Philip Miller, MD: Real Issues in Heart Disease

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Speaker 1: Now for the main event. Dr. Miller has been on our board of advisory for a long time. He's been coming to this Smart Life Forum Silicon Valley Health Institute for about 15 years now. He has become a leader and pioneer in anti-aging and longevity medicine, so if you don't want to get old, talk to Dr. Miller.

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 Board Qualified by the ABAAM Board, American Board of Anti-Aging Medicine.

Dr. Miller is the founder and CEO of California Age Management Institute. He just started a new software company with details to be announced soon. He's on our advisory board as I mention. He will be this evening about the real issues in heart disease. Please give a warm welcome to Dr. Miller.

Dr. Miller: Thank you. We almost had a problem in terms of the connection and I always worry about are we going to have a good connection or not. It kind of me, calm down, calm down. I'm always amazed when I see these broadcast studios or radio stations that got gazillions of dollars' worth of equipment. I said, "Oh, we've got some technical difficulties." I don't know how that happens but I think we've overcome it. I think we've got some clear sailing here.

I want to thank everybody for inviting me back again. I feel like when the host comes on Saturday Night Live and says, "You know, I'm up to number five. I'm up to number six. I'm up to like the number one." I think I've been here at least five, six, maybe seven times in the last 15 years so I always enjoy coming back. It's a great crowd.

What I have for you tonight is a really long lecture. It violates some of my rules, which is never put more than 90 slides or more than an hour, because you're all going to fall asleep. This is fast-paced and there's more information here that you've ever seen before in terms of what are the attributes, the causes, and the etiology of cardiovascular disease. We're going to cover the whole spectrum.

What [Kristin Greengate use 00:02:32] is like one area and one approach in virtually everything she said is a segue to what we're going to discuss now, in terms of what are the multiple etiologies of cardiovascular disease. It's not just one cause. It's not cholesterol.

I never believed the cholesterol theory when I was in medical school. They all called me an idiot because everybody knows that. I also remember even when I was a kid, when I was a teenager, the craze was, you got to all eat margarine because that's all really good stuff for you. I always thought, that stuff is junk. Even then I knew this. Intuitive ... This stuff is really junk.

Let's start out. This is just around the corner from here. Anybody know where this is? Nobody knows. This is Arastredero Preserve. This is where I go to clear my head out. Spend hours and hours.

What I call the new medicine series for you. It's everything you want to know about cardiovascular disease and probably more. Let's dive in. Let's look at heart as a metaphor for the philosophical, just a metaphor. What is heart? Follow your heart. He died of a broken heart. An honest heart possesses the Kingdom. The heart is wiser than the intellect. The only lasting beauty of the heart. It spans the ages, spans philosophical concepts that the heart was the center of the soul.

In 1511, Leonardo da Vinci contrary to what the Pope's wanted and what the religion wanted, started doing dissections along with Michelangelo and others. Started looking at the physiology and the structure of the heart apart from what the ancients had believed about the heart. The ancients thought the head was just a cooler.

Sir William Harvey was really important because what Sir William Harvey was one of the first anatomists to actually show that the heart was a vessel that pumped blood around the system. He is one of the first to actually show scientifically through some very elegant experiments that the heart wasn't just this organ that was a metaphor, but actually had some physiologic function pumping blood around the rest of the body.

This was pretty radical in those days. So we look at the anatomy the heart. When you go through this in detail, but basically ... Is my marker working? No, it's not working. It goes on and off.

Anyway, we're going to go through this in more detail. Here's the action of the heart. You can see how the heart pumps into each segment there. Goes to the lungs, gets oxygenated, comes back from the lungs, goes in the left ventricle, and gets pushed out the aorta into the body. Comes into the heart, gets oxygenated, goes and once it comes back in the left ventricle, goes in the aorta, goes in your brain, goes to the rest of your body. That's the basic physiology.

Great. Thanks.

Male: Still blinds you.

Dr. Miller: There we go. Where's it at? Okay. This is the way that I ideally would like to work up a heart and, again, what Kristin Green had said, we're just seeing more and more of this not being done in medicine because you only have 15 minutes, we don't care about etiology, this costs money, everything's been reduced to cost-effectiveness, how much does this cost? As opposed to, can we look at the heart from various modalities? What we look at, is that we can look at the heart electrical.

The heart is an electrical organ the heart is an electric organ. That's what an EKG, thus looks at the electrical output of the heart. You can look at the vascular, so you can look at angiography which is just basically looking at the pipes. You can look at wall thickness with carotid ultrasound, which is what we're doing. Carotid intima media Thickness. You can look at calcium scoring, which we look at the calcification in the

arteries. You can look at arterial stiffness which is pulse wave analysis. We'll show that what these are. Different ways of looking at cardiac function. We can look at biochemical marking, which is sort of my love because of my biochemical background. All the lipids, the HDL, the LDL, cholesterol and the triglycerides.

Structural we can look at the echocardiogram. We look at the structure of the heart. We look at the valves, which is totally overlooked by most cardiologists until it becomes almost too late. Again, the aortic valves when I see most often. We look at perfusion, which is dual isotope heart scan. Hardly anybody is getting that, which is the metabolic function of the heart. Finally, we look at energy, which is VO2 Max, how well are you oxygenating your system.

You look at this and now that you get a much more, richer picture of what is your heart. It's not a chest X-ray. It's not a EKG. It's looking at all these different modalities because everyone of these is important: electrical, vascular, biochemical, structural, perfusion, energy.

Let's look at some functional cardiology. Cardiology, vascular disease, this is what we were told, starts as an inflammatory process. We look at hormonal, we look at nitric oxide, we look arginine, endothelial relaxation factor, endothelian. In fact, it turns out the heart has its own built-in hormonal system that hardly anybody's really studying because we're only looking at this, again, this Newtonian mechanical device. As opposed to this rich plexus of vagal influence and all these internal hormones that are going on. Vascular you're looking at flow and rheology.

I'm really big on rheology, which is just flow characteristics. Is your blood thin? Is it thick? Where the red blood cells, are they ready to aggregate? Do you have anything that's thinning your blood out? We look at mechanical valves, electrical and biochemistry.

These are some aging fundamentals. What is it that happens as we get older? As we get older, we're rusting, we're oxidizing, we're getting stiffer. We look at inflammation, which is burning up and again this is the segue. Cardiovascular disease starts as an inflammatory process. Even in medical school, I remember being taught, first there was an injury and then everything else happens. You look at glycation. Glycation is what makes it stiffer. Glycation is basically high fructose corn syrup, too much sugars, thickens your arteries, thickens your lens, thickens your muscles, makes you stiff.

Finally, energy. This is an interesting thing, I'm not going to go into this in-depth because I could spend about an hour talking about this. In the 19th century, there was this debate in science about what is energy? Newton just told us what power was, which was a much easier concept to get across because it's a mechanical concept. Energy, frankly is much more difficult. You have to take a course in thermodynamics to discover Helmholtz and Gibbs Free Energies. Even when I had Chem 5 at Berkeley, I never mastered this course.

It's probably easier to think of as we're getting older, we're not so much losing energy, which is a difficult concept as much as we're losing power, locomotive power, vocal power, sexual power, energic power, political power, whatever it is. We're losing power as we get older. One of the things that I'm telling all my patients now is a year older, don't stop moving. Keep moving. That's power.

Let's look at integrative model. Integrated with model would be looking at rheologic, which is sheer stress, viscosity, elasticity, the calcium paradox, vitamin D, vitamin K, mitochondria. None of you are taking enough vitamin K.

Male: I am.

Dr. Miller: How much are you taking?

