Ivor Cummins	00:00:00	Well, great to see you again, Malcolm, first of all.
Malcolm	00:00:02	Many thanks!
Ivor Cummins	00:00:04	And Scott, I suppose.
Scott	00:00:06	Welcome! Thanks!
Ivor Cummins	00:00:08	So anyway, we had a quick chat in the last few days about what structure we'd used, and we reckoned it'd be good to follow the structure of Malcolm's blog, because that follow the logical kind of sequence looking at the evidence on various points. And if we have maybe Malcolm just given his thoughts on each point of evidence, or science, and then Scott, you give your opinion. And then at the end, I'll just give my thoughts, maybe refer to a couple of papers that lead me to that direction?
Malcolm	00:00:37	Yeah.
Scott	00:00:38	Yeah.
Ivor Cummins	00:00:39	Okidok. So the first thing is the soft plaque question. So this comes up all the time. And if I had $1 for every time it's brought to my attention or I'm asked about it, I could retire basically. So soft plaque, how important is it that CAC does not directly visualize or quantify soft plaque specifically? It more quantifies the calcified plaque. How important is that in this overall question of the utility of CAC?
Malcolm	00:01:11	Well, I think it always is to an extent it's key, I suppose, because for people who may be catching it for the first time, soft plaque or I think sometimes called vulnerable plaque is the plaque that is potentially most likely to cause problems. So some people call it vulnerable because vulnerable plaques can do this single rupturing and that is a trigger for a blood clot that might block your artery completely. So they're the kind of dangerous ones. I know this is taking about 5000 million bits of information and turning it into a sort of Mickey Mouse sized by bit. So clearly, the part of the problem with the CAC calcium scan is you can't really see these. So the assumption being that if there's a calcified plaques, then that's a sign that there's probably quite a lot of vulnerable plaques in there. And what you're seeing is the end result of vulnerable plaques becoming calcified.

	00:02:09	The problem is, of course, that you could be doing something that's good. And you might be getting more calcification, which would suggest things are going badly wrong. Whereas underlying that, maybe actually the ___ plaques are reducing a number. So I think there is a problem here is are you visualizing, can you visualize the right thing? That's sort of my understanding.
Scott	00:02:36	Yeah, completely agree. I mean, I think we've known for some time that the calcium in the plaque is just the tip of the iceberg. The fact that it's there at all means, the ___ going to be on a plaque ___ in there and an actual plaque, you know, calcification happening in the plaque anyway, whether it's microcalcifications, you know the CT scan or not? So, you could make the point that you know calcium ___ or any plaque there even non calcified, that non calcified plaque is calcifying and ultimately when that happens, that's where the risk comes in. It's just that in order to drain take one of that one tool in the bath water you need to be able to see this or at least to recognize that there is a problem going there. What is the phenotype of this calcium on the arteries? Is it a risky phenotype or is it not and I think that calcium is the tip of an iceberg. It does give you that information in terms of risk. So, it depends on whether you want to know that risk or not or whether we want to act on that risk or actually, we feel that by acting in some way, we are changing the risk. Question is, if someone comes up with a set of score, what stage they'd be at, and I think that's what we need to drill down to. So yes, soft plaque is important especially when it's ___ other plaque or plaque which is calcifying.
Ivor Cummins	00:04:17	So for my thoughts on it Soctt, a paper you brought to my attention, the SCOT-HEART Study published in 2019. It kind of answer the question in the sense single handedly, and to quote from it, "They analyzed all the soft plaque in a whole cohort of people. However, the only independent predictor of risk over the tracking period was the CAC score. A surrogate measure of overall plaque burden. And also adverse plaques did not provide independent prognostic information, where CAC was included."
	00:04:55	So I think that's the point. The CAC is the tip of the iceberg at least from my perspective. William Davis, MD the other day on the discussion on this very thing, he said calcium is an index, a virtual dipstick for total lateral sclerotic plaque and all its elements hard, soft, fibrous, cellular, non cellular, inflammatory, oxidized, non oxidized, and so on. I think for me, the CAC predicts a 20 times risk when high versus low. And it appears

		that although the soft plaque precipitates many events, because the CAC reflects it so faithfully its presence, it kind of doesn't matter too much this point. That'd be my thoughts.
	00:05:41	The more CAC can be good controversy, and you touched on it there, Scott. So we know and it's mainly from statin trials, when they realize that statins associated somewhat with higher calcification over a tracking period, there's a lot of energy put into saying that this was probably a good thing right? Makes sense. And there is some evidence that statin treatment increases calcification. So what would you guys say about this controversy and how important it is in the overall question of whether or not you get a scan?
Malcolm	00:06:19	Personally, your previous points about, is there any point in looking for vulnerable plaque as well as calcified plaque? Because a calcified plaque gives you a score that is basically what you need, although it's not looking at the actual thing that's causing the problem. I don't disagree with that at all. I think the statin one is a bit more, you know, my views on statins, which you probably shouldn't be described here, but I mean, it almost it struck me as a great irony and made me laugh and of course, you know, calcifications, terrible statins increase calcification, suddenly calcification is a good thing. So I wonder why you're saying that.
	00:06:59	You know, that's the kind of stuff that goes on. But of course, probably the statin 1 is when you can argue that what you're seeing is more of a plaque, it's actually stabilizing and it's becoming less of a problem, you can argue that. I don't look at the evidence is tricky. There's no problem with all these things is where's the outcomes? Where is our outcomes? Where do we see it? I think it's probably more interesting when you've got someone who's exercising madly, thinks they're doing a good job, they get a calcium score of whatever, 400, 500 and go, "Oh, my God, what can I do?" Well, at that point, what you're not going to say to them, "Well, what you should stop doing immediately is exercising because that's doing your calcification because no other evidence is well, if you exercise it's good for you overall and reduces your risk of cardiovascular disease." So I think in some people, you have this dichotomy of an apparently bad thing as a good thing. So what do you say to these people?
	00:07:54	I think that's very difficult. I'm not sure what you say to these people. But I think also the other one which is important is a Warfarin issue. Because Warfarin really does significantly increase calcification. Now you don't take a Warfarin to prevent

