Breakthroughs On Treatments For Progressive MS Transcript

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Introductions and Summit Context

Everyone. Again, thank you for coming. Being part of the multiple sclerosis and Neuro immune Summit. I am here with my very good friend, Doctor Ken Sharlin Now, Doctor Sharlin and I have known each other for many years.

Really, a decade now. And Ken is a board certified neurologist who's also trained in functional medicine. Integrative medicine. He practices functional medicine, does research, is a cutting edge neurologist, and he is where I send my complex neurological patients and they really get superb care with Ken and his team.

So can I am so pleased that you're here. Is there a thing that I left out in your intro that you'd like to stress? No. Thank you so much. Appreciate it.

Okay, so let's get right to this. You know, I know that people are very, very excited about some of the new DMT that are coming out there. And one of them is the BTK inhibitors.

I wonder if you could comment briefly about that drug type and whether or not it's worth all of the excitement that is happening in the conventional world?

Yes, absolutely. And I want to sort of broaden that if I can, and put it in context, because, I think, you know, we will really help a lot with the discussion and where things may go over the next 40 or so minutes.

You're interviewing the folks from the Octave Bioscience Group. But I know that was in the two point. And that particular test is very important. I think even in looking at these BTK inhibitors, we're looking at, we're talking, first of all broadly about what's called disease modifying therapy.

And, you know, in the management of the disease.

DMTs and MS Disease Activity

And as we broadly divide the approach, the clinical approach into symptoms management, maybe that's pain or spasticity or, you know, bladder function issues and things like that.

And then drugs or treatments. Many of the new treatments or biologic agents, they're not just the standard sort of chemical drugs, but they are really aimed at attempting to in particular, slow what we call sustained disability progression.

There, you know, as you know, and you're a researcher, you do clinical trials, you have to define a primary outcome or a clinical trial. And, historically, if we go back to the very first drug that was introduced in the United States in 1993, that was beta serine, and at the time, the study really just looked at relapse rates as the main outcome.

But as time went on, it was realized that while we should be looking at relapse rates for people at least who were relapsing remitting Ms., that there are other measures of primary outcome that may be more important that we really have to put first in terms of determining if a treatment is effective.

And so there's been a shift toward what's called sustained disability progression. And that's based on a gold standard and in sort of a measuring stick of how a person is doing with them.

That's called the ADFs or Extended Kursk Disability Status Scale. And it's largely a glorified physical examination in which different aspects of the exam are given points.

It's heavily weighted toward gait or gait dependance, meaning that are you walking independently? Can you walk 500m? Or do you use a cane? Do you use a walker or are you in a wheelchair?

If you're in a wheelchair, can you still transfer things like that? So those sorts of questions weigh heavily in the EDS score. All right. So if we look at broadly all of the different agents and there's some like 25 or 26 agents currently there in several different broad categories, which we can certainly talk about.

But if we use this test, the M. S. disease activity tests from Octa Bioscience, which really puts a microscope on mass at the cellular level, which very different from just eyeballing an MRI and saying, how's that person doing?

There's a lot that can be missed there. What we see is that the people who are on what I call B-cell depletion therapy, so that's sometimes it's rituximab, but that's not technically approved for multiple sclerosis in the United States.

But it's ocrelizumab or oak reverse oh for tumor NAB, which is key symptom. And then the newest sort of reiteration of for to nab a rather a Ocrelizumab or Brian V.

So when we look at the and that's disease activity score, I find and I have a lot of patients with Ms. in my practice and they all get this blood test when they come in.

And by the way, can't just go to your local quest station or LabCorp or talk to your primary care doctor and get this test. This is an exclusive relationship between the clinician like myself and Octave Bioscience to be able to perform this test.

So what we find is that the folks who are on those B-cell, depletion therapies have by far the lowest scores. These drugs are in red. And we should clarify that low scores are what you want. Yes.

You don't want a high score. So the octave you want to have a low score. So continue can. Yeah. So it is, it is measured from 1 to 10. So essentially I don't think there's a zero but one to, you know, or is low or point 5 to 7 is moderate.

And then, 7.5 to 10 is high and what's wonderful about this test is not only does it give you kind of a, a snapshot of your current level of disease activity from an immune perspective, how active is that immune system in attacking my linen nerve cells in the brain from a sort of a myelin breakdown perspective, and then also from a what they call neuro axonal integrity, which is the degenerative component of them that's independent from the destruction of myelin.