Male: 90 milligrams of MK-4 and [above 00:11:07].

Dr. Miller: 90?

Male: 400 of [them 00:11:11] [inaudible 00:11:12]

Dr. Miller: 90 milligrams?

Male: Yes.

Dr. Miller: Because the dose that I recommend is 15 milligrams. That's 15,000 micrograms. Hardly anybody is getting that. Let's start at the top here. Rheologic stress, viscosity, elasticity, calcium, vitamin E, vitamin K, mitochondria. We've gone way beyond cholesterol already haven't we.

Let's look at vascular health. I spent a lot of time talking about this. A man is as old as his arteries.

Thomas Sydenham, 1624. In many ways, a lot of the concepts I'm going to introduce you to, we've known about this stuff for 400 years. We're rediscovering inflammation or we're rediscovering arterial health as a marker of overall health. These are the major coronary arteries here. This is the right coronary artery. This is the left coronary artery.

Here's a left main. That's really important. Here's the left main. Here's the left anterior descending. I always forget. I think this is the circumflex right here. This is the left part of your heart that's really important. This is where most heart attacks occur. This is just important, the right part of the heart supplies the lungs. It's driving the lungs in the left side, here that goes to your brain and the rest of your body. The left side is really important, but the right is just as important.

This is the critical thing that's missed by most cardiologists, most physiologists, is that when you look at this, that is looking at the heart from a plumber standpoint. Look at

those. If you have a blockage right here, that's called a widow maker because if that gets blocked off, everything distal to that dies. The reality is, the heart has this rich plexus of microvasculature where, in many instances it bypasses all these.

You look in cardiologists we'll see how the cardiologist looks at an angiogram, but you have this ability to bypass these blockages with this rich microvasculature that's never visualized. You don't see this in an angiogram. You don't see this in any modality that we're doing. I think this is probably just anatomical model. I don't know any living way that you can see this rich microvascularity.

The common concept is, here's a normal artery. Here's the endothelium in the media and eventually what happens is it gets junked up and it gets junked up in the outside. That's the old concept. Too much fat, too much sugar, builds a bunch of junk builds up, and it gets plugged up. That's not how it works.

If you look at it, see there it is, it looks at, from this diagram that it's just artery just getting plugged up. Too much pig fat, too much bratwurst, too much bacon, too much green fried tomatoes. Anybody lived, grow down south you know what I'm talking about. The reality is you look at the intima and you look at the media which is the muscular. What happens is there's an injury to the media intima and everything starts invading this thing here.

This is the correct view here and I thought that maybe one of the problems is what they call right here. This is probably the basement membrane. We know there's a basement membrane in the kidneys. As the basement membrane in the kidneys gets violated, you got a lot of problems with kidney disease. I don't know anybody is talking about the basement membrane here, which is the the border between the intima, which is the inner lining and the media, which is the muscular layer there. Something happens here.

Once that that layer gets violated, that's when you got problems. What happens is, normal artery here, stuff happens, and it starts invading this here, and all these cells start invading here. Here's that vessel. Here's where it become thickened, right here. Let's go. We're going to come back to this in a little bit.

Let's talk about what I talk about a lot, lipids. Cholesterol, irrelevant, I have to tell my patients this every visit. Your total cholesterol is irrelevant. When you tell me your total cholesterol is 240, I don't care. I have lots of women who see me who have total cholesterols of 240 and they're healthy, totally healthy. Why? Because they have a high HDL.

Total cholesterol is that combination of your HDL, your LDL, and your VLDL. If you're one of these women, because guys don't have this, if you're one of these women who have the HDL of 140, you're going to have a really high total cholesterol. Total cholesterol, meaningless.

You want to look at triglycerides. The one factor that's totally overlooked. So, probably when I was a medical student, mainly what we were looking at was total cholesterol. Then we were starting to look at ... well maybe we ought to look at LDL and HDL, but mainly was the focus on total cholesterol.

Then we got a little bit more sophisticated and say, "Well, maybe we should look at some [subfractions 00:16:31]." Well, look at the LDL and the HDL. HDL, every talks about the HDL as the good cholesterol, and the LDL is the bad cholesterol, as if the HDL has little smiley faces, and the LDL has little frowny faces on them. These are not cholesterols.

It actually took me years to figure out LDL is a carrier protein, and it carries cholesterol to the heart for reasons that it's trying to repair an inflammatory injury to the heart. The LDL carries cholesterol to the heart. This is a big point number one: LDL carries cholesterol to the heart. HDL is like a little vacuum cleaner. HDL carries cholesterol away from the heart. What high HDL theoretically is this thing that's clearing the heart of all the excess cholesterol. That's where you want lots of HDL. Women always have much higher levels of HDL. Over the years, I've pretty much assumed it's estrogen-related. That more estrogen, more HDL.

Then we start looking at VLDL. VLDL is related to triglycerides, related to carbohydrates, it's related to sugar. Then we start looking at risk ratios. We looked at the total cholesterol of the HDL. The one is probably the least informational to me. I look at the LDL, the HDL. That's a little bit more important but frankly, I'm looking more and more at the triglyceride, the HDL. That is a diabetic risk factor. Carbohydrates, [fee 00:18:01] triglycerides, higher triglycerides is the one that's overlooked.

VAP we're talk ... Now we get a little bit more sophisticated. In the last 5 or 10 years we got the VAP. The VAP is acronym. I forget what it stands for, but it was a quick method of looking at some of these more sophisticated risk factors. Now the latest one, the last 3 or 4 years is LipoScience is done their little marketing shtick, and now what we're looking at total particle numbers.

First there was cholesterol, there was LDL and HDL, then we were looking at more sophisticated like the ratio. Then we looked at the VAP and the particle size. Now we're looking at particle numbers. It's always an effort to look at what is the one thing that's causing the problem. Cholesterol doesn't cause heart disease. Triglyceride to me is the one that's overlooked more and more.

How many remember Tim Russert? Tim Russert died suddenly at the age of 58 with a totally normal cholesterol. It was 150. He had triglycerides that were off the chart. Incidentally, also led a very stressful lifestyle. He probably went to Europe, drank a lot, ate a lot, rushed back home. Stuff happened. He had high triglycerides, normal cholesterol.

What else do we have here? In the last 15 years, you know one of my mentors was talking about cholesterol, was talking about, what we call Pattern B. This is a different concept. This is Robert Superko. Superko is the one who was trying to bring this concept into our consciousness, which is maybe if you have this big LDL, fluffy LDL particles, big LDL particles, that they just float on by. If you have this small little LDL particles. Dense, small LDL particles, they have a much greater likelihood of invading into the intima and the media and causing this inflammatory process that causes cardiovascular disease.

This was a big advance when we looked at pattern A, pattern B. Pattern A, big fluffy LDL particles. Pattern B, small LDL particles. What the big advance really was the pattern, because almost always high triglycerides, low HDL, and really I still think it's more of a triglyceride problem than it is a cholesterol problem.

So what are some of the predictive risk factors that I look at? I measure this on just about every patient, every time. And this not happen, you go to Kaiser you're not getting any of this. You go to your doctor, you're not getting any of this, because it's now all: How much is it cost? What's insurance is going to pay? My insurance doesn't cover this. Do I have to call for pre-authorization? Forget it. I'll just order four test and be done with it.

That's what's happening to all of you today. That's what's going on in medicine. So I look at fibrinogen. Fibrinogen is an acute phase reactant. It increase your tendency to clot, thickens the blood, more likelihood that you're going to clot, forming a heart attack and stroke. You're going to hospital with a heart attack, the first thing you look for is your fibrinogen. If your fibrinogen is elevated, they give you TPA. Big shot of that and say, "Hey, we just cured you".