		atherosclerosis, you're taking Warfarin to stop blood clots forming in your heart that then travel around your body and cause chaos. But the reduction in vitamin K is how Warfarin works, has a direction, I can never remember the exact pathway. But you can see that it will increase calcification. So what are you looking at there? And in this case the calcification a good thing or a bad thing? I've had a lot of people ask me this, "I'm taking Warfarin, I'm going to have a calcium score. Should I stop taking Warfarin or should I take it with vitamin K?" ___ Antagonist, antagonist. I'm going, "I don't know what to advise you on that sense."
Scott	00:08:45	Do you want me to answer that?
Malcolm	00:08:47	That'd be lovely.
Scott	00:08:50	I mean, what I'd say is that if you're taking Warfarin or non ___ it prevent the effects of ___. And then probably taking Warfarin nowadays, you should probably on the ___ whatever you want to call them, the ___ inhibitors ___, because the only real people that __ Warfarin now are people that got either metallic valve replacements or have a clot in the left ventricular apex or something that hasn't been studied in the newer population because they are non inferior to Warfarin and they don't have (that we know of) we don't have these data effects ___ Warfarin in terms of calcification of everything and that you have a better pharmacological response for fail and less interaction. So I would just see, you know, there is an alternate Warfarin if you don't want it and you're worried about vascular calcification and vitamin K as a side effect of the Warfarin. But yeah, that was one thing I would see.
Malcolm	00:10:08	An answer to that, you haven't spoken to my local ___ CCG you look at the goal is new and go Nope. So I do very well saying change it, but we might get the local prescribing advisor, at least in the UK coming in.
Scott	00:10:22	I think we probably just need some education on money they'll save from monitoring everyone's INR, and you know, x, y and z. But yeah, there are business cases to try and get around that. So anyway, that's the set away from this discussion.
Ivor	00:10:38	Yeah. So I think Scott, yeah, I saw that NOAC. And there's a couple of papers now, studies on this where exactly as you say, the NOACs give the beneficial effect but without driving calcification and causing arguably very negative or deleterious effects, but I guess it'll take a while for that to get into practice.

Scott	00:10:54	Well, I think maybe the point Malcolm was making is if you've got a soft, vulnerable plaque, you take Warfarin, it may calcified that ___ . But, you know, we don't know that. It's like the Donald Rumsfeld quote, isn't it, “the known knowns, the unknown unknowns?” I mean, there's so many different facets to this issue in a particular individual as well. You just don't know exactly what's happening in such a complex environment. Like with exercise, I mean, I've had people on the cath lab people who are... the one guy particularly ___ you know, good friend of Bradley Wiggins. He's like a massive cycle stole his life ___, it you know, sort of British team, all that sort of stuff and, you know, severely calcified lesion in the ostium of the led ___ into the left mainstem that needed him because of ischemia and breathlessness and chest pain. Really quite severe schema in his anterior wall.
	00:11:58	So I think that you know, just because you exercise doesn't mean that you're going to avoid heart disease. If you take statins all your life, you might not avoid heart disease. If you eat like a monk and drink like a monk and…. you know, an average punter ___ it doesn't mean you're going to avoid heart disease. And I think that that is the problem is that it boils down to what some ___ you're dealing with and what their values on ___ and how they see the world. And I think that's where Malcolm's getting a lot of people contacting him saying, “Oh, I've got calcium and what does this mean Malcolm?” You know, “I’ve been reading your blogs,” and they go to you, Malcolm. Is that where the blog came from? ___
Malcolm	00:12:52	One of the things, I do have a disclaimer saying I cannot discuss individual medical treatment with you because I can't and I don't. But I do get these questions back. To an extent, the blog I did on calcification was actually triggered by a ___ more than anybody else who asked me advice on a friend of his who had a very high calcium store, and wanted to know what advice I would give. And I thought, I'm not going to give them any advice. So I thought what I would do is try and get my thoughts together and do a blog on it. These are my thoughts. They are not a direct answer to anybody's question. But it's to give people further information and then hopefully… they can say, at least they can understand a bit more about the issue. They may come to a different conclusion, then I come to about the matter. But hopefully, I'm giving people information so they can say, “At least understand what it is now.” “I do understand a bit more about it.” “I would do X anyway or I wouldn't do X anyway.” So I suppose when you ask that question, my answer

		is I'm trying to do my best to give people information in a way that they might at least get the information. I sometimes don't put a conclusion done. In this case I decided I would which is actually not like me, but I did in this game. I can choose a coronary arteries clenching in Ireland, as I did it, no.
Scott	00:14:24	___ coronary arteries or clenching Malcolm ___ something, it was...
Ivor	00:14:29	There may have been other things yeah, contracting, shall we say? OKay, well look, the K2 and the antagonism and the warfarin, they're a little niche, but certainly there are some unknowns there. And I do get asked the question regularly, you know, "What if I take k2 which tends towards you know, lower calcification, possibly a slight regression effect, will it destabilize the plaque?" And for me, I go, but the science and everything we do know, we don't know everything, but if you take K2 and you are low in K2, And you improve your health and fix other problems and your calcification stops or lowers, I think the physiology is smart enough not to regress calcium, leach it back into the system in order to create a susceptibility. But agreed, we don't know.
	00:15:18	We'll get on to exercise in the moment. That's a specific question. But on the statin increasing calcification, just a couple of points from my perspective, people who take statins, because all of this is associational data in fairness, it's not RCT, they may be more careless with what they eat and what they do. There could be a confounder therefore increase calcification. Also there are mechanisms statin operates by, put forward like M2 macrophage phenotype promoting, M1 less so. So, you know, does it really matter if statins do increase the calcification a little more, and possibly in the beneficial vector? Does it really matter to the question of whether you get a scan to find out your actual level of disease? I guess that's more what I'm getting out.
Malcolm Scott	00:16:09	Well I think I'll probably answer that question, is if you're worried about calcification, then the advice would have to be to stop taking statins because then you'll get less calcification, which is not the advice that the mainstream can come up with. Of course it's not quite as straightforward as that. But how are you going to advise someone who's then… so you got a standard cardiologist, you've got calcification, they tell you to take statins because you're high risk and that increases the rate of calcification. How do you then speak to the patient and go,

