cholesterol numbers! We have seen major advances in monitoring and therapies of HDL (high density lipoprotein), Triglycerides, LDL (low density lipoprotein), VLDL (very low density lipoprotein), and specific ratios of these.

More recently doctors have begun to look at hs-CRP (high sensitivity c-reactive protein), fibrinogen, Lp(a) (lipoprotein a) and homocysteine. Each of these factors is either an inflammatory acute phase reactant or a substance that leads to a series of inflammatory events by modifying the integrity of the endothelium of the coronary arteries. These are probably more predictive and modifiable -- but not always with "modern" drugs, so these newer markers have been given short shrift.

Interestingly, the picture that is evolving of an underlying inflammatory condition while "new" may be a recapitulation of earlier work traced back to one of the Giants in Medicine, Rudolf Virchow in the 19 th century. He formulated the following list of factors predisposing to vascular thrombosis:

1. Changes in the vessel wall.
2. Changes in the pattern of blood flow (flow volume).
3. Changes in the constituents of blood (hypercoagulability)

A nascent industry gained momentum in the early 1970's when the trend shifted away from lengthy medical management of angina or coronary artery disease with the use of various vasodilators, chiefly "nitrates" such as nitroglycerin. Coronary artery bypass surgery started with the Vineberg procedure using the internal mammary artery to bypass single vessels.

One of the premiere cardiologists in American medical history, Dr. Eugene Braunwald, wrote a most provocative 1976 editorial in the New England Journal of Medicine (N Engl J Med. 1977 Sep 22;297(12):661-3) warning of the amassing “medical industrial complex.” How prescient that almost 30 years ago he warned us:

“An increasing number of patients are being operated on, not because of the presence of intractable angina but because of the hope, largely without objective supporting evidence at present that coronary bypass surgery prolongs life or diminishes the frequency of subsequent heart attack.” He further stated that “this rapidly growing enterprise is developing a momentum and constituency of its own and as time passes it will be progressively more difficult and costly to curtail it materially if the results of carefully designed studies of its efficacy prove this step to be necessary.”

In a most interesting political twist Eugene Braunwald’s wife, Dr. Nina Braunwald, was chief of cardio-thoracic surgery at UC San Diego at the very same time he was Chief of Medicine also at UC San Diego. So there were early warnings. The growth of angiography has now become big business. It is based on a static plumbing hypothesis that progressive arterial narrowing will inevitably lead to critical reduction in blood flow thereby increasing the risk of thrombosis and death of the myocardium distal to the blockage.

A narrowing at the left main artery has been unceremoniously called the “widow maker” lesion because so much of the entire left myocardium blood supply is distal and a blockage there leads to widespread myocardial injury or death. Looking at a 70% or more narrowing was seen as critical. But this hypothesis had limitations. It does not look at any “collateral” and compensatory circulation. The evolving picture which will be fully discussed in the presentation is based on “plaque stability” and endothelial dysfunction.

Molecular biology and more specific “knock off” mouse models are revealing just how the dynamic inflammatory model is overtaking the static plumbing model. Coronary thrombosis and the process of atherosclerosis is seen as a rich cascade of events involving inflammation, infectious co-factors possibly from the oral (mouth) flora, free radical and oxidative stress. Cytokines, activation of t-cells, macrophages, mast cells and adhesion molecules. The unfolding picture is dizzying much like the modern physics picture of the atomic level dynamic. The end result is plaque rupture with acute thrombosis and obstruction of blood flow. The challenge is to make sense of this new picture. What constitutes an adequate workup? How can we look at predictive vs. associative statistics? What are risk factors that are predictive and have modifiable utility? What would constitute a modern and more rational work-up?

Today a patient develops angina – chest pain from decreased blood flow to the heart. A stress treadmill is performed. This may or may not show EKG ischemia (decreased blood flow). An angiogram is thereafter performed. If significant narrowing is seen, immediate balloon angioplasty (forced dilatation of the artery with an internal balloon) may be performed or the patient could be scheduled for emergency coronary artery bypass surgery (CABG). The patient not infrequently is presented with the surgery treatment plan under subtle or even outright duress. There is very little time for thoughtful and rational consideration. The whole process is a trainwreck moving at 60 mph.

There is an alternative. Super fast EBT (electron beam tomography) scans with the GE-Imatron HeartScan, have been in use for the last ten years. [www.geimatron.com/02_technology.htm] These scans grade coronary artery calcification by location and intensity. Since, initially, these scans have been patient-initiated or directed they were ignored or even derided by the general medical community until the last two to three years. Then we began to see the use of these scans at the Mayo Clinic for determining the necessity of hospitalization due to chest pain. With a zero calcium score, you are probably going to be sent home. They are seen as more accurate than simply looking at cardiac enzymes.

Competing super fast CT Scanners with more generalized applications have now entered the market with similar calcium scoring but not identical technology. The jury is out. An intense medico-economic debate and political battle is being waged A comprehensive workup would include a dual isotope (sustamebe-technetium-thallium) heart scan, a stress treadmill, 2-D Echocardiogram combined with the HeartScan and a full VAP (Atherotech) or Berkeley Heart Lab (BHL) fractionated lipid panel giving us a far more complete assessment. Carotid artery ultrasound and the newer, more specific, IMT scan (intimal media thickening) provide even more qualitative and quantitative evidence of arterial plaque accumulation and stability.

On the horizon is the advent of non-invasive coronary artery virtual angiography. This will rival Fantastic Voyage within three-five years. And what are we to make of various therapies including the raging statin (Lipitor, Mevacor, et al) debate that hopes, or threatens, to broaden its application and appeal in the coming years? There are indicators that the focus may shift quite soon to the raising of HDL even more than the reduction of LDL. The essence of Dr. Miller's presentation is to make sense of this evolving theory and etiology of cardiovascular disease, coronary artery thrombosis and the appropriate workup that we should all understand and thoroughly discuss with our cardiologist or, hopefully never a thoracic surgeon. Our lives depend upon this.