But the people on these drugs have very, very, very low scores. And I think the folks who own the patent on key symptom are offered to Mab, which is Novartis Corporation.

It can project out, expect, you know, relapse rates over a long period of time. And that this drug can suppress relapse rates as long as 20 years. So you know, these are very powerful treatments, but they do have some downsides in that we're talking about attacking or really destroying what are called B cells.

These are the immune cells that make antibodies. But these are the cells that come from your bone marrow. They circulate around in your bloodstream. They're there, among other things, to protect you against viruses and bacteria.

They produce antibodies, but they engage in normally do engage what are called microglia in the brain. And that's thought to play a major role in Ms..

And by the way, these cells get infected with the Epstein-Barr virus, which is probably a major trigger. We know people who have EBV are about 30 times more likely to develop than this.

So bottom line is these are very effective drugs. But they do have some downside insofar as you know, in

functional medicine we talk about like root causes or upstream versus downstream.

And when you inhibit those B cells and not all B cells just these auto reactive ones, what are called CD 20 positive B cells. Not surprisingly, there can be some consequences.

BTK Inhibitors and Progressive MS

And that is, you know, more often you have infections, upper respiratory infections, bladder infections, things like that. And since Ms. is a central nervous system disorder, meaning the brain and spinal cord, not your peripheral, nervous system, the nerves, like your immediate nerves, the carpal tunnel nerve doesn't have anything to do with that.

Question might be, well, what if we can go even more downstream if we can really engage these B cells when they are in the central nervous system and stop them from interacting with the central nervous systems, own, immune cells, the microglia, rather than mess with them when they're out there circulating in the periphery.

But we have this enzyme. We all do. It's in our in our, immune cells called a Bruton tyrosine kinase inhibitor. If this particular enzyme is blocked, essentially it, it appears in, you know, an animal models at least that we can inhibit the activity in the central nervous system, but still protect the immune activity in the periphery outside of the central nervous system.

That's a great theory. Right. So there were about five different compounds that have been or are being in different stages of testing and clinical trials.

And we've recently gotten some results which are very interesting and in some ways very exciting, but and with some surprises as well. So we've seen and or these are like early press releases, you know, doctor walls, by the time this interview is aired, it will be past us.

But when we're now interviewing, there's a major European meeting on our that happens every year called electrons and atoms is coming up in about a week.

And and these big, big meetings are platforms for the the researchers to, sort of roll out, to show their results, to unveil their results. So we have two main, of the of the 4 or 5, we have two main BTK inhibitors.

One is from Roche Pharmaceuticals, Roche Genentech, and the other one is from Sanofi, Sanofi, Genzyme. And actually we've been involved with researching both of those.

There's there was another one from Serrano, which I'll touch on in a minute. I believe Novartis has one, and I think there's one other one. I don't know the status of the Novartis one.

So what did we learn? Well, we learned that in the peers that in both the Roche compound, which is called fen ibrutinib and the serrano not serrano Sanofi compound, which is called toma Bruton in that the BTK inhibitors appeared to have a very significant effect on people with progressive multiple sclerosis.

There's a caveat here. There are very few proven or approved drugs that are effective in this, but we've got stage of then this. And you know, typically classically we talk about MERS as having a relapsing remitting stage as, you know, where there attacks.

And then the person may be recovers. They may not recover entirely. So it's kind of like climbing steps where you're going up and your level and then up and your level and then up and then your level.

But in this case, these changes leave people with more and more disability, potentially over time. And there are many drugs approved for relapsing remitting.

And that's there are no clinical trials today that study new compounds for relapsing remitting and that's, that are

placebo controlled. All of them have what are called active controls.

And I call this sort of the like the who are we going to beat up on. And the popular drug to test these new compounds against is called obagi yo or Theraflu.

And which is a fairly low efficacy drug. And what has happened, especially what we saw with the Serrano one and then with the Sanofi Tola Brut nib. Is that it?

This study, this large study failed to show that there was a difference in efficacy between objeto and these BTK inhibitors, even though they're sort of close cousin predecessor.

The cell De leaders like Oakley's Mab were highly effective, highly effective. However, what both Roche and Sanofi have found, in different stages of clinical trial, Roche is in state two is that these drugs are effective in shutting down new acquired lesions in people who have progressive mass.