		"This is good calcification, not bad calcification." How do you answer that question?
Scott	00:16:52	There is data to see that statins do a whole host of effects and I think we've been lately just move away from the statin thing because, you know, a certain extent, it ends an argument of what it does and what it doesn't do. But at the end of the day, if it's increasing calcification within the plaque, I think what you could see is that there is a certain level of calcification where plaque vulnerability becomes at ___ or stops to improve, becomes less vulnerable. There are some studies showing that once you get above a certain level of calcification, that in actual fact that depends on the density of that calcification in the individualized nature of the plaque, it is possible that the risk of that plaque is less. So we're dealing with two different things here. And one just gets a calcium score and you don't know how much noncalcified plaque in risky, you know, 40 plaque, etc, etc.
	00:17:59	Then if the calculations ___ at a level where you can do a CT coronary angiogram which is a move on from___ we will do in the UK if your calcium is above zero and in some cases the __. But we can then actually see the relationship of the calcium to the vessel wall to the non calcified plaque and to the lumen. Often at high levels of calcification, it can be quite difficult. It was certainty sadly what ___ altercation on the ___ ability to the lumen. But I think you're getting onto a separate question there, Malcolm. And if you then have calcification, but you do something about it, like you exercise or you're on a statin, if you're going to do a CT, __ point, then you might be able to see well in fact, this is blocking calcium in the LED. They're not obstructing the vessel. It's sitting on top of the vessel. We can see that there's no reduction in the lumen size here. An actual fact, there's no surrounding non calcified plaque. There's no plaque around them that looks like high risk plaque which has like a napkin ring saying, "I don't really have the __" but that's where there are very low attenuation plaque, the weight next to slightly tougher plaque and in some cases they can say that __ watching in through the ___ that's actually coming into the plaque.
	00:19:40	And so it's a lot more detailed, it can be hard here to try and see it's an individual, but this looks like your arteries are cemented. And there's going to be any reason for you to have as higher risk as someone who's got soft plaque next to calcified plaque, if that makes sense.

Ivor Cummins	00:20:09	Well, you know what, I'm going to have to be a timekeeper here as well, of course, so we need to move on quite swiftly. But just on that point, I think, there's a paper my kid sent me a couple of years ago. I'll put a link in afterwards, put on the screen, but it goes through the density and volume from the CAC standard scan. So many people just get an Agatston score. But there's some good research which shows that if your density is increasing relative to the volume of calcium, it's a much more stable. So I link that paper afterwards.
	00:20:47	So for people who are into the whole serial scanning and looking at this, which is quite specialized density and volume will probably be the key unless they want to go further like you say Scott, and get into CTCA and plaque characteristics. But that's kind of downstream of finding out, "Well, do I have a big disease or small?"
Scott	00:21:04	Yeah. It comes down to the SCOT-HEART heart thing that an actual fact, if you could go fill the plaque characteristics, but in the end your calcium, whether it is 14, and a plaque is very important for risk.
Ivor Cummins	00:21:18	And also I might attach a link to, I interviewed Dr. Arthur Agatston who invented the algorithm for the score and he goes through this whole topic in great detail with loads of patients examples and scans. So people are interested, they can really get into it there.
	00:21:34	The other one we touched on is the exercise question, which Malcolm brought up and believe me, I sympathize, Malcolm. I've been pinged again and again since these papers came out. And I think it's association of all cause and cardiovascular mortality with high levels of physical activity and concurrent CAC scoring. And it was DeFina et al in 2019 but there's some others. Now what I noted ___, a) you're got exercise, more exercise, people or athletic people may carb up a lot more and drink the sugar drinks. There's a confounder, this is all associational. I know there's some evidence that more exercise, you can drive perhaps more medial calcification unless intimal unstable. And there's a whole load of reasons for this. But for me, the key thing was that the high exercisers in these studies with a zero score, they have say a .2 rate per thousand human years. And with a high score, they have 1.8. So they still have 10 times the risk with a high score versus low. And a high exercise or with a high score is that vastly higher risk than a low exercise or with a low score?

	00:22:49	So it's kind of like the density thing. It's a nuance, but it doesn't really change the fundamental question about getting a score to assess largely a real risk.
Malcolm	00:23:02	I mean, yes, clearly, that point is there. But if you are an exerciser, you've reduced your risk overall. So if you have a heavy exercise with a higher CAC scan, and someone's going to say to you, “Well, what are you going to do now? Do less exercise, the same amount of exercise? Stop exercising?”
Scott	00:23:22	No. You do the same because essentially, you're training your arteries to be resilient in a way and that you've developed plaque, just like any other person would have developed plaque in exercise. You'd actually, you know, sort of in a way, they’ve been treating your plaque to be able to withstand the abuse, that you send down your arteries when you exercise, and that your average __ does nothing but sit on the sofa watching homes under the ___ and then decides to go out and shovel snow at Christmas, who then raises his blood pressure and heart rate for the first time and rips off his plaque with that. And so it’s completely and utterly different from the person who's got arteries that are, you know ___ in a way to deal with that.
	00:24:18	And, and so I think ___ you could make the argument that replacing the soft plaque elements with the other plaques is in a way of stabilizing the coronary situation. I wouldn't say stop exercising because you've got a high CAC score. Yes, I would make sure you didn't have a symptomatic ischemia by maybe putting you on a treadmill and seeing what happens. Especially if the calcium was in the left __ or in the ___ the so called prognostic places. And that would be reassuring for you if you went on a treadmill ensured that you can do 10, 12 minutes without any bulging your ECG or your Echo. So I wouldn't tell people with a high CAC to stop exercising.
Ivor Cummins	00:25:05	As a suggestion, so if you're a high exerciser, so you've got one risk factor pretty good, would the suggestion not be a better focus and all the other risk factors? For instance, find out that you’re pre diabetic, in spite of your exercise, there's millions of those out there, or find that you have heavy metal toxicity or are you low on magnesium? So I suppose for me, if you're a high exercise, great job guy, but the fact is, you've developed massive disease. Now the next question is, it’s not lack of exercise. So for me as an engineer, okay, then it's one of the other pareto items. So what are the other things could have driven it? Let's go guys, let's go after those.

