[MUSIC PLAYING]

**DR. DANIEL E. FORMAN:** So my talk's a little bit different than Dr. Greenspan's. I'm a little bit outside the weeds. I'm kind of going up to a little bit broader elevation of some of the distinctive challenges-- as I imagine all of you deal with cardiac disease in your patients and particularly as patients get older.

Aging, as I'll describe, really is conducive to cardiovascular disease. And then some of the challenges that are inherent is that when you're dealing with a cardiac disease, there's more than just that issue going on for that patient. So, again, the perspective of disease in relation to age and the conundrums of the care that are almost inevitable.

So in my talk, I'm going to be focusing on the issues of demographics, which I've already implied are really quite relevant-- the fact that cardiovascular disease is entwined with the issues of aging. And so there is the issues of physiology, for which I suspect every one of you has been trained and you have a thought process.

But I would argue that it really goes beyond that disease. Because as a function of aging, as you just heard in the last lecture, there's other things going on besides the cardiovascular disease. There's osteoporosis, to be sure, but many other things.

And so then I feel very bound by the guidelines that tell me what to do that really don't incorporate aging. So I think it becomes a very significant shift in how we try to align the issues of our patients and what our patients are saying they want with what the precepts of care are that are coming from cardiologists and other thought leaders.

And so I'll talk a little bit about some of the topical themes that are evolving in geriatric cardiology. So I've been in Pittsburgh for about four years now. This is my fourth year. It was a complete shift of my career. I was a traditional cardiologist. And I shifted into geriatric cardiology.

And I am one of thousands of cardiologists that are doing something similar right now. Because they're recognizing that the demographics are relevant. So you all live with this reality that if you look at who your patients-- it's driven by the data on this slide, that the number of people that are 65 and over is climbing from year to year.

So this is a population, 65 and over, in America that was only about three million in 1900, currently about 50 million, soon to be 70 million by 2030, which is one out of five Americans. So I think only three million in 1900, 50 million now. That's an enormous change. And it really reflects the fact that people are living longer.

So it's not just the baby boomers. The baby boomers kind of compound that. It's the fact that people are living longer because the things that you're doing in your office, because of improved [INAUDIBLE] and hygiene and a variety of other very basic factors.

But what really is even more important than this slide is this one. It's the population of the old old. We just heard about the old old in relation to osteoporosis-- but the old old in relation to everything. All the socioeconomic burdens, all the housing, all the health issues.

The 85-year-old population is the largest growing demographic in the United States, in China, in India, throughout the world. We're suddenly facing this overwhelming burden of people for whom the data are very wanting, and for whom the complexities are very high. So this is really what changed my career and which is why I'm here in this talk.

So you see in this picture that the average age of life in America is about 80. It's much older in Norway. It's older in Japan. But what the implication here and there is that when someone who's 75 walks into your office, he or she has over 10 years of life on average in front of them.

So the notion that somehow we come and say, oh, you're getting old, it's really very insufficient as I see it. Because people have a long life in front of them, probably many of you in this room. The average 90-year-old has four or five years of life in front of them on average. Many will become centenarians.

So it's a very different thought process of how we think about your disease. So then you see Bernie Sanders and Barbara Walters in 2015 being interviewed on TV. And whether you like them or not, that's not the issue. It's that they're compelling. They're people. They have things to say. And they're taken seriously.

So does it matter that he's 75 and 87? Maybe, maybe not. But if that same day they walked in to our hospital and you said an 87-year-old woman with heart disease-- how does that really factor into your thought process?

And what is your evidence-based rationale for how you're going to treat her? And if think, OK, well, I know she has HFpEF, how does that-- do you have a driving thought process because she also has many other things going on, including perhaps osteoporosis? So how do you organize all that? And how do you respond to her?

Does she care about her BNP? Does she really care about that or does she really want to get back on TV? So this is how I think about my patients. And this is why it's a growing issue in the care among many cardiologists to be sure, is that all of our patients are Barbara Walters and Bernie Sanders.

They all have these complexities by a function of getting older, our waiting rooms are filled with older patients who are prone to disease. Age is the driving of risk for cardiovascular disease, but almost every other disease process, too.

So we really are challenged by how we take care of people that have multiple things going on-- changes of body composition, changes on how metabolism occurs. So this is really the issue that I think faces all of us. No matter what practice you have, I suspect aging is really creeping into your thought process.

Even if you take care of kids, you're dealing with grandparents and families of longer age. So these issues are pervasive. So, again, we talked about aging. And as I've implied several times, as you see here, cardiovascular disease skyrockets with aging. Women outlive men. So ultimately, it's more women than men that have cardiovascular disease in old age.