How about if we look at just fibrinogen before you go into the hospital. So I look at fibrinogen in everybody. Lp(a) is a marker that people sort of, kind of put on the wayside. It causes or may accelerate atherogenesis, thickening of the heart, more hardening of the arteries.

This is one that was studied by Mary Malloy at UC San Francisco 15 years ago, and it's never gotten a lot of press. The interesting thing is the Lp(a), women can have higher Lp(a)'s and it's not as harmful. Again, I think it's estrogen-protected. Men can have Lp(a)'s that are elevated. Most of you are not having this measured. My thinking is the Lp(a) is more harmful for men as a cerebral vascular problem, more than a cardiovascular problem. I think it's more a problem, in you're 60s, 70s, and 80s. You say 70 used to sound really old to me. It doesn't sound as old anymore.

All this 20 years older than you. When you're 10, 30 is old. When you're 30, 50 is old. When you're 50, 70 is old. When you're 70, well 90 is old. When you're 90, I'm not sure what is old, Harvey.

Male: [inaudible 00:22:47]

Dr. Miller: 110, okay?

Male: [inaudible 00:22:51]

Dr. Miller: Okay.

Speaker 4: [inaudible 00:22:53]

Dr. Miller: CRP, we talked about CRP. CRP is just another measure. These are inflammatory measures and Lp-Pla2, I use this also as an inflammatory marker because the LDL, when the Lp-Pla2 is elevated, it means the LDL's now becoming more inflammatory, mast cells are coming, hystiocytes are coming in. That whole inflammatory cascade that I'm going to show you. These are predictive risk factors. I look at these. When I say predictive, in a sense.

Let's look at this. Let's see if this works. I'm going to keep my fingers crossed. Let's see if it works.

Male: [Skip 00:23:31] is not processing. You say something about [inaudible 00:23:33]

Dr. Miller: See, it's not going to work. It never works.

Male: Are you talking about audio?

Dr. Miller: The audio. It's not working. Homocysteine is an amino acid. It's partly metabolized as we age. It does require the MTFHR gene, but it turns out the MTFHR gene, that mutation is actually really common. I've almost stopped testing for it because it's so common. Homocysteine gets metabolized to methionine. Methionine is really important for DNA synthesis. So methylation is a really important process for DNA synthesis. If you don't have that enzyme, that convert homocysteine into methionine, and homocysteine builds up, it's a big irritant. As homocysteine rises, that's found to be a really high risk factor.

Same as gout. Gout is when the uric acid ... There's a gene called xanthine oxidase, uric acid gets converted to urea, urea dissolve when you pee it out. If you don't have that xanthine oxidase, uric acid builds up, you get gout. That's what all these enzymes are for.

Male: Can I see the video one more time?

Dr. Miller: Let's try one more time. I don't think it's going to work, because it should be bigger than this. I have three videos on here and they have never worked. It's really to bad, because it's really a cute video. Someday you guys will be able to see this video. It's a really good video.

Male: [inaudible 00:25:11] to YouTube and point [inaudible 00:25:12]

Dr. Miller: [Mark it 00:25:16]. Here's some new risk factor. Here's ones that you've never heard about. This is really interesting to me because a lot of these have to do with physics. The only course that I did terrible on when I was in college was Physics, because I made a mistake in my freshman year of Physics. I took Physics 4 which was for all the engineers and they're talking about Newtonian Mechanics and I couldn't get it. So I switched to Physics 2B and I got As without even going to the class.

I've always come back to Physics. It's like maybe I could finally get this as I get older. Mechanical stress is a big deal. Viscosity is a big deal. Loss of elasticity and calcium shifts.

Let's talk about this. Again, I told you about these LDL particles. Here's a large fluffy LDL particle. Here's a small LDL particle. Apparently, these, you see how complex it is. It's a carrier protein with a lot of cholesterol in it. This is smaller one here. Actually, I'm sorry. This is HDL so the carrier protein that's carrying cholesterol away from the heart. Here's the LDL particle that's carrying cholesterol to the heart.

Now, why do I say about Tim Russert, it wasn't about cholesterol? Because the Framingham study which is probably the best study that we've had ... Little community outside of Boston where they actually study they actually studied the entire hypothesis. They've been doing this for at least 40 years. We have a really good ongoing cross-sectional population study. Probably the best study outside of, I think, Iceland where they're studying the entire country. That would never happen in this country.

What you see is the difference between those who had a heart attack and those who didn't have a heart attack. There was no difference in their cholesterol. None. Which you can conclude from this is cholesterol is a bad predictor of heart disease. It goes back to what Kristin said and you could talk about this for a long time, difference between association, causation.

Causation was talked about endlessly by Aristotle who had four postulates of causation then was discussed by David Hume was a great Scottish philosopher. This idea of causation has gone on for a long time. What is causation? Cholesterol does not cause heart disease. You can see that it's a bad predictor.

What do we do that we go beyond [inaudible 00:27:44] biochemical markers? You got to do imaging. This is what I'm really big on is imaging. I've seen some people come to me at 39 years old and their LDL is 250. Their total cholesterol is 400. They're worried. I do imaging on ... Arteries are clean as a whistle. Cholesterol doesn't cause heart disease.

You look at imaging. Originally what we're doing 15 years ago and I was the one of the first doing this, and then it was kind of far out. Nobody was doing this. Then suddenly, maybe about 10, 12 years ago, Mayo started doing. Once Mayo started doing ... It was okay.

Male: The bandwagon.

Dr. Miller: Now it's on the bandwagon. They would do it in the ER. Normally if you had chest pain, you had a normal super fast CT scan, then you were cleared. What it does is electron beam was originally ... Was the Imatron, which then GE bought out. Now it's a GE Imatron. There's actually two types of scanners and [inaudible 00:28:46] the hospitals what they'll do is they'll use their normal scanners, the spiral CT scan and they'll kind of re-adapt it to your heart. This is not the way it works. The right one is to do the electron beam, the EBCT, and that one is specifically dedicated doing scanning for your heart. It's really quick, it's really fast, it only takes about 4 or 5 minutes and that's the one that shows you calcification of your heart.

This is an imaging study that say, here's your numbers, here's the risk factors, this is what happened to your mom, this happened to your father. What's actually happening to you? What's going on to you? Now we've come from population-based studies to individuals. What's going on with each one you? That's what you want to know. You really don't care about five million other people who been studied. You want to know what's going to happen to me? What's going to happen to my husband, or my wife, or my mom, or to my Dad?

This was the GE Imatron, again, it started out as the, I think it was the Imatron, then and then GE bought it out. That's what it shows. You can see there's a little bit calcification here, I think a little bit of calcification here. I always find these a little hard to read because they're cross-sections. That's what they do is they calcified, they quantify these calcium scores. Here again, and then what they can do it in color so you can get a better idea of exactly where the calcium is. Now you're looking for calcium.

Calcium is end-point of a build-up of inflammation. The body trying to react to that walled off calcification. It's the end stage. But calcification alone still doesn't necessarily mean you're going to have bad stuff. It just means your arteries are not clean as a whistle.

So furthermore, this is what we're doing in my office and I'm one of the only people in the Bay Area who can do really quality CIMT scanning. In the CIMT scanning is what we're doing is we're looking at, again this thickening here of ... from here to here, where it's thickened here. This is a chart that was given to be by one of my consultant showing that there is a correlation between the CIMT and cardiac events. Then it becomes kind of a controversy. I really like to look at the CIMT. This is what we see in the office. So we look at the traditional risk factors through look at your LDL, your HDL, triglycerides. You look at your blood pressure, you look at family history and we look at diabetes. And we look at what are the traditional risk factors here. Then we scan up and down. Right here, that is the common carotid. Then, we look at the internal carotid and the external carotid.