So it's really important to understand that. I can tell you Serrano made a decision not to just shut down the drug. You know, maybe it was effective from a, you know, compared to if you look at the objeto group, that was equally effective.

So we couldn't show a difference. But if you're going to develop a new drug, that's a huge financial investment and there's no point in rolling out a treatment that's not any more effective than something that's already out there, probably a lot less expensive.

So they just shut down their whole program. Now, the same thing happened with the Sanofi tola ibrutinib. No more effective than, turf amide. But we have now, you know, tola ibrutinib and then a brut nib that appear to really stand out as promising treatments for a progressive mouse, which, like I said, even if you look at Oakley's an ad that has an approval for progressive than that primary progressive, the benefit is, is very, very marginal.

So a stop for a mo, because I really want to highlight this, because I have secondary progressive M. S. in most people with relapsing remitting Ms. assuming that you continued to live, you will eventually convert to this progressive phase of the illness where we're not having relapses.

But the concern is this relentless progression of worsening disability. Yes. And so this is a big deal, that there are a couple of drugs there aren't yet approved yet.

So you can't we don't want people to think that I've had these kind of questions. They people you know, everybody's on Google. Let's watch okay. That's great.

Our patients are sort of more informed, although there's something to be said for the experience and the knowledge that we have to be able to interpret the data correctly.

But you can't get these drugs or anything, but they will be available. I would say, you know, just looking at timelines of other drugs, we were involved in bringing key symptoms to market.

We've been involved in bringing the new Alzheimer's drug to market called Donanemab from Eli Lilly. So from the time that the studies are finalized and, paperwork goes in to the regulatory agencies, the FDA, and then eventually what's called CMS centers for Medicare Medicaid Services, you're generally probably looking at about, I would say about six months or so.

It's what I've seen. They undoubtedly will probably be. So they'll eventually be approved. And for people with the progressive phase of the illness, this would be a useful drug.

Now, you and I both have the perspective that while drugs may be FDA approved for your condition, it's important to also go beyond the drug in terms of what is it that we can be doing if we have progressive Ms.

or relapsing remitting Ms., because maybe the listers will have relapsing remitting Ms. from your point of view, in addition to a highly effective drug for either relapsing remitting or for progressive, us, what else is important to be doing?

Well, I think you know, I want to I want to if I can very briefly circled back around to some observations that are indirect from these studies because it's really important.

Look, what have we learned? You know, about and this besides just does the drug work or not work? And there was a wonderful presentation I actually sent to yesterday.

There's a lovely, platform that doctors have access to, which is like watching a, academic lecture every day on different relevant topics, say, in this case and in this.

And it's called, view, Maddie. But anyway, we look at and that's over a lifetime. So we talk about the average age at diagnosis as being somewhere around 34 years old certainly is pediatric.

And that's that's a whole other issue. You know, there's a whole other set of issues to deal with when children get M. S., but they certainly do. But what's very interesting, and I this is something I learned yesterday, and really never given much thought to it is if we look at just the population in the United States and we just break down the population by age, we're the largest, you know, it was say, you know, what's the largest population like 25 to 35, 35 to 45, 45 to 55, 55 to 65 and so forth.

So what we talk about, the point is, while we talk about M. S.

Age, DMT Decisions, and Functional Medicine

as being a disease that affects younger people, in fact, the largest group of people affected by M. S., this growing, shifting population that's just getting older and older and older, it's not the young people, it's the older people.

Right. Because that's the demographic of the United States. And so we are dealing the the average age, the largest number of people in that in that demographic, age stratification, you know, strata is something like 48 years old.

And why that's really important is and this relates to your question is, as you know, whether I'm working with someone with Alzheimer's, whether I'm bringing some of the Parkinson's, whether I'm working with some with ALS, or unless these are not one disease, right?

There are common manifestations that I'll quote. Dale Bredesen, our friend, who says, you know, when you look at Alzheimer's disease, it's and think about all the causes.

It's like thinking about a roof with 36 leaky holes. And so in order to tackle that individual help work, we're put that person with, say, Alzheimer's, mild cognitive impairment due to Alzheimer's.

We can't just target one thing, we can't think of all timers as if it's just one disease we can't think of, then that says if it's just one disease. So what is observed?

And this circles right back around to those B-cell depleting therapies. And the Bruton's tyrosine kinase inhibitors is that earlier in the younger people with M. S., right, those 34 year olds, even those children, the pediatric cases that this disease is much more robustly expressed in the peripheral immune system, that it is a very immune driven disease.