So we have disparities of gender that are compounded by disparities of age and complexity to the body composition. They all become our realities. These are the patients for whom we care.

So I was enamored for many years in my career by Ed Lakatta. He was at the NIH. He still is there, although he is about to retire I believe. So Dr. Lakatta is the world leader in the biology of cardiovascular aging.

And so he's often talked about the fact that as you get older, the metabolism of all of our cells generates a lot of inflammatory mediators, many reactive oxidative stress mediators. and that it has downstream changes, constitutional changes.

So here he's describing the phenotype of aging. So there's predominant changes that really distinguish even the healthiest 90-year-old from someone who's 50-- is that there's a stiffening of the vasculature. This change of the elastin, the collagen, the calcium, the proteins with advanced glycation end products smooth muscle migration. This is a complex physiology. This is the stuff of a seminar.

But the bottom line is you look at that picture of a vessel-- this is a pig aorta. But it's meant to highlight the predominance of elastin and it is a a sense of distensibility that it confers. And all that changes as one gets older-- as a fragmentation of the elastin increases of the collagen-- all these things are changing.

The endothelial cells, which are so important in generating nitric oxide and other peptides which have a benefit for inhibiting atherosclerosis, that starts to diminish in its productivity. So there's more atherosclerosis, more stiffening.

This notion of hardening of the arteries-- it's an old term. But it's still relevant. And it really changes the way blood perfuses into our brain, to our kidneys, and to our muscles. So it has all kinds of downstream effects. And so Dr. Lakatta has been talking about this for about 20 years.

Likewise, there's been an associated literature on the heart. What happened to the senescent heart, the older heart? So fundamental changes of the myocytes, they're stiffer, with changes at the cytoskeleton, the filaments, the titins. But also changes in the interstitial area. This is an even hotter topic of research that continues with fibrosis and collagen increasing.

Backstream effects into the pulmonary pressures-- so there's higher pulmonary hypertension increasing with age, changes in the conduction system, more chronotrophic incompetence, heart blocks-- all these things are the realities of just getting older.

You're older-- suddenly you have heart disease. Why? Because you're older. It's like people seem to be shocked, because they've been so healthy their lives. But this is the reality. It's inevitable. But this always has seemed somewhat wanting because why do these things happen? And it's really led to an even more vigorous thought process.

But just to just-- the big picture-- again, as I've just been emphasizing-- you are older. You are likely to get disease. Heart failure goes up with age, both HFrEF but also particularly, HFpEF-- Heart Failure with Preserved Ejection Fraction cause the heart's stiffer-- coronary heart disease, because you've lost nitric oxide and all these other constituents that inhibit the artherosclerotic process.

Valvular disease-- so aortic stenosis, HFpEF, AFib-- these all skyrocket as a function of aging. These are not diseases of 50-year-old people in large part. They're diseases of the 85-year-old patient we just heard about. And they all occur in ways that are very compounding upon one another-- peripheral arterial disease, strokes. You say go exercise. When people have peripheral arterial disease, these things become very hard as they kind of interact with each other.

What's going on in Pittsburgh is really, I think, particularly important to highlight because this slide highlights a literature that complements Dr. Lakatta's. There's a whole world now-- this is a schematic by Carlos Lopez-Otin. But it's part of a very big thought process where going beyond this notion of wear and tear and inflammation is that there's actually molecular mediators that we're beginning to understand.

And you're going to have a talk, I believe, by Dr. Finkel who was recruited here about a year ago from the NIH, NHLBI, because he's a world leader in looking at these mechanisms. So you say, oh, you're getting old, deary, in that patronizing way. But no, it's a science. This is a rigorous a science as goes into the thought process of cancer, or heart disease.

It's that there's changes on the telomeres and how the cells maintain themselves, changes in the way the genes work with epigenetics, changes in the way the proteins are configured with the proteostasis changes in the way the mitochondria work with [INAUDIBLE]. All of these things go through-- now we understand-- predictable changes that are potentially modifiable. So when you say you're getting old, it's that something biologic is going on.

So there's a really profound evolution of thought, of thinking about aging as something potentially modifiable. And it is very scary to say, well, will we live forever? That's really not my issue. Because I do think there seems to be an end point at about 120, no matter what you do.

But it's really can you live better? Can you really modify these things so that this notion of all the infirmities that we associate with age get postponed? Can we postpone the osteoporosis so that all these drugs you just heard about may be less relevant and all the heart issues that I just mentioned may be less relevant, because they get postponed in a way that really changes the whole course of disease?