Internal carotid goes into your brain, external carotid goes into your face. What happens is the common carotid bifurcates. What I'm going to show is first of all, what

I'm looking is the thickness right here. The thicker it get that means there's more inflammation, more infiltration, and at a certain point there's a fibrous cap here. Fibrous cap pops open, blood pops out, stroke or heart attack.

What I want to know is what's your thickness here? Because if you're down right here, that's supposed to be good. In this case here, this person here actually did have some plaque, okay? So you see this plaque right here and you see right where it is, it's right at the bifurcation. This is really interesting because most what we're seeing is at the bifurcation. I'm going to show you why it's really interesting. It's a flow characteristics. This is not necessarily cholesterol. This is flow characteristic. It's a physical characteristic, because I'm going to show you there's more eddy current.

This person here did have pretty significant blockage there. Not necessarily catastrophic. It's still probably about 50%, but probably needs further evaluation. This person here didn't have any risk factors. Nothing. We'd looked at right here. This score right here was really low. This score here was really low, so they're into intima media thickness was really low. It was really low here. This person here was really cool and I think this one ... This one had an LDL ... Just the LDL alone is about 160. So again, the LDLs was really high. Clean as a whistle. I told you I had one patient. His LDLs was about 250 or 300. Cholesterol goes to 400 if he's not treated. All the drugs don't do anything other than it's going to wreck his health. He has clean as a whistle arteries.

I like this modality. There's about 88% correlation between this and the heart. So now you can put this together with the heart scan and think you're now up to about 95% certainty. What's going on with your carotid arteries? What's going on with your coronary arteries?

Let's look at some other. This is a tradition. This is what should happens when you go to your cardiologist. Cardiologist says, "Okay, let's do an angiogram." There's a couple of reason why he's doing the angiogram. One, because it's been validated. Two, because to be honest with you, the only way you survive is your cardiologist. You don't survive as cardiologist doing what I am doing: talking to people, talking about the lipids, talking about the risk factors. There's no money in it. The only place you can make money as a cardiologist is doing angiography and then putting in stents. What does the angiography show?

This one did come out. You can see here's the beating heart and you can see right here there's calcification. There some stenosis right here. The question is what do you do? Are you going to stent that? Yes, I'm probably going to go ahead and stent that. I mean I'm in there I might as well put a few stents in there.

This is the angiography. What you do is you snake up through here. You go retrograde, go back into the heart. You come back out and you look at each one of the coronary arteries. You're looking for calcification. You're looking for stenosis. You're looking for blockages.

This is really interesting. Eugene Braunwald ... I think he never really totally realized how great some of your professors were until you're like maybe 20, 30 years out of school and you're really in retrospect. Eugene Braunwald was our chief of medicine. Scared the hell out of everybody. Was a very imposing, almost arrogant figure, but in retrospect, probably was one of the most important cardiologists in American history.

The people say Dudley White was the most important one. I think Eugene Braunwald was. Eugene Braunwald was Chief of Medicine, Chief of Cardiology, was a brilliant in terms of cardiology. His wife was Chief of Thoracic Surgery. Here you got his wife was Chief of Thoracic Surgery who's doing the original bypasses. You have Eugene Braunwald, Chief of Cardiology. Imagine what that was like at the dinner table.

In 1977, Braunwald said, "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 the coronary artery bypass surgery prolongs life or diminishes the frequency of sudden heart attack.

This rapidly growing enterprise is developing a momentum and a constituency of its own. As time passes, it will progressively be more and more difficult and costly to curtail it materially if the results are carefully designed studies of its efficacy prove this step to be necessary. In other words, what he was saying is what Eisenhower said in 1958: "Beware of the military-industrial complex." What Braunwald was saying was, beware of the medical-industrial complex. I still think in 1977, these words still ring true. Very prescient.

Let's look at this. This comes from circulation, 1996. Plaques occupy space, yet as pathologist studies have shown in the past, the arterial lumen is not necessarily compromised. Remember what I showed you about that rich vasculature? Implying that the angiogram would be a poor tool to assess atherosclerosis in living subjects. However good it may be at detecting high-grade stenosis that cause symptoms.

Look at angiography. What's the value of it. The limitation angiography increases has come apparent that the amount of narrowing the coronary arteries is only of minor importance. This is really a mind blowing concept that nobody pays attention, nobody gives any credence to. Where does this come from? This came from non-invasive heart center San Diego, which is from Braunwald's Chief center.

Look at some other modalities. Echocardiograms. I'm really big on echocardiograms. I love echocardiograms. Shows the structure of the heart. The left ventricle. Show the atrium. Shows you ejection fraction. Ejection fraction is how much blood you can squeeze out any one stroke. It's like the efficiency. It's like in a big cylinder. Like a big Norton 750. How much can you squeeze out in one compression.

If you look at the echocardiogram its ejection fraction is what I'm looking at. I want to see, optimally, I want to see above 65. 50 is considered optimal, or acceptable, but if you're at 50%, that's not too good. If you're at 30% or less, that's pretty ominous sign. I've got guys from 30 back up to 40 and 50. It can be reversed, but it's a tough go. I

use a lot of CoQ10, I use testosterone, I use other modalities. This is generally a bad sign when it's 30. Efficiency of the heart, echocardiogram.

The interesting thing is this is estimated. It's not a definite number and there's different ways of estimating. If cardiologist kind of gloss over it, its' the primary thing that I'm looking for. Other images study sustainable technetium, thallium, dual isotop heart scans. This is a way of looking at the metabolic flow. I just showed you structural. I showed you bio-chemical. Now, we're looking at metabolic, which is: are there certain segments of the heart that are not beating correctly? Are there dead segments of the heart?

This one shows you here a normal one, and an abnormal one. This doesn't look good here. The point of the dual isotope scan, which I can't hardly get any longer is that dual phase seem to show an image of the heart better than just one, which is either thallium or the technetium. I'm seeing here the thallium or technetium, but you need to do the dual to get a much better imaging study. Again, metabolic shows you the structure of the muscles. Do you have dead muscles?

Let's look that they physics of the ageing heart. Let's look at classic pathology. This is what we study when I first started as a medical student. I never could understand it, and it's still hard for me to explain it. Basically what it's showing you is a normal curve, a curve that declines here. You're basically the physics of the heart is declining. The stretch and the ability of the heart to deliver it's blood decreases with an aging heart, or a failing heart.

What happens is in the mitochondria, you see this in a healthy heart. Then, it starts getting a little bit more disorganized, and even more disorganized. In the end stages the muscles look a lot more disorganized. That's an unhealthy heart. Meaning, a heart that's had a heart attack, a heart that's got a lot of vascular disease. We can see again, the heart is mainly a muscle. If you look at sarcometers, or the striated muscle, which you look at the structure you can see it degenerating with a failing heart.

This is really interesting. This goes back to the physics. I showed you something here. I'm going to tie some things that I just showed you earlier. You look at the red blood cells, you look at plasma viscosity and other constituents. Look at the viscosity. Viscosity is the thickness of the blood. Do you have thin blood? You have thick blood? There's different ways to look at it. I'm probably going to be on thin ice here. I know either in Chinese or [inaudible 00:41:19] medicine, there are different ways of looking at blood throughout the seasons and different states here. But the way I look at it is, I look primarily at fibrinogen. I look at thickness of blood. Thicker blood is more likely to clot.