In fact, I just this past week saw a couple of new folks for my brain tumor program, like functional medicine program, very aligned with doctor walls.

And now they're sort of very young people with very, very active disease. Lots of and lots of lesions, lots of

enhancing lesions. Fortunately, you know, from a from the disability perspective, they were they they were good.

But that does not speak for a good prognosis. And we certainly need to jump in on that. So if we think about that, it didn't say that those older folks, those 48 year old plus.

And by the way doctor walls, it led me to do some math. I hope it's okay. I know how old you are. Yeah. That's it's do you want to say it publicly or whatever?

But I went okay. Your your video that went viral and kind of puts you on the map 2011 minding your mitochondria. And I said, how old was Terri Walls when she did that video?

How old was Terri Walls when she described her personal transformation? 2005 2006 in that time frame of going from the tilt recline wheelchair to riding your bike, well, you were exactly that age.

And what we learned, at least back then, back 20 years ago, 25. What we've learned about these older individuals is that the disease shifts from being more robustly peripheral immune, those active B cells crossing getting into the brain, and that that blood brain barrier that's become leaky, like a leaky gut to a disease that is more internal, more compartmentalized, internal meaning the brain.

Right. More involving mitochondria binding your mitochondria, oxidative stress. Right. Microglia. So now when we go back and look at these BTK inhibitors and say, why did they not work in relapsing remitting M. S..

But they worked in progressive then. But the B cell deplete ers, which are really kind of related to BTK inhibitors. But they're more a little more upstream if you will, because they mess around with your peripheral immune system.

They're really effective, really, really effective in these younger people right, with relapsing remitting M. S.. So I think that's that is so super important to understand that when we say what can we do from a functional medicine perspective, we're thinking about the big picture that everyone is unique and everyone is different.

And and age does make a difference. Yeah. Age. Age, Which then, you know, gets fixed. The next question as we get older. Yeah. And people ask me this a lot.

You know, I'm getting older are the risks of the DMT that I'm taking now outweighing my benefit because I know that relapses decrease over the age of 45, particularly over the age of 55.

And at what age should I think about coming off? And I think that's a complicated question. And I don't know that we have good research to guide. And yeah, I tell people I want to do the functional medicine stuff to get you all fully tuned up.

And my conventional neurology colleagues are we have some very interesting debates over what is the age where, and they aren't doing what you and I do with the functional stuff.

They just say, okay, well, we'll stop the drug because you're too old. What are your thoughts about stopping DMT and how does age and your functional medicine approach interplay with that?

Right. Yes. And I apologize because I didn't fully answer your last question. So we'll answer that now. And so, you know, as I've spoken for many years for you on what we have learned about discontinuing disease modifying therapies, and since then there's been at least a couple of clinical trials.

There's one called disc disco. It could be discoms, but it's disco. M. S., the discoms trial, which was a multicenter, randomized, single blind phase for Non-inferiority trial examining the risk of new disease activity in multiple sclerosis patients who continue versus discontinue disease modifying therapy.

And, you know, better or for worse, what the trial found is more or less what we've observed previously, which is that age does age is really the only major ten permanent that we have.

And I would be for it. And we have to again treat everyone different. Right. You can have all the hope in the world and that's really important. But you also have to have objective data.

So we do want to circle by the way back round to that octave test which none of these included. Right. But some of the strongest data really looks at people who are older than 55 and even 65 or 60, just depending on the trial, where if that person has been clinically stable, there's been no changes on their MRI of their EDS score is stable, no changes, no disability progression.

Discontinuing that drug really only leads to about a 10% likelihood at worst of relapse or or recurrent disease recurrence or progression. So age is really the main factor.

But if we flip it around and we say okay, your newly diagnosed with, then that's how's it going to go, you know, what is the crystal ball. Tell us. Well we know that well and this is more prevalent among women.

Men can do much worse with them now. So they've spinal cord lesions if they have what we'd call posterior fossa lesions meaning like cerebellum, brainstem.

If they have just old, large, large lesion burden, you know, this degree of their disability at the initial presentation. These are all major considerations when when predicting how's it going to go for that person.

And we really have to take that into account when we talk about functional medicine. Because in my you know, I've taken a bit of a shift in that, you know, I was a conventional neurologist and then I was like, wow, functional medicine.