And this is really where we're going in 2018. This is all becoming the reality. In the meantime, what you deal with today in your offices is something more akin to this. That your patients, I would predict if they're older, are going to have heart disease for all the reasons I've mentioned. But they're going to have heart disease in a context of complexity.

And this is the reality that is not caught up in my opinion, with most cardiologists. Because it's never just the MI it's the MI with AFib, with osteoporosis, with rheumatoid arthritis, with many other things concurrently. And so when you say, just take this one biphosphonate, it's really in this context of complexity.

So it's the multimorbidity. It's the polypharmacy. And there's this other issue of frailty-- very topical term. So it's cardiovascular disease and all this very broad context.

This is another Lakatta slide. And it's a complicated one. But I think I can describe it briefly, because he talks on the [INAUDIBLE] of this issue of aging. And he talks about the fact, for reasons they have implied, that there's a biology that increases vulnerability. And that it leads to cardiovascular disease. He's the cardiovascular disease leader in the NIH in aging. But it's multiple diseases. It's not one disease. It's an aggregative disease, of which cardiovascular disease is almost inevitable.

But what's really important is that he goes beyond that to say that it also includes something independent from diseases called frailty. It's a biological substrate. So this slide is really meant to highlight that there's a biological drive, that there's a wear and tear called allostatic load over the course of a lifetime that really drives the vulnerability to disease and frailty.

And that there's a drift in how our cells maintain themselves-- this fancy term, stochastic epigenetic drift-- that the template for repair and homeostasis starts to go through errors as a function of aging. And it leads to the disease. But it also leads this thing, frailty. What is frailty? Why is it on this slide?

It's this notion that there's an entity that's even more complicated in terms of our body wearing down. Let me just take a step back to look at the multimorbidity. These are CDC data. And it's meant to highlight, if you look at just the cardiac disease of individuals with the chronic disease, that among those that have all the cardiovascular diseases, that almost all of them have multimorbidity.

I like to highlight the heart failure on the bottom-- that those that have heart failure in the Medicare population have five or more diseases on average-- five or more diseases. So then if you talked about the Entresto or the spironolactone or the new drug du jour that's going to come along, you talk about any procedure for these patients, it really has to be in this context, which is really not in any of the guidelines.

And you say talk to your patients. How? How do you make these decisions clear to our patients? And so this is really now a predominant shift. This is being driven by the NIH saying it's not just cardiovascular disease with comorbidities dragged along the side, but we have to think about this as an aggregate.

So this is a big NIH RFA looking at these diseases in aggregate among those with these types of challenges.

Likewise, the medications-- so we say we have new drugs that we can potentially treat all these patients. We're so smart. But how do we really put them together? Cardiologists-- this is part of a paper that was written in response to an NIH workshop.

And the cardiologists-- this was published ultimately in *JACC*-- a cardiology journal-- but the editors really didn't find this compelling. We talked about dyads and triads of cardiac disease-- hypertension and heart failure. And they said, well, cardiologists can deal with that. They're smart enough. And perhaps, you feel that way about your offices in whatever practices you do.

We can put together hypertension and heart failure. We can even put together AFib, although that gets a little bit more complicated. But what the seminar or the workshop is really trying to highlight is that this is the reality. It's the osteoporosis with all of these things. It's with the statins, which confound. It's with the anticoagulation which confounds.

So how do we have a thought process that's really rigorous for all of our patients who are so prone to cardiovascular disease in this complicated context? This slide isn't bad enough, because it just talks about the cardiovascular disease in this aggregate context. What really is the reality too for our patients is that we compound it. We give a proton pump inhibitor so that someone can take clopidogrel.

We give more drugs-- there's with this notion of prescribing cascade. There's very little thought with the FDA in terms of dealing with the fact that pharmacodynamics and pharmacokinetics change as a function of aging, loss of lean muscle mass increases the fat. We tend to think about giving little doses thinking we're doing our patients a favor. But there's a whole cardiovascular literature we should be pushing the statins, pushing the ACE inhibitors.

So you're carrying mixed messages in terms of what we should be doing for our patients. And then I think what gets lost in this is really what are our patient's goals? you have heart failure, do you want this medication? It's a very abstract concept. And how do we really put this together with all the other medicines they're taking?

This is my thought process about frailty, because as I was mentioning, the fact that it's part of aging. It's coupled biologically to cardiovascular disease and other diseases. And yet, we don't have a really sophisticated thought process that's generalizable to everyone in this room or among your cardiology colleagues or among anyone in the health care system.