What happens is the easiest way to look at it besides fribrinogen is just looking at your hematocrit or red blood cells. Hematocrit used to be a measured amount. You would spin down the blood with a centrifuge, but nobody does that anymore. What you have now is just flow cytometers. The flow cytometers are now measuring the

amount of red blood cells that are flying by and then they infer the hematocrit, which is the number of the hematocrit.

Most docs don't even know this. I spend so much time doing Q/A in my lab, that I know a lot of the inside information that the flow cytometers that they're using now, it turns out if your hemotocrit is high, everybody worries about it. It's actually the number of red blood cells. If they get it to a certain number they can clump, stick together. Now you got a problem with heart attack or stroke. These viscosity figures are important, because thickens the blood, the risk for heart attack or stroke.

I showed you earlier this is a problem. Any bifurcation here, that's where this stuff is going to be a problem. You got these eddy currents here. You have areas of high shear strength, and low shear strength. I always thought it should be the opposite. The point is, where you have low shear strength that tends to be areas where you're more likely to have injury. Injury leads to inflammation. Inflammation lead to heart disease.

Now what we have, is we have ... Here's just a pure mechanical model. Forget the lipids, forget the cholesterol, forget all the ... Maybe just from the shear mechanic model. There's one or two books that I've read that are incredible beautiful books, all physics, showing just from the shear forces, stress forces, and the mechanics of blood flow alone. When you come to these bifurcations and you have these eddy currents, that alone there, over time is an area where you're going to more likely to have blockage of the artery at these bifurcations.

The same thing of the bifurcations of the left main, the left anterior. All these areas where you have the bifurcations is a problem. Again, you can see these eddy currents right here more likely to have blockages there. Again, showing you what happens. Again, this shows here lower strength, and higher strength, shear fractions here. This again, I don't know if this is going to work. This is really great. It's really great. Never works.

What it's showing you, again as we get older and older that the shear forces are building up and just the simple shear forces ... It's [inaudible 00:44:04] like a bridge that's been around for a hundred and fifty years. The shear force and the stress force begin to take their toll.

I'm going to go through this kind of quickly. This is something I was really interested in a few years ago. I wanted to get this machine. Looking at elasticity. Just the elasticity [inaudible 00:44:21] another way of measuring arterial health. Just simply how elastic are your arteries? You have this thing called elasticity, extensibility. You look at pulse wave velocity, augmentation index, and stiffness index. It gets pretty complicated and you can shift these curves right here. We lose elasticity.

Arterial stiffness is a major cause of heart attack, stroke, and myocardial ischemia. Again, remember I told you the glycation increases stiffness. It could be that the shear forces are increasing stiffness, but as we get older everything is getting stiff. Muscles

are getting stiff, our legs are getting stiff, our arteries are getting stiff. It's a major cause here. This SphygmoCor was a way of looking at the pulse. Look at this pulse wave here. Right here. You get these things called reflective waves. Then you look at the amplitude of these reflective waves and you come out with an augmentation index.

Basically, you're looking for the difference between the radial artery and the aorta. The aorta is really where a lot of disease starts. Again, this is a great video. Boy, I really thought this was going to work. This one I did on myself. I said, okay. I said, here's where I was and here was the ... I think this was the augmentation index and I went here and said, "oh, that's cool." I think I was about 55 or 60 when I did this. I might have been 60 when I did this. So I thought 60 down to 40, that's good. There's different ways of saying, "Oh." Again, back to what Sydenham's said, "You are as old as your arteries." More pliable arteries, less stiff arteries, better health.

Inflammation and stiffness. It's a chronic inflammation associated with endothelial dysfunction. Inflammation may stiffen the arteries. That's where you come into Arginine and Nitric Oxide, Endothelium. CRP, again, we've talked about this, associated with brachial artery pressure. Pulse wave is an inflammatory marker. Arterial stiffness is an important factor. Here's where I look at it. CRP is not really related to infection. I look at small artery stiffness.

Let's talk about this evolution here. This is a perspective in the 1990s. Inflammatory Prolifitive Process insult to the endothelium smooth muscle, large number of growth factors. Cytosine, vasoregulatory molecules participate in this process. What happens is you have these correlator between, here's a thin cap and here's large lipid. You have these other factors and you put a correlator. Here's what happens when you start getting this filled up. Here's a progression of an artery that's getting more ischemic, more cerebrovascular disease, peripheral vascular disease. It's a progression of this.

I'm going to skip ahead here, because what's happening is you've got this endothelium, smooth muscle, inflammatory cells. I eluded to this earlier, what are those cells that are floating around? There's macrophages, lymphocytes, and mast cells. What's happening is these monocytes, and lymphocytes are found in the atheroma. They're expressed by the endothelium, expressed by the microvessels. This is what's going on.

This leads to what Kristen Green was saying. Is it chlamydia is it some other factors. The end process is you got all these smooth muscles cells. You're going to have these migrating cells area here. Inflammatory [inaudible 00:48:08]. This is vascular adhesion molecules. You got another molecules here, and you got these macrophages in here. All these inflammatory cells are trying to reduce their inflammation but they're actually making it worse.

As they get worse you get this whole cascade here of LDL, HDL. You get all these thing chemotaxis. It looks like a complicated thing but basically it's LDL being oxidized. All

these other factor coming and the systems goes ari. It just spins out of control. The point of this is this. LDL and cholesterol did not start or cause this process. LDLs recruited in this process initially to repair the process, but eventually comes in here and the LDLs starts getting oxidized, and you start getting all this stuff here.

All this inflammation, all this oxidation, LDL oxidize, but all these macrophages, histocytes, and eventually this thing pops open. This pops out here. Blood pops out. Platelet comes together. Stroke or heart attack. Inflammatory process. LDL didn't cause it. Gets recruited in the process. One of my friends in a really great lecture said many years ago, "Cholesterol, found at the scene of the crime. Not guilty."

Let's move ahead. I've got a few more slides here. Calcium Paradox. I love this. As we get older what is happening is ... I try to tell my grandkids, I tell my younger patients, "when you're younger, you're banking calcium. You want that calcium going to your bones. You want to get as much calcium in because you're banking it, and you want that to go into your bones." What's happening is we get older for one reason or another we have low calcium. You believe in the hypothesis that I don't believe in wishes. We should restrict our calcium. Really don't take any calcium, because when you restrict your calcium down to nothing. I'm not going to get any extra calcium. Body says fine. I'll just steal it from the bones.

What happens is we get this reverse calcium flow, where calcium is not getting recruited out of the bones. When you're younger calcium is going into the bones. As we get older calcium is coming out of the bones. We said fine, we're going to bank that calcium, but we need it. We need it for muscles, we need it for cardiovasculartone. We need the calcium. We don't have enough going on here and there's other factors. Not enough vitamin D, not enough vitamin K.

Vitamin K. Another point. I want you to remember this. Vitamin K. What's going on here is parathyroid hormones starts getting turning on. What happens is the body senses, for some reason or another, there's not enough calcium. Parathyroid, which is here, turns on. They start taking calcium from the bones. Parathyroids functions start reabsorbing calcium from the bones. That calcium then goes right into your coronary arteries, into your valves, which nobody is looking at. Valves in your coronary arteries.

This is a very complicated thing here. Calcium is really essential. There's this vascular process here. This is a normal aortic valve. this is a disease aortic valve. It's calcified. Even when I see on an echocardiogram it says, "a little bit of calcium. Don't worry about." Always bothers me. Because a little calcium always end up being more calcium, and more calcium. Eventually that valve becomes a diseased valve.

Speaker 5: Would this be picked up on an Imatron?