This is what I want to do. And then I realized that both toolboxes have limitations. But together they are very, very powerful. And as long as we're approaching each individual person on that level that you are a unique story.

You are in a unique narrative. You are. And this is not the same as doctor was in this or my this or another, you know, person might have M. S. then we can say, I think it would be okay if that's what your motivation is, to take this wall styled approach and hold off on disease modifying therapy versus another person.

And I'm going to look at everything and say, I'm going to be honest with you. And I'm even though look at your readiness for change, your willingness to make a commitment.

Have you been able to sustain that? Maybe you're in this disease activity score right, because that's not even taken into account in any of these studies to say, look, you should always be following a wall style approach.

There is no reason that you shouldn't, you know. But for you, I'm also going to recommend disease modifying therapy. And then we're going to track that. Right.

We can track your disease activity, score your MRI, your physical exam, all those things over time, your 25ft walk, your peg hole test, all these things that we look at, you know, and then we can say, okay, done really well.

Everything's stable. Where are you in your heart and in your mind in terms of stopping your drug? Because I can't tell you with 100% certainty where you're going to kick back into, you know, gear in terms of having active disease.

But I feel like you've done your homework and you've created a solid foundation for yourself. And if that's what you really want to do, I'm going to support you.

Illness Narrative and Patient Readiness

But we're still going to measure. We're still going to track. That is so important. Faith is very important. Hope is very important. Belief narrative is very important.

Very important. But we still have to be sort of transparent and honest with ourselves. And if the numbers are going

in the wrong direction or the MRI is going in the wrong direction, you know, you need drug, you need drugs, but you should still be following the walls protocol.

And to everyone who's listening, I want to reinforce that yes, for some people you can do really great with a functional best approach. No drugs, everything stays stable and that's fine.

But others, you'll benefit so much from taking the disease modifying treatments can use them in his practice. I can't prescribe those drugs because I but I send them to the back to the neurologist.

It's a strong encouragement that yes, I think you should do both diet, lifestyle plus DMT. Anyway, in that circumstance you might do perfectly fine without DMT.

But everyone, everyone who's listening, please be sure you're getting your MRI eyes. Senior neurologist making a very informed decision about how DMT play in your care.

You mentioned a nother concept. The narrative. And you sort of glaze over that pretty quickly. Could you expand a little bit more on what you meant by a person's narrative on their illness?

So there's been a lot of work done in this concept of the illness narrative. The pioneer of this work is a Harvard psychiatrist named, Arthur Kleinman, who wrote a book called The Illness Narrative.

And then for me, there was a very transformative was actually a PhD thesis, done by a physician at Michigan State who, took clinicians concepts and wrote his thesis on what was called the diagnosis narrative.

You know, we have as long as human beings had been on the planet, and as long as there has been the spoken, not necessarily the written word, but the spoken word, we have told stories.

Story is actually the most powerful thing that we have. It's more powerful than any drug. It's more powerful. IT stories create every aspect of our reality.

My wife uses the word manifestation, right? Very important. And we we play out the story in many, many, many different ways through faith, through formal religion, through our own personal belief systems.

What we tell ourselves, what we see. We're in the midst right now of an election time with two very different, you know, candidates. And the two sides can't really talk to each other because the narratives are so different and the people that support one can't, you know, I don't get into politics here, but, you know, we all know what I'm talking about, right?

You don't see what's going on on the other side because they're so close. They've cling so hard to the narrative of the candidate they prefer. So this is the same thing with our lives, with their mass, with the symptoms that a person may present with.

Right. And coming to the doctor. Right. A painful loss of vision in one eye. I have numbness on one side of my body. And then do you have that mass? Right. That's often the referral that I get.

This person may have that mass. There are formal diagnostic criteria. But what's very interesting too, because much of our health and wellness and disease and illness is driven by a part of the brain called the limbic system that really receives information from all our senses sight, smell, touch, sound, everything, and then decides, is it safe?

One of my favorite lines from a movie called Marathon Man, is it safe yet? And is it time to defend yourself, to be in fight or flight? And when you're in fight or flight, no matter what is driving that physical imbalances, emotional, spiritual imbalances, the brain will send out signals.

You may have muscle cramps. You may have tingling, you may become very sensitive to light. You may have pain

all over. And in some ways, as individuals can look like they have Van ness or of sound like that.