So what is frailty? And people argue about this. There's been literally hundreds of articles written about frailty. So a picture's worth a thousand words. So if you see this picture, these people-- this is a Google picture. It's not anyone I know-- they look frail.

So as a cardiologist, I'd say, well, if you put a stent in these patients, are they going to do well? I would challenge that-- I would be very nervous about putting a stent or giving them Entresto or giving them anticoagulation, even though they might be candidates from a disease-based perspective, which is the perspective of our health care system.

So then how do you have a thought process about not giving them these medications? How do you talk to them about this? How do you just define their risk? And I imagine everyone in this room may have a very different thought process.

There is consensus about what frailty is on a very abstract level. And, again, I don't know how this really translates to how you would talk to your patients. This notion biologically-- going back to that Lakatta slide-- a cumulative decline across multiple physiological systems. And that it translates to you as providers to diminished reserve to tolerate stressors.

So here's-- if they have heart failure, they're going to be really sick because they're frail. But likewise, there's increased vulnerability to adverse outcomes because there's things you might do. So if you do a procedure, these patients have the worst outcomes.

So you're, I would argue, caught between a rock and a hard place. I feel caught between a rock and a hard place.

So how do we look at frailty? And this is the real big conundrum. Because we can't really deal with it because it's very hard to define in a way that everyone agrees upon. So many of the surgeons have argued for years, I can eyeball a patient. And I can just look at them and I'll know. And there's literature that supports that. That a good clinician can do a lot with his or her eyeball.

But I've always been very-- in my career, in my evolution-- very skeptical about that. I think you eyeball it differently on a Monday morning than you might on a Friday afternoon. And you might eyeball it differently if you're someone in training versus someone who's been in practice 60-- for 40 years. So how do you really make this into something actionable?

So Linda Fried, I suspect you've heard the name-- she's been preeminent to this literature for years. 2001, this seminal paper talked about the frailty phenotype. So she based this on one dataset doing regression analysis. And she came up with this notion of slowing down, weakening, shrinking, inactivity, and exhaustion. And that has become the free criteria for the frailty phenotype, which has endured 20 years.

So why is that? The real reason I think Fried has been so entrenched in our thought process is that with her work at Hopkins and work from other people, they've really supported that with a whole biological thought process. Frailty is not just what you eyeball. It's the fact that it's really tied to a very robust science.

So again, this notion of inflammation-- but going beyond that with various other hormones and mediators factoring into this phenotype and it compounded by the wear and tear over the course of a lifetime. So there's lots of intricacies to the thought process. But it's really the driving science beyond the eyeball which is really made it so powerful.

So then how do you measure that? And so Fried has various criteria. You walk 15 feet. Other people say, well, maybe four meters. So how do you compare those? Weakening-- so she talked about grip strength. Well, how do you do that in your offices? Do you have the same equipment? Shrinking-- do you look at weight loss-- as how reliable? So people have argued and argued and argued about how do you even do the Fried criteria?

So then people tried to simplify it. Stephanie Studenski, who is in Pittsburgh, did this seminal work saying just gait speed. And she had a method. You use [INAUDIBLE] speed. You start when someone's standing still. And then you walk four meters. So you do four meters. And so she tried to make this a standard that we could all adhere to.

I would argue it's still not standard. It's still not something that people agree upon. Many other advocates, many people say sit to stand over 30 seconds. That measures strength as well as various balance factors.

Timed up and go-- you stand, you walk for a few meters. You come back. It's another way of looking at it. Hand grip strength as I've mentioned-- many proponents but many different methods arguing. So, again, if you Google, if you look at PubMed, you'll find hundreds in your health resources in where you are may accommodate them or not. And, people, it's not standard.

And so then how do we act upon them? Who do you recommend Entresto to? Who do you recommend to treat with NOAC when you don't-- based upon frailty when we can't agree about frailty is? And what's make it even more complicated-- I think this is important to emphasize, because I suspect you live with the ramifications of this slide-- is that there's a totally different thought process about frailty that's talked about in terms of deficit accumulation.

And this was championed by Ken Rockwood who is an extremely prolific, extremely bright, who's not talked about grip strength or gait speed. He's talked about deficits, hearing loss, various diseases, various infirmities that he changes from scale to scale based upon what data set he's using.

So if you look up Ken Rockwood and frailty, you'll find literally hundreds of different frailty indices based upon the different data set he was looking at. Who's going to be more likely to go to a nursing home? Who's going to be more likely to die from surgery? And so there's all these Rockwoodian indices called frailty based upon deficits.