Malcolm	00:25:43	Yeah, I think obviously, there's an element doing that whether or not that's jumping ahead, ___ this morning speaking to my brother and my sister. My brother is an engineer and my sister is a mathematical physics at university. So they both love their numbers and figures and were saying, "Well, I would get a ____. I don't know what you're talking about. You're talking rubbish." I think there's an engineering approach to the world which is nice and straightforward and has absolutely nailed everything down to a nice reductionist viewpoint. So I think that the issue there though, if you're going to say to someone, "Exercise is fine, but if your exercises, well, one of you don't discover the other items? What if they're not to be discoverable?" You know, your risk is still lower through doing exercise, and that would be ___ didn't do exercise, even if you see your CAC scans going up. So this leaves people I think with a difficulty because if you're saying to people, essentially, "Don't stop exercising but what you're telling me the exercises, creating CAC, higher CAC score, to me there's a little bit of a conflict there, I'm not quite sure of that.
Scott	00:26:58	Something else is causing plaque to develop and enter the exercises then changing up plaque morphology, just like the statins would do. You know, that's what I would see. So I would, I would say that it's not definitely the exercise, it's causing soft plaque to build up, it might be something else as Ivor said. But, you know, certainly overtraining or doing too much or you know, pushing the boundaries of what you as an individual should be doing and that element in your life can certainly potentially push you on another path you know, increase oxidative stress or something else. I don't know the ins and outs ___ someone in exercise, but…
Ivor Cummins	00:27:48	I might, just as an analogy had occurred to me this morning. So exercises you could call it a risk factor of sorts or risk mitigator. And I think what illustrates things nicely perhaps, there's two papers and I'll put them up after on smoking and coronary calcification. And very, very, very interesting, but in one of them, they analyzed tracked over a long period and they found out that a smoker with a zero score had seven times less risk of an event in the following years than a nonsmoker with a high score. And in another study, they found out that smokers had three times the risk of mortality of non smokers, but in the same data, the smokers who had a zero had the same risk of mortality as non smokers with a zero. So I think similar to exercise, there's all kinds of risk factors and we know from your blog post and much of what I put out, there's many drivers, but

		it doesn't really take from the calcification scans ability, particularly a first scan to tell you what the role of some of all the risk factors in your life have led you to. And that's probably for me the biggest value. You actually find the answer regardless of you did or didn't do in your lifetime, and hopefully gives you impetus to then dig deeper and say, “Okay, what did I miss? Because I missed something right?”
Malcolm	00:29:10	Well, clearly you missed something. You’re doing everything you think. You got the CAC score ___. You’re not doing everything, by definition. Again, anyone can argue with that.
Ivor Cummins	00:29:21	Well, in the second half we get into whether you should get one or not, and then we'll get into the the the actual nuances.
	00:29:29	So the next question is the Masai and you raised that Malcolm in your post. And I've always been really interested in those ___, papers about the Masai. And because if you're opposed, I actually went back to them, had them all on file, and I gleaned a new understanding. So thank you. And when I went back, I realized something. I had always said to people, because anti low carb and plant based people point to the Masai having thick arteries and atherosclerosis, and these are the guys who are eating blood, meat and milk. So they're kind of saying, “Oh look, they got loads of atherosclerosis.” But interestingly, when I dug deeper, the Masai have got quite thick arteries and they've a lot of fibrous plaque, but they have no heart events or nearly none, and they analyze their arteries every three millimeters all the coronaries in around 50 hertz and they found loads of thickening of the intima and fibrous plaque, but they found no calcified plaque. And by inference reading the paper, they found no necrotic cores, you know, no one stable plaque and no calcification and I checked with a couple of pathologist friends, “Could they have missed the calcium and ___ score a couple of hundred in the scan?” And they said, “Absolutely not.” So it looks like the Masai developed a physiologic type plaque, fibrous, maybe like the exercisers. I don't know. But they don't develop the western style vulnerable plaque with necrotic cores and the subsequent calcification. So what do we think about what that means for all of this?
Malcolm	00:31:14	___ me the last time and I kept reading about them and thinking, “Well, they've got lots of atherosclerosis.” And yeah, apparently, they're doing everything I would suggest as a good thing to do. So what the hell is going on? I do think and I think I said in the blog, I think, I think there used to be a concept of arteriosclerosis and atherosclerosis. Arteriosclerosis is kind of

almost a thickening adaptations during exercise, which is possibly a positive thing, because the lumen of the arteries don't know they seem to adapt, so they don't get __ on arteries. So, they're plaque. The problem is, if you have one word lateral sclerosis, it doesn't actually __ to describe everything that's going on. I read the AHA article series on what is atherosclerosis? You know, subsection five, Part Four, paragraph nine, atherosclerosis type VII B. I have absolutely no idea what you're talking about and I'm giving up, I think they gave up as well. It's like looking at a rash on your skin and saying, "That's meningitis." But no all rashes are meningitis. Some of them are just like a rash. And look at all arteries and saying, "That's the same thing," I think it's completely ridiculous.

00:32:27 So I think my interpretation is, is that you can get ___, you can get what they called lesions or things that are not absolutely normal, that are actually they are adaptations, possibly to the exercise to be like going back to what Scott was saying, with exercise, you could be strengthening your arteries up potentially a bit and ___ them for life. I think there's almost two different forms of atherosclerosis, because as you say the Masai didn't seem to have any calcification, but yet other people throw it back and saying, "Oh, they've got lots of atherosclerosis." I say, "I don't think it's atherosclerosis, I think it's a different thing." So I do think that we have to perhaps redefine what we're talking about carefully, which is always a problem. So that's my interpretation.

Scott 00:33:21 I think that's entirely right. I think there's not just one atherosclerosis. I think it's a multitude of potential outcomes within that particular plaque. It may change ___ coronary artery so you can see your one plaque which is calcified and there's nothing else the ____ one that looks more vulnerable. That's that's the issue with the atherosclerosis and vulnerable plaque ___. And one of the reasons why I stop doing the hunt for the vulnerable plaque because when you do an IVUS or an RCT on a coronary artery, you just find that it's everywhere. That's the thing too is, you can't just see all those abominable plaque there because there's one, two centimeters from the ___. You want to __ on an angiogram and that's one of the difficulties we've had in the past in cardiology. Everyone's just gone and had an invasive angiogram that you will see that on a CT and the proximal coronaries, which is the most important thing.