And that's from the story they're telling. But when you do an MRI, when you go through the formal diagnostic criteria, they don't write. So alternatively, they might have beams as you go through that evaluation, but you have to get that person to the point in their narratives and understanding what is going on.

That's my role as a doctor or a physician, some might argue a healer. What is a healer? A healer commands a narrative in a very powerful way. You know, the shamanism that is medicine.

And when the person then hears that narrative, and that's the gist of that Michigan State thesis is that then defines who they are, that determine that that's the pivot point where once you believe, if I tell you the narrative that resonates with you, then you're ready for change.

But until you do, I mean, if I use the wrong narrative and you could come to my office of the completely preconceived notion I have Lyme disease, I don't have that unless and until that person says, you know, you, this is Lyme disease, I did the special task that nobody else can do.

And you have Lyme disease. You're not ready for change. So, you know, I always say that even in functional medicine, our tests don't really diagnose things in functional medicine.

Not the way that we use conventional testing. Our tests help us tell a story to our patients about who they are, what's going on with their body, and how ultimately we can take a path toward healing.

So narrative is absolutely critical. And moving forward, narrative is critical because you can say, I love Doctor Walls and I love Doctor Falls. But, you know, I can't tell you how many people will come to my office and they know they're in what we call that sort of contemplation stage where, yes, they they understand, sort of, but in fact, the work is really hard and they're not really ready for change.

Right? They're not really ready to do the diet and exercise and the mind body work and get their sleep dialed in and things like that. Yeah. You know, I think telling the story back to the patients so they can understand what's happening and have it make sense to them.

And as I'm listening to you, I'm sort of thinking, about my time in the VA when, I was very cognizant that as I'm helping people with their story and understanding, we had to have metaphors, a wide variety of metaphors that people could relate to as they would to try to understand how they became ill and over what processes they had control over.

So with is interesting. We developed farming metaphors, engineering metaphors, auto mechanic, metaphors, school metaphors to help people understand, and that it was most remarkable when people could tell the story back to me, tell their story back to me in a way that made sense to them.

And the folks who could do that were ready to really embrace the changes that were going to be very helpful to them. If people couldn't tell me back a story, that made sense to them.

I knew they it just was not going to work out because they weren't going to be able to make, any of the changes to improve their nutrition, improve their self-care routine.

Does that match your experience? Can it does. And sometimes those narratives are very obvious, you know, and I I've worked with veterans, I trained in VA hospitals and I love the veterans, I really do.

But they're a great example. And you'll see a I'll have a 80 year old guy walk in my office. He's still wearing his USS whatever hat right? I mean, he wasn't in the service.

He's not been in the service for 65 years or something like that. But he's still got is that's who he is, right? That's who he is. So veterans very much.

And you know, I am so grateful for the service of veterans. So when I say those, I don't want it to be misunderstood because a lot of bad stuff happens to people in their time of service and deployed, whether it's burnout, it's an Agent Orange and getting injected with peanut butter vaccines.

Those of you know what I'm talking about. But the reality is we also move through this thing called life.

Remyelination, Exercise, and Neurofitness

And so the person that comes in and they're, you know, experiencing symptoms and maybe they're obese and diabetic and they've been a smoker and they've dyslipidemia and all these other things, and lived a high stress life and been pretty sedentary.

But by golly, it was the Agent Orange that did it right. So really, it's it's really tough. It's a challenge sometimes too. You really have to listen.

And that's the part that unfortunately, too often has been laid to the wayside in, in a world of conventional medicine where volume is king and you have to see as many patients as you can and you don't have time to listen to those stories, I do, because we, you know, I'm more of a functional medicine doctor, but it doesn't happen often enough.

And that's why we get stuck on pills that, you know, no one's checking on. And to your point, I mean, I see, I say to look, you know, you're on this cholesterol lowering pill and a blood pressure bill, and I'm not telling people to get off those drugs. By the way.

But if you were I said my my narrative is, you know, if you have a clipboard and you're standing at the entrance to the emergency room and you're you're counting people coming in with chest pain and coronary syndrome or stroke, and you say, hey, by the way, you're diabetic and you're on medicine or you're hypertensive young medicine, cholesterol, you're on medicine like yep yep yep yep yep.

So you're taking that pill and thinking that it's just going to fix everything and prevent the bad stuff from happening, not happen. It's going to happen. Right.

So I see folks who are diabetic and they feel it. They're taking their medicine is permission to have a piece of cheesecake. You know that you can't do that. Right.