Dr. Miller: This is ... No, because they're looking at a blood valve. It could be, but this is one you will pick up on an echocardiogram, but not the imatron.

That's a disease artery or a disease valve. That artery is headed for replacement. You don't want that. So, what do we do? Here's a central dilemma. It's a calcium dilemma. Interestingly, here's a inverse correlation between osteoporosis in aortic calcification. Osteoporosis, calcium in the matrix is decreasing. It's going to your arteries. It's going to your valves. Incidentally, coumadin greatly enhances that bad process.

Here's calcification. Here's osteoporosis. Here's hip fractures. Vertebral fractures and hip fractures. You can see they're correlated. Here's aortic calcification, osteoporosis they're going up. Calcium coming out of the bones, osteoporosis. Calcium going into the arteries.

This kind of an interesting thing. I tried to make this correlation with a bone with arterial flow in an artery. I couldn't quite make it but they're very similar in terms of how the bone is perfused, and how the bone is laid down, and how basically what's happening with our arteries, they're becoming ossified. Or is it more like petrified. That's what's happening with our arteries. They're being petrified as if they're now becoming bone like. How do we prevent that?

How do we prevent that, vitamin D. I get the story of Vitamin D here. I got that from Bill Grant. I kind of picked up ... Bill Grant was really the guy that Rick Tutt kept talking about vitamin D showing all the epidemiologic studies. Vitamin D is critical. I put virtually everyone of my patients on 5,0000. I have a lot on 10,000. I've seen some need 15. I've even some patients need 20. I think the 20 means that somehow they're not absorbing fat soluble vitamins, but it's not uncommon to see people need 15 or 20,000. Everybody goes on 5,000. Occasionally, in rare instances, tiny little women that get by with 3,000. But 1,000 will not do it. You need at least 5,000, but it's got to be with Vitamin K.

Vitamin D goes, you can get it through sunlight, but you'll never get enough by sunlight. If you go to Hawaii, you go to the Caribbean you lay out in the sun, you will get enough vitamin D. We don't live in the Caribbean. You won't get enough. I remember many years ago it was Claire ... There was a lady that use to come from Berkeley.

Speaker 6: Claire Felix.

Dr. Miller: Yes, Claire Felix. She used to come from Berkeley. She was in a set at this alp, this latitude. 38 to 37 to 38 degrees. You can not get enough solar insulation to raise your vitamin D. You got to take it internally. You got to activate it. Vitamin D receptor binds here. Infinity is such and such. There's numerous effects in bones. It's a transporter, regulator bone matrix, provides proper balance of calcium, potent effects in growth differentiation, but it does more. Vitamin D, whenever I get a cold I take 50,000 units of vitamin D for 5 days. I personally think that vitamin D ... my experience is actually way more potent than vitamin C.

Vitamin D is actually is a hormone, and has all these other properties. Here is the vitamin D receptor. Let me talk about vitamin K. They work together. You got to do

them together. Vitamin K. You have this vitamin K1, vitamin K2. Here's vitamin K2. Here's what they call the MK4. Has four tails. This metaquantione, so there's four tails here. This big long lipidphylic molecule. Then you have the vitamin K2 that's the MK7.

Now, [Ruff 00:55:37] Housworth talked to us about eight years ago. He was one of the guys who's really promoting MK7. Now you'll see MK7. That's the big thing. You got to get MK7. It's way more powerful than MK4. I don't believe it. No one has ever showed me any studies. The MK7 that everyone is taking, if you're looking at what you're getting, you're getting 40 to 60 micrograms. I want people to have 15,000 micrograms. There's no way you're going to compare 40 micrograms with 15,000 micrograms.

The MK7 story is one that I've never bought into. I want people to having a lot of this. Buying MK2 and vitamin D are essential for bone health and arterial health. Why? Because it goes beyond that. Vitamin K goes to gammacarboxlyation, then it goes to the GLA matrix protein. You need vitamin K for GLA matrix protein. GLA matrix protein reverses calcifications. GLA matrix protein is vitamin K dependent. It's what prevents calcification or reverses calcifications.

Speaker 7: [inaudible 00:56:55] matrix blockers [inaudible 00:56:56].

Dr. Miller: They're related. Here's the MGP. The MGP right here is what's inhibiting vascular calcification. That's the point. The point of this is, it's the vitamin K that is making this active, and that is what's preventing vascular calcifications. Vitamin k is really an important one. Population that was found with a high dietary vitamin K2 intake reduced aortic calcification, risk of myocardial infarction, cardiovascular disease by 50%. The effect of K1 was less evident. There's some randomized study showing high intake of vitamin K complete blocked the process of our age related arterial stiffening. I really wanted to make this point about vitamin K.

GLA protein. Matrix GLA and osteocalcium. They're all related. Vitamin K dependent. Let's talk about anti-coagulants.

Speaker 8: [inaudible 00:57:57].

Dr. Miller: I don't want you to look at that. We got to move ahead. You're not going to get that. I want you to just get the basic concepts here. This is what causes clots here. Here's platelets. Here's fibrinogen, causes fibrin and it causes clots. You have two systems. You have the intrinsic system, the extrinsic system. Intrinsic system is all this stuff here. The extrinsic system is platelets. Platelet is what causes an initial clot. Cut your hand, platelets adhere, stops the bleeding.

The intrinsic system is this long cascade that comes down to fibrinogen and long term stops the clot. Why is this important? Because you can take nutrients that effect every one of these without ever touching coumadin. Here's a nattokinase, fairly complicated thing, but basically I still look at nattokinase, which is comes from the natto bean. Awful tasting stuff. I put most of my patients on nattokinase. I take it

everyday. Morning and night without fail. Nattokinase will reduce fibrinogen levels or prevent fibrinogen from being a problem.

Fibrinogen, inflammatory factor, thickens the blood, causes clots, heart attacks, and strokes. Nattokinase very easily nutritional way of reducing fibrinogen deaths. Risk. Here's the conventional answer. This is a conventional answer. When I wrote this news letter that you should've read. Even I had this moment because I wanted to work through, it took me a few days, and I finally looked at all these studies and I came to this ah-hah moment. I can't believe I never saw this just in one thing.

Here's statins. The idea is you have this cholesterol synthesis and it goes through these various stages and it goes through farnesyl, Sualene, and eventually comes into cholesterol. Here's the statins. The problem is the statins work too high in the char here. Here's your acetyl coA, Here's HMG CoA, here's Mevalonate. See here's cholesterol. The HMG, which are statins go high in the cascade. Bunch of other stuff happens in between there. Here's the biphosphonate, terrible for you. Here's steroid hormones. See that. I use to say faleshesly. You know you need a lot of cholesterol for your sex hormones. It's actually true. I've never been able to actually prove this, but I know as we get older one of the reasons I think our cholesterol rises because our testosterone, and our estrogen levels, and all our sex hormones are dropping. Whether it's true or not, I haven't been able to nail that down. But there's' no question that cholesterol is the basis of vitamin D, bio-acids, sex hormones, and modified proteins.

What happens is if you go back and you block it, all these steps are getting cut out. Plus CoQ10, vitamins E, A, and K, lipid anchored proteins. High up in the category. All this stuff is being short circuited. I did look this up. Somebody told me about this. I looked up at ... I think it was Merck that took at a patent about 1992.

Speaker 8: Right. CoQ10.

Dr. Miller: CoQ10 and a statin. 1992. They even knew then. They for some reason said, there's no money in it. [inaudible 01:01:25]. Somebody is going to put it on the shelf. Say well we got the patent. They knew even then that statins were going to do bad stuff to your CoQ10. Any of you who are still on a statin for any reason, you got to be taking a lot of CoQ10. These are all the problems right here. Here's that patent. Actually, it was 1990. I thought it was 1992. Here's the patent. I actually looked it up, because I never trust anything people tell me. I look it up. They say there's a patent. Fine, let me look it up.