And they really don't correlate with what Fried says, which is more of a phenotype based upon physical attributes. So the literature is rife with inconsistencies and controversies as people argue about what is frailty.

In the midst of this all, I think the ramifications are going before the science. Because Andrew Clegg in Britain, he divided the entire health care system of Britain into frailty using a Rockwoodian scale. And he's saying that the people that are frail shouldn't even get certain procedures. They're not candidates for certain kinds of surgeries, cardiac surgeries. Because they're too frail.

So the ramifications are enormous in terms of how we're trying to think about frailty. But as I keep emphasizing, the science is not quite robust. Many people think-- and [INAUDIBLE] doctor think like that maybe if we think about aging enough, if we think about the biology, we can find some kind of biomarker that will mark frailty-- that that will supersede anything we think about with grip strength and all those other parameters.

So it's an active thought process. But it really has not resolved. In the meantime, the fact is that people that are frail don't do well. And that's your reality this afternoon or tomorrow when you're back in your office. People who are older, particularly older-- frailty is not necessarily age. But it goes way up with aging and way up with cardiovascular disease and it's endemic.

So people who are frail don't do as well. So this is work by Tom Gill. And he's shown in many studies of thematically the same thing that people who are frail don't do as well. They never recover fully after they go to the hospital-- even if they get the stent, and they get the drug and they get treated for AFib-- they don't rebound to the same level. And they are more susceptible to future hospitalizations and to a spiral of decline.

And this is the reality for most of my patients. How do you really slow down that spiral? And it's not necessarily by being the most aggressive and giving them the most guidelines-based medications. It's really focusing on the composite. And who they are, the multimorbidity.

So older adults are inherently prone to cardiovascular disease. And this is the reality. You have the multimorbidity, the polypharmacy. They're wired. The frailty, the loss of muscle mass called sarcopenia, the exhaustion-- it's compounded by the osteoporosis you heard about-- more susceptible to falls and imbalance, because of all these issues. We make it worse often with the medications.

We push the cardiovascular meds. They destabilize. We've given them an anticoagulation. We really set up to fail. It's compounded by losses of cognition, which are insidious and quite endemic, losses of vision and hearing, changes of confidence, increases of depression, poor nutrition. This is our reality. These are our patients. This is bread and butter cardiovascular medicine.

So we talk about precepts of patients under care and all that, I would argue the guidelines do a pretty poor job right now. So this is one paper that-- I've written several papers that are somewhat similar waving this flag saying, wake up. This is so real. It's in front of our noses. And I suspect cardiologists don't see this as much as the internists.

Because the internists you're dealing with is with your patients on a regular basis. The geriatricians see this. And they wave that flag a lot. But I think it's really medicine, because of the demographics. It's that these are the patients that are most likely to be in our offices. And this is the reality of most patients. And it's not consistent with what the guidelines say is important.

So this is work with a colleague Mike Richards, Washington University, where we kind of went through systematically through the guidelines and showed there were just major gaps in terms of what we should do for an MI. What we should do for heart failure.

Every thought process is informed by big studies based upon younger people that are screened to exclude multimorbidity. Because they want data to focus on disease. But that is not the reality of the patients.

So we're really at a disconnect. But it's beginning to change. So this is not just a doom and gloom message. It's saying that, one, that I suspect that your thought processes in your office don't always jive with the guidelines and to validate the fact that your judgment is very important-- and that the guidelines are guidelines. They're really not-- they should not be rigidly applied.

And to say that, two, the guidelines are beginning to change. And I'm part of that in the way that I really enjoy. Because it comes from my gut saying this is so important. We're missing this. Actually, I just recertified for the cardiology board.

So Rick Nishimura at Mayo Clinic is one of the luminaries of cardiology. He is super brilliant. And if you do anything with cardiology, he is a voice and an image that will probably come up.

So I love this slide. Because it's one of thousands of slides that looked at the study for the boards. So this is NSTEMIs. This is a disease of aging. NSTEMIs go way up with aging, more than STEMIs.

They're particularly hard to treat in older adults with bad morbidity and mortality. And here are all the answers the Rick Nishimura answers-- knowing when to give clopidogrel versus when to give [INAUDIBLE]. And you have to memorize this to pass the boards.

So there's nothing about age here. This is a disease of aging. So when you talk about geriatric excellence in this picture, we are the proverbial square peg in round hole. Because cardiologists as a whole, as a culture are oriented towards procedures and medications and top science, evidence-based based medicine that is really excluded the thought process as geriatric-- the touchy, feely geriatrics, the touchy, feely dimension that you deal with your patients as somehow being irrelevant.