And so it's a stepping stone. The drugs can be helpful. But we have to adopt these therapeutic schools. So yeah, you know those meds for the high cholesterol, high blood pressure, they slow down the disease process.

And so they're very helpful. But unfortunately people are not necessarily taking the time to reinforce just how critical what we do is for managing all of those internal medicine type diseases that I take care of, that drive all the terrible neurologic diseases that you take care of.

One more thing I want to touch on before I let you go, Ken, is I know many people have this hope that this going to be a pill that I can take, or maybe an infusion that's going to tell my brain to remind itself.

We might, you know, repair all that myelin. And I know there's that many, many drug trials that are out there. You can you give us, a quick update on.

What do you think? Is there going to be an infusion or a drug that will fix the reimagination problem that is happening? As part of aging, as part of, Alzheimer's and of course, as part of, Ms..

And for everyone who's listening, we all want our brain to get re myelinated correctly. Of course we do. Yes we do. So there actually have been a few investigational compounds.

Biotin high dose biotin has been looked at and compound called and I may not be pronouncing it right. That's protein which is a retinoic acid receptor gamma agonist and monoclonal antibody that targets something called

lingo, where when lingo one is activated, it prevents re myelination.

But unfortunately, all of these clinical trials have failed in their efficacy to promote brain remodel innovation. So even though it is certainly hopeful that we will soon, maybe or eventually have something that promotes re myelination from a drug perspective that does not exist.

However, don't be too disappointed because there are some very good things that you can do that promote re myelination. And probably the biggest one is exercise.

Exercise promotes regeneration nerve cell growth and re myelination. So if we're sitting in our easy chair, as we've kind of alluded to this whole time waiting for the drug to do something to us so we don't have to do it ourselves.

Well, do your exercise program as that makes a big difference. There are some other things that may be supportive, and I don't want people necessarily running out to the health food store and grabbing things off the shelf, but it appears that CDP choline supports re myelination in women, particularly post-menopausal, progesterone.

I I'm a firm believer in bio identical hormone replacement therapy. It appears that progesterone supports re myelination. The flavonoid compound quercetin may support remind the nation.

So there are things out there that and again most importantly exercise. Something's going to be fixed just with taking a supplement. But definitely exercise.

We can do that today even without a drug. You know, as we age, if you become mobile, it really accelerates the aging process. The atrophy, the demyelination that occurs with the aging.

And I know many people with M. S., we we're not exercising enough. We're feeling fatigue. And so it becomes hard. People ask me, like, you know, I'm already exhausted by ten in the morning.

How could I possibly exercise? What do you say to that? I mean, I think that first of all, we have to meet people where they are, and we can start with simple things.

Maybe some chair yoga, maybe the stem that I know you're, you know, very, very fond of. We have to get the body moving. It has to be a graduated exercise program.

And then on the other hand, or the people I ask them that exercise because a big component of what we do here at Charlotte Health Neurology is, neuro fitness or neuro fitness program.

Not everybody we have query giving them their whole neuro fitness plan and working with them. And do you go home and actually do it? Well, I walk, I walk, jog, I walk, run.

Okay. But is that your neuro fitness program? Are you challenging yourself? Is there enough of a stressor? A dose of a stressor? That's a healthy dose that actually stimulates your body to grow and change.

If you do the same thing over and over again, you're only going to get the same outcome, right. You're not going to grow. You're not going to change. So that use it or lose it.

The other cliche that's very true applies. You know, it applies here. Okay. Well, it can this has really been wonderful. I always, always love our conversations.

And again, everyone's listening. And I want you to know, Kent, practice is where I send my complex patients. And now that I'm not seeing patients directly myself, Kent's office is where I see.

I send all of the people who are coming, asking, to come to my practice. So, Kent, the most important thing here is how do people find you? We have a website which is functional medicine.

Dot doctor spelled out doctor. And you can go on that website and sign up for a free telephone consultations. We can learn more about you and make sure that that your situation is a good fit and you'd be very happy.

But we see lots of folks courtesy of Doctor Walz and have done so for many years. We have many patients who are able to, stabilize, even reverse the roundness.

We have cases of one case in particular of complete resolution of the MRI changes, which is astonishing to me. But, you know, it gives people hope that if they can emulate those changes on an individualized level, they can have similar outcomes.

Thank you again. Thank you so much. Thank you.