00:34:25 So, I think definitely there definite phenotypes in terms of how an individual in a particular environment or the combination of

		___, all those things with the ___ . So actually if you took the math ___ where I grew up, that the Masai wouldn't last very long because they'd be the ___ every ___ and thinking ____. It depends in individuals and there's definitely not one ___.
Ivor Cummins	00:35:06	I agree with both of you there, agreeing with two Scotsman ____.
	00:35:24	No, I agree totally. And the Masai actually, like I say, new insights there, because I had never realized they had apparently no calcification. But then the Samhain people have no heart attacks in Bolivia, and they have effectively no calcification even in their 70s. Now I'm not sure about there thickening. And ___ similarly. So for me, the calcification is the big flag, not just of some form of arteriosclerosis, but it's a flag of modern inflammatory atherosclerosis quite specifically. And there's something huge in that which I have to think about some more, but these indigenous populations, interestingly, Mann and his team in 1972, who did the autopsy on the Masai, they actually call that the root cause in their mind, and they were surprisingly accurate, exact same as Weston Price. In their mind, it was the dietary component, which was as yet not identified, but they took a shot at sugars, refined grains and vegetable fats, were beginning to be introduced into the country.
	00:36:34	Weston Price likewise, he pulled it out as sugars, refined grains and vegetable shortenings which he saw that city people from the same tribe who are getting their teeth rotting, you know, hypertension as they aged, and all these degenerative diseases, and then they're their cousins, same genetics back in the wherever in the forest, we're not. And he pick this tree, same thing. So that's just an interesting sideline, no proof, but it's funny how we all come back to those three things as real biggies.
Scott	00:37:06	There's someone you should talk, there's a guy called Paul Manovich?? ___ and he worked with ___ Hoffman Estates and he coined the term napkin ring plaque __ is a vulnerable plaque. And he's a fantastic guy. And he's got identical twins studies who were separated at birth and lived in definite environments. And I did publish a case of identical twins, one to another, an acute coronary syndrome, and one who hadn't ___ similar back developing. One of the guys was a runner. That's available online, I can probably give you that top load with a transcript. But what he showed was that interestingly, if you put the CTs of these individuals ___ you've got a very somewhat poorly architected in terms of bifurcation, ___ because the angle of the

		bifurcation can match In terms of the issue of stress that comes up particular areas. But what was noted was that when you grew up in quite difficult environment compared to in terms of socio-economic deprivation, the coronaries was starting to develop an element of ____ . So, your twin if I can call them good and bad twin, good twin had great coronary with bifurcations, but when you actually had metabolic syndrome, hypertension, other risk factors developed from their ___, which is starting to bend because of hypertension. That's one of the the issues with hypertension and also micro vascular structures. If you've got micro vascular disease, there's a backpressure up the coronary and also you've got hypertension coming down, the coronary starts to bend. If you start to create bends in the coronaries, you start to create more ____ stress areas. So that is where the plaque was developing in the bad twin efficiency. But you should probably get ___ to explain what.
Malcolm	00:39:16	I'm unaware of that. That's interesting.
Ivor Cummins	00:39:19	Well, that ties Malcolm, you've talked extensively about this, of course, endothelium, low share nitric oxide. So in fairness, these are all the things we fully agree with, but I hadn't quite realized that example, Scott. So I'll connect that in afterwards and maybe catch up with that guy. So again, I'm conscious there's all kinds of rugby and sport going on. So I keep moving.
	00:39:58	We got through all the ___, and then we get to the really important second part. So, if a person CAC scores shooting up Malcolm, you said in the blog and there's evidence of diabetes, dysfunction, smoking, steroids use, air pollution, etc, etc, and you have a long list, then you need to take action on behalf of any of all of these and more, I would say, does this suggest CAC is actually very useful to know for someone who's willing to tackle and take care of the root causes?
Malcolm	00:40:29	In fact, my concerns about greening and scanning almost beat back to reading Bernard ___ who I'm not sure if you have heard of him.
Ivor Cummins	00:40:39	I know.
Malcolm	00:40:39	Fantastic, man. I think he's still alive, 90 something. It first started when he was looking at angiograms when they came in, and they used to do scans on people, angiograms. And if they found a obstruction in the left anterior descending, they would call it a Widowmaker. And then said, "Do you want to have

something done about it or not?" The answer was, "Uh huh. Yeah, of course." So he was concerned ___ people having these scans who then ended up with embolisms having strokes and things like this. So we realized that there's some quite serious downsides to having the scan, which is not the case with CAC. Then he tried to do a study where he looked at people who got this blockage and tried to find out whether it be better just having medical treatment or whether they went to coronary artery bypass graft. And of course, he couldn't get anyone involved in his study, because the moment anyone saw this Widowmaker, they wanted an operation. He then went back and try to, but he managed to do through ECG tracings, etc. to define whether or not somebody had what wouldn't be a Widowmaker without doing angiogram?

00:41:45 So he managed to get enough people in his study to prove as he saw ___ that actually medical treatment was just as effective in many cases, or most cases can't remember the exact details as having the coronary artery bypass graft. He couldn't get published for four years because obviously people were making vast sums of money out of doing coronary artery bypass grafting. And I think we can see the same thing in a lot of cardiovascular interventions is it takes you back to, "What good did this do?" You know, screening and scanning can often have negative connotations. You do say for instance, prostate cancer screening, you find you've got prostate cancer, low grade, what do you do? Well, actually, it's probably better to do nothing.

00:42:28 So the primary problem I have with treating and scanning is, there's always this one of if your investigation is not going to change your treatment, why are you doing investigation? If you know what the treatment is, you know what to say to people, you know, lower this, don't smoke, take a certain amount of exercise, we think all the things that are beneficial. I suggest that we don't really know all the things that are beneficial, but we're going to advise people. Does it change your advice? Would it change your advice to people significantly? Because there is always a downside to scanning and screening as well. And it can scare the living bejesus out of people. And I've seen a lot of people, I get a lot of emails, "I've had a CAC scan, it's high, oh my God, I'm gonna die of heart disease," virtually. Now, if we could then say, "Well, don't worry, I have an intervention. It's guaranteed to make things better." And we had such an intervention, I'd say, well, that's fine. Now we can do that, because we can sort that out. But at the moment, I think we're not clear enough about the interventions that will improve the

outcome sufficiently to justify having a lot of people being worried that the anxiety levels can be very high.