Yet it's completely relevant in terms of your patient's experience of what you've just done for them or not done. But really was the turning point for cardiology, I think, or is a turning point that's evolving now is the fact that TAVR really shifted the focus. TAVR was a change-- a replacement of the aortic valve. And it was targeting a disease of the elderly aortic stenosis.

So the predominant patient population is very old. And they're dealing with all the infirmities that I mentioned. And TAVR was particularly promoted for those that were too sick, too frail to endure surgery to replace the aortic valves. You could have this procedure that's done almost as an outpatient. People go home the next day. And it goes-- a catheter through the leg is the conduit through which a new valve is deployed.

So the theoretical notion from cardiologists, the procedurelists, is that this is easy. And I can do this for anyone. And so, indeed, the technology has advanced super fast. But this is a slide of old data already in the scheme of things for TAVR. 2014-- this was Partners' data. It actually reflects data was collected about 2012.

And it showed at that time, about a third of the patients that were in referred-- these are people that are about 83 average-- complicated patients with aortic stenosis-- about a third died after a year. And that's improved. And now it's very few die from the procedure.

But the other two graphs are pretty much the same. The changes of New York Heart Association class-- about 50%. The change of the quality of life measured by the Kansas City Cardiomyopathy Score-- about 50%. So this is a procedure that costs many $70,000. 50% enjoying improvement of New York Heart Association class and quality of [INAUDIBLE]. Is that a good outcome?

So I think that's really the conundrum. How do we look at frailty in a way that's actionable and reliable? How do we look at multimorbidity? How do we look at body composition that's relevant in terms of this cardiovascular outcome which is endemic with our older patients? And this is really where we have challenges.

So as we deal with cardiology in 2018, aging is really relevant. Because aging drives disease. It drives all this complexity of cardiovascular disease management. What do we do? So in Pittsburgh, Bob Arnold and other thought leaders in palliative care saying, don't go down that cardiology path, the bad cardiologists.

And so you as the internist should steer your patients other ways. Palliative care makes sense for many patients. But is that right for all patients? And how do you describe that-- this sense that they're not going to get potentially the valve or the other treatment and that medication?

Is that always a good thing? And how do you really discriminate the right from the wrong? How do you have the language? And do we even know? Do we even know if some patients are going to do great with that valve and some won't?

Do we modify the procedures? This is what the procedurists will say. This is their bread and butter. They'll say, I'm going to deploy that TAVR better and better. I'm going to deploy that stent. I'm going to get the medication in the right dose. Is that going to do it? And that's their culture. Because that's a driving on the paradigms from which we now-- we function.

So you have a lot of the people who are empowered saying, I'm going to refine this. We'll do it faster. We'll do it better. We'll just make it-- but that TAVR schematic I showed really raises questions. 50% of the people didn't get better even when the TAVR went just fine.

So I think we really have to go beyond the disease to think about what's inherent with the patient. And we don't have that measure yet. We don't have that index. My thing, which you've heard from the introduction, is I focus on rehabilitation. I'm a big believer that we do not use cardiac rehab anywhere near enough for the patients that need it most, the old patients.

Only about 15% of eligible patients who are older go to cardiac rehab. I feel like that's a tremendous underestimate. Because we don't refer many diseases that should go to cardiac rehab. We have these very rigid criteria that are really counterintuitive to me about why we don't take HFpEF into cardiac rehab, whereas we take HFrEF. It really doesn't jive with the clinical reality of my practice and I suspect many of yours.

And can we enhance cardiac rehab so we want to drive-- and do it safely at home? Can we use TeleHealth? Can we use other methods so that we can really make it safe and beneficial for our patients? And can it be reimbursed?

And so we have all these challenges that make it really not worth the while for most physicians and providers to even deal with cardiac rehab, it's so difficult.

And finally, this notion that interrelates is shared decisions. How do we relate this to our patients? The big push now in cardiology is not only technology, but language. How do we really talk to our patients to discuss these things and start to really bring them forward in a way that's actionable?

This issue of MACRA which I suspect you will live in some way, shape, or form or soon will, is driving this. Because MACRA is this whole notion of health care in terms of increasing the value of care. And it's really emphasizing this abstract concept of value.

How do our patients value what we do? How do we generate a sense of value? And we're being measured by this. And it's going to actually generate some sense of our remuneration. And this notion of value is driven in part by cost, but also by what patients say. And there's been efforts to inform this.