So I looked it up. There it is. It's very clear. I got this right off of the US patent office. There it is. A pharmaceutical composition method of counter acting HMG, CoA reductase inhibitor associated myopathy. You mean they knew about this in 1990 and they still say, well this only happens to a few people. Only a few people. Guess what, maybe I see all those people, because I see people who are wrecked on statins. It's not everybody, but enough of them were. It's not a minor thing. It's a big deal. I see peoples memories goes out, and other factors.

What are the statins? Crestor, Lipitor, Mevacor, Pravechal. Then let's look at anti-coagulants. Coumadin, Heparin, Xaralto, Pradaxa. They keep proliferating. Aspirin, Plavix, Ticlide, Persantin. These are conventional approaches to anti-coagulation.

Speaker 9: What's the total revenue for those therapies?

Dr. Miller: Gazillions. Actually, probably in the hundreds of billions. I mean, I'm just guessing, okay. It's probably in the 50 to the 100s of billions. Coumadin is the one ... Actually, they're starting to phase this out, because now we can probably make more money on Xaralto and Pradaxa, so why don't we go to those. I'm telling you, and I've seen this over and over again. Coumadin risk benefit ratio is too high. The risk of accelerating coronary atherosclerosis, valvular calcification, bleeds, hemorrhages is way higher than the benefit.

This one lady came to me. She's 85 years old. Pretty [inaudible 01:03:30] lady, she had atrial fibrillation, she was on Coumadin. I talked to her and her son was there. I said, "Here's a routine that you can go on." I gave her my routine. I'll show you in a second. I said, "here's the routine. You don't have to be on Coumadin, but I don't want you to do this. I'm just showing you this is what could be done. You can't do this if you don't feel comfortable doing it." Two weeks later I get this email she's stroked. Thinking, oh man, I blew it. Turns out, she has never stopped her Coumadin. Even on the Coumadin she still had a stroke. The Coumadin prevent the stroke and mean while I've seen lots of people over the last 15 to 20 years. I know it's accelerating atherosclerosis, cardio-vascular disease, and valvular calcification.

These are the traditional approaches HDL trumps LDL. Know that HDL, there was this drug here that was developed by Pfizer. It was suppose to raise your HDL, but stupidly what they did, and every science student should know this, ,they didn't do the experiment where they did just this. No, they did it with Lipitor and this. It wasn't a pure experiment. They said, well it didn't work. You never tested the drug. You tested the combination. We'll give them a double whammy, and it didn't work.

Here's the HDL protects. Here's HDL protects. HDL protects. Here's the reverse process. HDL is reversed flow. It's taking cholesterol away from the heart. That's why HDL is really protective. If you have an HDL 70, 80, 90, 100 really protect it. I see some guys with HDLs in their 20s, even going down to 12. It's really hard. In that one individual we gave them a lot of coconut oil. That actually did work for him. Niacin works in some cases.

This is something that I want to get across. What is the percentage decrease from this to this? What is that percentage decrease?

Speaker 10: [inaudible 01:05:37].

Dr. Miller: What is it? Right. 50% right? What did you say?

Speaker 11: 2x.

Dr. Miller: It's 2%.

Speaker 11: No, 2x.

Dr. Miller: It's 2%.

Speaker 11: You're right.

Dr. Miller: It's 2%. When you go from 4% to 2%, it's 2%. If I'm trying to sell a drug. If I'm trying to sell Tide, If I'm trying to sell any product I'm going to tell you that's a 50% reduction. You're going to say this is a great drug. 50% reduction. It's a 2% reduction. You got to understand this concept, because nobody gets it. Even the New England Journal came out about 5 or 10 years ago, and they said, "You got to stop with this relative percentages change and go to absolute. Nobody will do it. Because you're not going to sell any drugs this way.

Here's the deal. How about I show it to you this way. Can you see the difference there. All I did is I changed the scale. Here's it's 5%. I just changed the scale. Can you see the difference there. No. Now you can actually see there's hardly any difference there. I just changed the scale. That's all I did. So, I want to sell this product great. If I don't want to sell a product, I'm going to show you this one.

This is point number three I want you to take away. Relative versus absolute statistics. Here's the deal. Here's why I'm telling you this. I looked at all the statins studies. These are all the major ones. I've gone to a number of lectures. I was trained into starting a regional disorder center by Robert Superko. We went to a number of lectures. He's one of the best. He founded Berkeley Heart Lab. Great guy. Went through all the studies. What you come out with is, here's all the major coronary artery events. This is what you'll hear. 43% reduction. 67% reduction. 54% reduction. The average of all these studies here was 30%. Wrong. It was 1%. It's just simple math. It's just simple math.

You'll never see this here. All I did was I just took these numbers here and I just did the math on it. Here are the relative percentages. See 30% reduction. This is a great drug. No, it's 1%. Cancer, 5% reduction. No, it's nothing. All cause mortality. 11% reduction. Here was 28% reduction. 30%, 40% reduction. No, it was 0.5. it was n change.

In everyone of these categories, coronary artery events, cancer all cause mortality. Statins had no effect. None.

Speaker 12: No beneficial.

Dr. Miller: No beneficial effect. Here's the thing. Here's where I have to qualify it. It doesn't necessary mean that any individual, one individual in this audience here maybe it will

help you. In my estimate it has been, because I don't think anybody has really done this, my estimate has if I take everybody with elevated cholesterol, maybe about 20% of you, it might have some beneficial effects. The other 80% it's either going to cause you problem or have no effect whatsoever. 20%.

Then you go to this thing called intention to treat. How many people do I have to treat? How many thousands of millions of people do I have to treat to get the desired effect? But if you look at population. No effect. If you look at individuals, maybe there's an effect. Pretty astounding when I looked at this.

Let's look at the endothelial rejuvenation. We look at arginine, nitric oxide. There was a lot of things about arginine. I like certain products that increase nitric oxide. So, what's our goal? Our goal is to arterial reconditioning, rejuvenate, revitalize, re-energize, optimize. Nutrients orthomolecular approach as much as possible. Nutrients.

I prescribe statins probably never. I'll write the prescription but to actually initiate it ... The only time I'll ever write if the patient says, "I'm really nervous about my cholesterol." Okay, fine if you're really nervous fine. I'll give you a statin, okay. I'll give you a little statin. I hardly ever initiate a statin. I'll write it if someone wants it. I'll tell them lets start with a low dose and we'll modify that.

Let's look at this, high dose fish oils. Again, none of you are taking enough. You're all taking caps. You'll never get enough fish oil with caps. You will never get enough fish oil with caps. You're counting caps, you'll never get it. Count it up. I want my patients to get 5000 EPA, 4000 of DHA. That's 10 grams of fish oil.

It's the EPA and DHS want my patient, I want 800 - 1200 vitamin E, high dose [inaudible 01:10:31] protecting the brain. Vitamin C. A lot of vitamin C. Vitamin K, 15 milligrams. That's basic and I do that in every patient. I don't know if I achieve that but that's the goal in every patient. High dose fish oil rheaology. Red blood cells will not aggregate with high dose fish oils. How do we know this?

Every time we go for surgery ... Today, your surgeons have now gotten smart enough to say, "Incidentally, before you do this surgery stop your vitamin E, stop your fish oils, stop your aspirin, stop your nsaids." They gotten smart enough to know that this does thin your blood. Surgeons know this, internist don't. Surgeons see, internist think.