00:43:39 So for me at the moment, it's… these things are about some people, robust people, engineers, they're going to get a scan where they write, “I will do X, Y, Z. This will be unofficial. I'm fine. I'm going to carry on, etc.” But I see the fallout of a lot of people going, “Oh my God!” And the “Oh my God” people have to be balanced against the benefit people. And to my mind at the moment and that can be convinced either way, I see the downsides is benefits, not enormously like any scanning program, there's always benefits. There's always downsides. And you have to look at these things quite carefully and do them.

00:44:17 So if anyone said, “I want to get CAC scans, I want to know exactly what's going on because I want it sorted out.” There's no way I'm going to say, “Well, just don't be an idiot.” I would say, “But you have to remember that, you know, what are you going to do?” So it's a nuanced area, but I still think it's like I would never tell anyone to get a PSA score to ___ prostate cancer, because it's a rubbish screening test, because the specificity is rubbish. And therefore we were giving a lot of people concerns and worries when they don't need it. So that's kind of the background philosophy to it if you like, and that becomes an issue of, as you say, have we got the data to say these things for sure. These are difficult things to analyze.

Scott 00:45:05 I was gonna say was that yeah, I think if people, let's take the example of someone decides to get a CAC score, and gets the result. I think that, the issue, he, if they're extremely concerned about what they don't understand then we should be going be emailing you. You should be going to ___ and come see a cardiologist because there are things that we can do to really drill down a bit further so that they can regain confidence wired in their particular phenotype. And I think this is the point is that if someone wants to know, and your points were made about different people, maybe aren't prepared for that, and I think there's a really good framework for that called the ___ Foundations and late stage segmentation. It actually tells you what you are in terms of, I think there's five different people now, I know I'm a balanced compensator with a bit of healthy realism. In Liverpool in particular, there’s 50% unconfident fatalists. So if you give them that information, then they can ___ they don't have the health literacy and health is maybe number six or seven on their list of priorities. And then you've got

		people who are like ___. It depends on where you are on your life stage segmentation on how you can then deal with that issue, like you said about engineers just going away and ___. But if you know where you're at, and then you're worried about it, then you should really be in front of a cardiologist who says, “Well, you know, how high is the score?” “Have you had any symptoms?” ___ symptomatic in the next stages to consider actually trying to make you symptomatic because a lot of people say, “Ah, I've got no symptoms at all. So what do you do?” “Oh, you know, occasionally go up the stairs once a night to go to bed, or you're definitely symptomatic.” You know, I guaranteed someone who's asymptomatic I could make them symptomatic. And I would be able to know whether we've got a significant amount of ischemia effect in the heart muscle by simply putting them on a treadmill or doing a vitamin stress echo ____ a asymptomatic if you put the asymptomatic ____.
	00:47:35	__ and ischemia is sort of ___ of plaque bump in the end. __ is I covered by calcium scoring. So if you look at the paradigm that calcium scoring tells you, ___ then gives you a target of ischemia and ischemia is what eventually will give you symptoms or give you risk. Then I think there's a way of walking back through that by stressing someone or going on to do further tests. But even then... I shared yesterday a case report we did from our group, someone who had a CT scan for another reason. But actually when you look at that scan cost event, you see that there's a plaque in the middle of the LED. And he went on and had a stress test, we did show a couple of ___ of lack of blood supply, but they were just placed on standard medical treatment, __ statin at that point. But 15 months later, after the knee operation, they had an event, so that plaque was responsible for it and it was shown on the subsequent angiogram. So I think that there are a few things you just need to walk through here that people should not just be emailing Malcolm Kendrick if they've got a calcium.
	00:48:57	And ___ see an influx of people who've had calcium scans and they'll be like, “I don't know what to do with this.” The point then would refer to a cardiologist and then hopefully we can bring cardiologists up to speed ___. You're going into the cath lab and I'm going to stand the ____ because that's not ___ either.
Ivor Cummins	00:49:17	But this is a very fair point and actually in fairness, Irish Heart Disease Awareness I work on behalf of and David Bobbett, he funded the Widowmaker movie - $2 million dollars. And it says exactly this. It's the story of the stent not changing outcomes.

		And we have the Corer IR trial and the ORBITA trial more recently, and even now Sham trials or those Sham surgeries, where the stent doesn't even seem to greatly impact in many patients symptoms.
Scott	00:49:47	So __ the ischemia tile was just been released. It was the if you have __ on approximately D stenosis under a significant ischemia, they are in the ventricle and you've got X, Y and Z on the risk factors. There are techniques such as measured wire to measure the reduction for... you know, if you have got a really sort of non stenotic plaque there, that revascularization __ does prevent late myocardial infarction from that. So there's a balance there, because it could just stimulate ___ supply and you could get this really great collateralization of the heart, which means that you __- symptomatic and that ___ that's why doing nothing, something helps. But if you've got disease in the artery that's collateralizing it and you don't have very good collaterals because you're diabetic, then you're on to a__, because when that plaque goes, and you can't ___. So I think that's why you do need to investigate people further.
Malcolm	00:50:56	I have somebody who's a regular on my blog, who's got no patent coronary arteries. Had them for 15 years. He lives in Sweden. In fact, my father in law did go, and he had no pain coronary arteries and ____ to the cardio rehab gym and stuff like that he couldn't do a huge amount. My concern, ___ is more of that, as you say, we need to educate our fellow cardiologists what to do. The problem we have, the most people getting a CAC at the moment and getting a high result will be going to people who may not in my opinion, be necessarily giving them a terrifically good advice about what they do. Like, “You need to stop eating fat and eat more carbohydrates,” and “Take a statin ___.” And I'm thinking, you know, “Well, you know what, I'm not entirely in agreement with this philosophy of what you're saying.”
	00:51:59	There's another aspect there which I didn't really raise in the article, but it's partly, I can hardly stand back ____. accused of being enough of a maverick, like saying, you know, “Don't go to a cardiologist. They don’t know what they’re talking about.” Because previously, you would have already had someone stick a stent in you, or whatever. So a lot of dimensions. So it's this thing of, if you're going to do a scan or a test or any sort of screening, then you have to be very clear about what the pathophysiology of the disease is, what exactly is it and what interventions then work? What interventions are effective? Because like the screening, I would say, Well, if it picks up