So this is one of the many efforts that was done by the National Committee for Quality Assurance saying, what do our patients say is important? Do they say it's being [INAUDIBLE]? No. They really have different things that they talk about as being commensurate with value. They talk about things that are mostly to do with function.

So I really think about function as a really driving endpoint. It's not that clean artery after stent's put in. It's really whether someone can walk up the driveway, whether they feel independent, whether they can play with their grandchildren in the way that they feel confident about. That's what people really care about. And we don't have ways of saying, if you take this medication, you're going to get there.

We kind of hope. But we really don't have-- we don't look at that in terms of our trial data. We just infer it. So in terms of themes of current care, I just want-- just to almost recap my emphasis, I was not part of this guideline, but it really factored into my thought process. This came out this year-- the hypertension guidelines.

And I think you'll have a lecture dealing with hypertension. But I'm very reactive to this guideline. Because here it says 1A, 1A data, which is you should be doing this. And the granddaughter is going to be angry at you if you don't do this for your old patient. He's going to call you and give you grief. Because treatment goals for a systolic hypertension is 130 or less.

You should be doing that even for that patient that has osteoporosis you just heard about or the susceptibility to the falls you just heard about. And certainly, for the patients that I treat-- take care of that have all kinds of cardiac issues to say, 1A data, lower that blood pressure.

But I am concerned, as you just heard from Dr. Greenspan, about the fall that patient can have and all kinds of other things that really might be inadvertently potentiated by treating so aggressively. And the guidelines really don't give me too much flexibility. They have a level 2A consensus of the experts-- a very weak recommendation to use your judgment.

It really pales in comparison-- if they have comorbidity and limited life expectancy-- I don't even know what that wording means. You have to have both. Somehow we can't have one or the other. And then it's only reasonable. It's a much weaker recommendation.

So just very briefly-- because you're going to have a whole lecture-- but there was a SPRINT trial, that I suspect is familiar to many of you, that really kind of informed this. And SPRINT had two arms going. One that treated the blood pressure less than 140, one that treated blood pressure less than 120.

And then the general population-- a population was middle-aged, there was a 25% primary outcome reduction dealing with this composite of MI cardiac ACS events-- stroke, heart failure, or death. But then all [INAUDIBLE] mortality also at 27% reduction.

But then and looking at a subset that was close to 80 of about 1,000-- excuse me, about 3,000 patients. This data showed that the primary outcome and the secondary outcomes were preserved even greater-- 34% and 33% reductions respectively.

And in this same trial-- these are very bright people. They did a measure, a Rockwoodian measure of frailty-- so based upon a composite of deficits, they said even the frail patients, as you see here in the far right, gained tremendous benefit from this intervention.

So we should be treating even our frail patients based upon 1A data to what these treatment goals. Is that what you feel comfortable about? I think that frail patient I showed earlier-- and I do-- is that the goal I have for them?

So I want to highlight, I think, what you will live with is that there are benefits. There are conceptual benefits because we are wired to develop cardiovascular disease as a function of age. This wear and tear, all these mechanisms from the Carlos-Otin schematic-- they drive the susceptibility to us and to all of our patients.

So if you treat aggressively, there's the theoretical logic that's consistent with SPRINT that you can reduce the events and mortality and morbidity. All these things can improve.

But on the other side of the coin for another subset-- really probably the dominant subset of our patients-- you increase risks with hypotension and function and fatigue going down the wrong way, increasing frailty and disability. And all these things that could go wrong are really quite part of the spectrum.

These data by Adam Brest that came out shortly after the guidelines were changing talked about the fact that once they're implemented that the likelihood of all these clinical sequelae is almost inevitable. And they're going to go way up. Because within SPRINT, the smaller trial, they said, well, they weren't significant. But Adam Brest says when you compound them by all the population they're going to occur.

So this is the reality if you use guidelines to drive your care without a thought process. And then just to highlight-- one last thought in SPRINT if they-- cause this drove the guidelines. So this is why I think this slide is so important. This is all 75 [INAUDIBLE] patients and they're waiving the flag, saying it's good for us. Who did they exclude?

They excluded patients with stroke, diabetes, all those in nursing homes with dementia, heart failure, anyone with functional limits, homebound, orthostatic hypotension. So about a third of the patients I suspect will do well with that blood pressure. But 2/3, you're going to have the daughter yelling at you.

So this is, I think, a concern that I have as a cardiologist in this community that thinks about these things. So I think we're really challenged. With our patients, with all of your patients, what do you want to do for them? You heard, again, in the last lecture-- we overlapped, Dr. Greenspan and I overlapped-- time to benefit.