This is as basic approach. Then I look at Alpha Lipoic Acid. Is it really important triple action anti-oxidant. CoQ10. I try to take 200 - 400 mg a day. I used to think that was a high does. When I treat cancer I treat this with 1000. So, to me 200 - 400 is an average does. Mitochondrial function needs CoQ10. Estradiol is really important for arteries. Really increase vascular tone. It keeps you younger.

It turns out that estradiol is important for men as well as women for bones. Testosterone really important for health. Taurine. Taurine we're starting to play with

a lot more. Taurine is really good for your eyes. It's good for your arteries. Calms you down. It's great for your heart. Carnitine. I use big doses of carnitine. I can't get most of my patients to take enough carnitine. I take 3 - 6 grams a day. It's a big dose. Carnitine comes from the Latin word carne, meat. That's where you get carne. People are not getting enough meet will not get enough carnitine. Carnitine is essential for cardiac muscle function. Carnitine is really essential, and nobody is getting enough of it.

I do Ribose 2 - 8 grams. Ribose is a precursor to ATP. It's a substrate for ATP. So all of these and then benfotiamine. I'm really big on that. It's another activated form of B1, thiamine. Which is really important. This is sort of my large list here. If you're taking all of this you're in really good shape. Niacin, methyl folate, B12, B6, and Neo-40. Neo-40 is a different way to get arginine. It increases your nitric oxide. Really heart healthy.

Niacin, methyl folate. It's a 5 methyl because all of us have probably have this NTFHR gene. It's not folic acid. Incidentally, Steve will know this. I know this even when I was studying bio-chemistry, there's no such thing as folic acid. There's the family of folates. There's the 5-10 methylene folate, there's a 5-methyl folate, and there's three different pathways. Quite frankly, as good as I am, when I look at folate I've never been able to master it. Even the people who I know, some of my buddies, that claim to master it, no one has ever explained this pathway clearly. It's very complex.

Simply put, 5-methyl folate. I use folate pro, I use a lot of vitamin B12, little bit of vitamin B6, folate, and Neo-40.

Nattokinase, 100mg twice daily morning and night. Fibrinogen, prevents heart attacks and strokes. I take this routinely, morning and night. I'm bummed out when I hear physicians now having strokes and cancer and so forth. Physicians used to be really healthy.

Heart friendly exercises, resistive and aerobic. High protein, low carbohydrate. I'm really into the paleo diet, low carbohydrate, high protein. No smoking. Alcohol, we talked about this at dinner. I'm hammering people. Some people get it, some people dispute me. Alcohol. Steve was the one who said alcohol is a carcinogen. Didn't you say that? Alcohol is a carcinogen.

Now I know you think, okay alcohol, whatever. My theory is this, and nobody can do this. If I were to buy into alcohol being really healthy. [inaudible 01:14:45] case half of a small glass at night, I'll buy into that. Stimulate the gastric juices, settles you down a little bit, probably not harmful. Two glasses a night, not healthy. We talked about that at dinner, not healthy.

Stress reduction, meditation, yoga, love. Dental hygiene. Teeth are a big source of bacteria. I never believed this but that's why when a dentist says, "Oh, you got mitral valve prolapse, we're going to give you some antibiotics." Direct connection between your teeth and your heart. Avoid chlorine and chloramines. I have a filter on my

shower. I get all the chlorines. It's either chlorine or chloramines. We're being poisoned by chlorine. You'll actually get more chlorine by your shower than you would by drinking water.

I was nervous. When I went to dinner tonight they gave me tap water. I never drink tap water. The water in this valley, in this whole Bay area is poison. It's killing cats, it's killing everybody. It's poisoned. This chlorine and chloramines is bad stuff. My father in law worked in a chlorine factory. I can say this is a little exaggerate. He worked in a chlorine factory, in those days when I was in the ER, when he worked in the chlorine factory, kind of macho. We didn't have mask or anything, died of lung disease. Chlorine is a terrible thing. There's ways to get around it. I filter out all the chlorine and I never drink tap water.

Friends and lovers, social bonding. It was Dean Hornish that said, "that actually the biggest risk factor for heart disease is disconnectedness." Lack of connections. I worry about his in younger people today. Because all I see is ... I see. They'll be walking down the street like this. I see couples walking down the street like that. I hear now, teenagers are now doing this at parties. They're not even talking to each other. They'll talk to each other like that. Disconnectedness.

Is Facebook making us more connected or more disconnectedness. We're near the end. Look at this here. This is my routine. High dose fish oil, turmeric, ginkgo biloba and nattokinase, clean water. This is how ... This is my substitute for Coumadin. For all the anti-coagulants. To keep your heart from clotting. You have atrial fibrillation this is what I recommend. High dose fish oils will prevent red blood cells from aggregating. The turmeric, fibrinogen. The vitamin E, aggregation. The ginkgo biloba, they're just as powerful as aspirin and all the other drugs that are being given today for anti-platelet drugs. Nattokinase again, fibrinogen. Clean water I told you is really important. You need to be hydrated.

When I used to work with Whitaker in the 90s, his stock answer ... Every time someone ask him a question he'd say, "You need to drink more water." If someone asked him another question he'd say, "You need to drink more water." What happens is when you're sitting in our audience and like everybody leaving, they'll be going to the bathroom, they'll be leaving, because everybody was drinking more water and they'll all be saying, "Good they're flushing their kidneys out." Water is an active metabolite. Water is not a passive thing. It's for your disc, it's for your heart, it's for your brain. Your brain's mainly water. We need more water. Even I don't drink enough water. It's got to be clean water.

There is vitamin C in the heart. This also was a great presentation. I have to skip over that one. This is Ascoraine-9. This was something that was sort of a knock off of Liones Pauling of a way of preventing the LP(a) from becoming a problem. At this point I was going to say intermission. I was going to put some slides up here. Some of my own photographs, just to make sure you guys were still awake. We're rearing towards the end here.

The Ascorsine-9 is pretty good stuff, so LP(a) another way of looking at cardiovascular health, this is a good formula. Vitamin A, which I don't like. Riboflavin, Pyridoxine, Vitamin E, Folic Acid. These are not real high concentrates but I really like the Carnitine. I really like the Taurine, the arginine, the proline, and the ascorbic acid.

We can get this individually. Here's ascorsine-9. Here's the Omega three studies showing the HDL, and the LDL. Adhesion molecules. Fish oil really help with adhesion molecules. I want to go back one thing here. We're getting toward the end.

Every six months you're going to hear a new oil. It was ... I'm almost done. It's flax oil, then it was salmon oil, then it was krill oil, then it was [inaudible 01:19:40] seed oil. Now it's got to be New England Green Lip muscle oil. Actually it's Omega-7 oil. No, it's got to be this other oil. No, it's the Omega-7 oil because [Peskin's 01:19:53] says we're doing it all wrong. Every six months you're going to hit something different. I'm still sticking with pure fish oils. Not cod-liver oil, pure fish oils. 10 grams a day. The fish oils are still really important.

Greatest risk factor is disconnectiveness. Best way to reduce stress is love. I got that from David [Zobb 01:20:16] that talked here once. I said to David, "what's the best way of reducing all the cortisone?" He said, "Love." I said, "No, biochemically?" "Love." Again, will you still love me? Will you still care when I'm 64? When I was 25, it seemed a long way away. It's not now.

Hungry heart. "Everybody needs a place to rest. Everyone wants to have a home. Don't make no difference what nobody says. Ain't nobody like to be alone." Bruce Springsteen.

With that I thank you for staying awake and listening. There's some books you can read. These books here, here, here, and then read my book. Then go off into the wilderness. Thank you again.