		disease early, and it's 100% sensitive and 100% specific, and you then act and you know that when you act you are going to definitely provide benefit, that will be seen in outcomes, then who can object to that? Well, nobody. Well I wouldn't object, someone else would. But at the moment, I think there's a little bit of gaps, just a few too many gaps is where I see it myself personally.
Ivor Cummins	00:53:07	Now, Malcolm will agree on that and certainly David and myself have discussed this. And we always say it's a two hander, you've got to get the information out the right to know about the calcium scan. And I might pop up a couple of studies here, which blew me away years ago, __ et all, 2016 __ and 2012 and you see 0,1,2,3, or more risk factors versus a calcium score prediction or risk, and the graph will just blow you away. So I guess we'd agree that the calcium scan has enormous ability to tell you your risk, but then this question of what happens to you. Now, if a guy or gal gets a high score because they found out about it, let's say for non arrogant purposes from me, they'll get a lot about the root causes about William Davis, MD, the cardiologist who has stopped progression in countless patients and almost eliminated secondary heart attacks, or primary ones in this patient population. And we know all the root causes as you went through and I go through. So the person who finds out about the calcium scan and all of the stuff they need to do is without question, I would say, going to dramatically lower the risk from that day or week onwards.
	00:54:25	The other extreme is someone who wanders in willy nilly gets a scan and the doctor's clueless and just gets a med. Now, you could argue that someone with a high score from recent studies, a high scoring CAC statin can show and you've acknowledged yourself, Malcolm, it has a benefit. Well, in someone with a high score, it's actually pretty good benefit. So at least they get that and then someone with a low score, it's no benefit. In fact, it's harm. So calcification then at least would give the person who does not know anything with a clueless doctor. It'd give them something. You know, something. But I think for me, the real focus is because the calcium scan tells you vastly more than the risk factors, it's a no brainer if you want to know and take action to at least get a proper test, because the risk factors, I mean, they're so misleading. It's quite shocking.
Scott	00:55:21	I think there's the other thing. Sorry to interrupt, but we focus ___ article, as well, which focuses on the anxiety created by getting a positive score, __ 50 to 60% of people getting negative score, breathing a sigh of relief, haven't gotten worrying, "Oh,

		my God, you know, I've got no high cholesterol, or I've got a family history of heart attack. In actual fact, now I've just realized that my actual answer of a heart attack is less than point 5% per year." And even up to 13 years of follow up in a London cohort and some of the stuff that put on those years you've done with ___ you know, you're showing the no one died of cardiovascular death up to 13 years. So, you know, I think we're focusing on the people who get the positive scores but in actual fact getting a negative score is a good news for you.
Ivor Cummins	00:56:16	Well, interestingly on that and the stats really coming out the woodwork now is the power of zero is the hashtag and ___ is doing incredible work. But his latest paper out a few days ago is a very high LDL levels. The power of zero works here too. And he makes the absolutely appropriate point that high LDL and the new guidelines now above 190 is automatic medication for life. And that's based on the west of Scotland, WOSCOPS Study and don't get me started on that.
	00:57:00	Well, long story short, the only data they had from ___you could call in anyway, credible to justify 190 or above, automatic medication for life is WOSCOPS. And the thing is that was mostly diabetic man. And they're applying the rule to non diabetic men and women with the new guidelines. So that's a problem. But __ pointed out in his latest paper, and again, I'll link this in the screen, that high LDL people, super high, 40+% of them have calcium zero as well. So you've got all these people scared about LDL and cholesterol and myriad other things around the world, and the calcium scan can actually tell them the truth in huge number of circumstances. Now, we've got to…
Malcolm	00:57:46	___ I agree, though, you may be unaware there's a study done in Nottingham, I think, where they actually looked at 48 factors with regard to what the actual risk of heart disease was. And LDL came 46 out of 48. _____.
Scott	00:58:06	I've got ___ for that, Malcolm. And we have drugs for triglyceride to HDL and, you know, morning triglyceride and lorring LPA. ___ I'm being facetious, but, it is driven by the fact that there's a drug for that, not that there's a lay still or a…
Malcolm	00:58:26	Absolutely. If I may make a general point is that the risk calculators are rubbish. More than rubbish, completely rubbish. Our understanding of the risk factors, the mainstream understanding of the risk factors is poor. So if one was to say every screening test becomes valuable, if the interventions that

		are based on that are effective and good at altering the results, so one of my main things yes, if you did discount Ivor and ___ Scott, you discuss these things with patients, I think the outcome would be positive. But the problem I have at the moment is that we're relying on outdated ideas about what is actually going to create benefit, and what people are doing?
	00:59:11	This is the world in which we are in, right? If it was a perfect world, you know, we analyze people, we tell them what we're looking for, we see a scan, the negative ones are reassured and everything's fine. The positive ones we can do something about. Again, I couldn't object to this. I don't think we're in that world at the moment. So that's why I'm am advising against it for people. But in the right hands, I think it's, as I say, we're not talking absolutes. These are __ of benefit versus ___at the moment. We need more people to do this stuff properly, and then we can create outcomes upon which we can say, look at these results.
Ivor Cummins	00:59:54	Well yeah, it is a challenge or as I said, a two hander. Both things have to be fixed and they both have to move forward in parallel, in my mind. I mean, that's the way we always do it. You don't wait to replace crappy risk algorithms and risk factors with a proper scan or proper diagnosis until everything's all lined up. But I agree, of course, the whole medical business, there's a lot of challenges and a lot to do. but we at least can get the risk measurement correct and ___ hopefully the other will improve.
	01:00:28	___ et al had a study there, which I often quote, and basically on a group of people all on statins, so they were all medicated, blah, blah, blah. The people who progressed at a high rate and calcification had 17 times the event rate in Kaplan-Meier as the people who had the same very high score, but they didn't progress. There were slow progressors. And just to give an indication of what might be possible, let's just take magnesium, which I know Malcolm, you think would be a significant thing, magnesium deficiency. So a very recent study, and it was an RCT in humans with chronic kidney disease. And basically they had diabetes type 2 and hypertension, the usual risk factors, so the very people would be concerned about. And for two years, they gave one group, magnesium oxide, fairly high dose, and the other group ___, nothing. And the former group of magnesium oxide, they got 11% progression on average, and only 22% were above 15% progression per annum. Now, that as per ___ and other progression studies, that means relatively super safety. And guess what, the people in the control group without the magnesium, they're an average of nearly 40% progression, and