If you're going to treat, you really have to think of the window. You're going to treat-- because I talked about blood pressure. But I am on the statins guideline. You're going to hear about that in a few weeks. It's going to be released. And I can tell you that this is specific language about who not to treat. Because statins are good for some people-- maybe a proportion just like there is for hypertension treatment aggressively. But then for others, maybe it's harmful.

So thinking about the time to benefit, the time to harm, the fluctuating health status of all of our patients, the comorbidities, the medications, even the fluctuating circumstances like as you've heard month to month, things change. The diet's relevant, alcohol use, which is another huge problem among the older populations, the ambient weather.

If it's very hot, if you're treating aggressively for blood pressure, that could be a disaster, the social supports or lack thereof. And so one of that very topical themes in cardiology right now is to de-risk. In middle-aged patients we think about all these risk scores. Who should we treat aggressively based upon their BNP or their proponents? We [INAUDIBLE] risk.

But, again, by as a function of aging everyone is at high risk as a function of aging. Once you are over 75, you're pretty much vulnerable for everything. Does that mean that everyone should be on every pill and every modality to modify risk? It becomes overwhelming and really quite counter beneficial. So can we de-risk?

So this is-- again, this is the tip of an iceberg of a thought-- I'm ending with it-- but that within the cholesterol guidelines, I would say there's a lot of focus on the calcium score. Because even in the older population, the bioimage study was a study of about 6,000 people.

And about a third of those that were over 55 in men, 60 in women-- about a third of that population had a zero calcium score, for which the benefits then of treating with a statin become really nil.

Why are we giving these patients statins when their likelihood after having a calcium score of zero, their likelihood of having diseases is so minimal? So the thought process there is to do less when we can de-risk patients-- de-risk patients.

And this overlaps with the whole thought process of what drives aging. If we can really refine the notion of biomarkers, knowing who's going to really be at risk as a function of aging, then we can treat them appropriately. But if we know who's at low risk despite age, because of some kind of more biological perspective, we can really change the whole paradigm of care.

So my work in cardiology is really part of a broader shift. The NIH is really endorsing the fact-- and it's particularly the National Institute of Aging-- that they're oncologists, endocrinologists, surgeons, emergency room medicine people, trauma-- that are really trying to look at aging as a very big factor of how we take care of patients.

I want to just anticipate Toren Finkel's talk saying that we really-- in Pittsburgh, Mayo Clinic, Harvard, all over, people are really thinking how can we look at these mechanisms of aging? I'm going to end with one thing which kind of hit *New England Journal.*

So you've all seen this I suspect or heard about it on NPR or the equivalent, that Paul Ricker looked at cardiovascular disease and said if we can reduce inflammation-- that has been his whole career, looking at inflammation-- we can modify cardiovascular disease.

So he took people that had known cardiovascular disease who happened to be in their mid 60s, and he reduced inflammation using a drug, not a statin, nothing to do with cardiovascular disease directly. He reduced it using an IL-6, a very fancy inhibitor, modifying inflammation. And he showed reduction of cardiovascular events. *New England Journal*-- big stuff.

What makes it even better is that this same week in*Lancet,* he published this. It's showing the same patients, he reduced cancer. And *New England Journal* won't even put them together. They said, no. We're disease specific. We don't want to have two things like this together.

But this is the beauty of it. This is the aging issue. This is the whole paradigm shift. Because it's not just treating one thing, it's treating the composite. And so if we can treat aging, if we can treat inflammation in this case, which is such an inherent part of aging, yes, we can reduce cardiovascular disease. But we can reduce cancer, perhaps.

And the work that we're doing right now with Dr. Finkel, we're looking at this to treat frailty, to improve mobility and improve body composition. Counterintuitively, we give this fancy pill to do all these things. But if we can treat elements of aging, maybe that's where we're going to go.

So in summary, older adults are inherently prone to disease. And our current treatment patterns that we all are bound by in terms of the guidelines and pressures that we get from administrators and such, are really not complete.

There's much more complexity. I really think you have to think about cardiovascular disease and every disease in a context of aggregates. And that the medications are not necessarily right. Because they're not really studied in this kind of complexity.

Frailty's relevant, but it's hard to measure. Cognition is relevant, because it compounds what patients really want and the notion of adherence. But we need improved decision tools. And that's actually being prioritized by the cardiology world as really a big part of how we administer best care-- and that there's a real big future in terms of geroscience in terms of how we might change care. Thank you very much.

[APPLAUSE]

Thank you, Dr. Forman.