		65% I think of them were over 15% per year, which is kind of the magic number. So that just kind of looks magnesium sufficiency, but we know there's a list of probably six to 10 major drivers of heart disease. You know, imagine what you can do when you find out you've got an issue. It's huge and the people who can be saved are the people I think we're really thinking about, not so much the people who are going to worry. You could argue that the people are just going to worry, __ for doing what needs to be done, you could argue why did they get one in the first place perhaps?
Malcolm	01:02:33	Well, as I say your intervention are going to be beneficial. It's the problem at the moment is primarily. A lot of the interventions are going to be triggered by this on the mainstream, in my opinion, are not going to be beneficial. We're going to do things that are not necessarily going to alter the risk factor. Because, as I said, it's the risk calculators and are perceived key risk factors are wrong, then we're really not going to achieve a great deal of benefits. Just speaking to someone in America, his LDL level has been 18.5 for the last 30 years. He has no detectable aterosclerotic plaque or calcium. The most people will then be immediately placed on medications, lots of medications, many medications for the rest of their lives. This is reality, what's going to happen ___.
	01:03:33	Is someone in the right position who understands what's going on, then I think we can see significant benefits. My concern at the moment is, our interventions may do more harm than good in many cases, or we're not looking at the correct interventions. So therefore, that's really where I'm coming from. And to an extent, I wasn't hoping there'll be a debate and hopefully this will trigger a wider debate of some sort and people will be interested in the area. Which is that we need to be looking more closely about the things that we should be doing with evidence that we benefit. Just saying your study on magnesium. That's, fantastic. What about potassium? Potassium is another one. What about etc, etc. __ to see more of this data that we can then say, right, we now have an intervention that we can say is liable to be extremely beneficial and we have data to demonstrate that. And once those things happen, obviously, the shift shifts towards, you know, “Let's do this thing,” of that make sense?
Ivor Cummins	01:04:38	Right. Well, actually, yeah, it is a huge point. And I read that in your blog that, you know, not so much that we don't know, but orthodoxy perhaps does not know all the really important stuff that should be focused on. Therefore you lose a lot of the

		benefit for someone who walks in clueless and meets with clueless people in the business. ___ though is kind of given the proof point, I mean, his study on 45 people over 34 months, he went in with fish oil, vitamin D, low carb, magnesium and he achieved 42 out of the 45 to have regression, or no movement or slow progression in the following 34 months, which is groundbreaking. Now, of course, Malcolm I agree. The problem is no one's reading his paper, but I mean, the proof points are there.
	01:05:30	I had one other thought and Scott or Malcolm, you might tell me what you think. We mentioned magnesium, and there is a trial on that. We're lacking trials and all of the other stuff we know is important, we pretty much know. But if you take for instance, diabetes type 2 dysfunction, that's a huge driver and the EUROASPIRE Study recently ___victims ages 18 to 80, 26 countries across Europe, and 76% were essentially type 2 diabetic, most of whom were undiagnosed before they looked. And the others ___ measure the insulin. So we know that most heart disease victims are essentially diabetic along with other problems. But we also know from Virta and many others, that we can intervene when we know there's a problem and largely resolve the type 2 diabetes dysfunction. So that alone, if the calcium scan identified you have major disease and you check and find out about postprandial glucose and glucometers, and like David Bobbett did, he went digging, and he found out some of this and he found out he was a screaming type 2 diabetic undiagnosed. So that alone if we start identifying all the people with undiagnosed diabetes, and they find out which we are finding out, that it's resolvable, not with orthodox methods, or drugs, but with other interventions. That's huge. That's huge amounts of lives that could be saved you know? What do you think of that one?
Malcolm	01:07:03	Yes, again, the problem is that you're you and you're one person. And so this discussion perhaps or a small group of people, this whole diabetes, high sugar level issue is not recognized. It's just not recognized __ and therefore, you're not going to find your average doctor or your average cardiologist dealing with it in that way. It’s not going to happen at the moment.
Scott	01:07:32	I think if you do deal with it, it's going to be by prescribing an ___ ate more sugar. I think the problem goes back to __ individuals Ivor, because you know, not everyone can take vitamin K2 and magnesium and vitamin D and and then you know, sort the lifestyle. You find your average ___ still

		wandering the ___ on Friday night. ___ dreams and, and if everything is absolutely perfect in an ideal world, we'd just be pondering tomorrow and you know, tramp with ___ , "Guys, you better watch out for your postprandial glycemia." Or people would start wearing CGM or Kim Kardashian CGM on Instagram and instantly everyone will be worried about what the postprandial glucose is and how to stabilize the metabolic profile. But that's not going to happen because people are people. And it filters back to this __ segmentation thing, because I think you will always have your healthy users, you know, your vegans, vegetarians and people who are concerned with exercise, blah, blah, blah, healthy user bias, and therefore, perceived healthy user bias. And then you always have young working class man who just wants to go to the pub ___. So you know you're not going to change in those people. It is very, very difficult.
Ivor Cummins	01:09:09	So, if you are harsh about it Scott and Malcolm indeed, you could say, well, there's a huge amount of people out there, millions, if they found out that they had major disease and they've been lied to about vegetable oils and complex carbs and all that, and were willing to go and dig, that could save themselves. But equally, there's a huge amount of people who will not do anything. Who don't care, and you could say, "Well, okay, we're not as worried about them." It's a fair point, we're only worried about the people who will actually take the responsibility for their health and act on finding out they have a major issue and that they were essentially lied to about how to prevent such an issue, largely. So that cohort is where I'm focused.
Malcolm	01:10:00	You're being a bit negative. We'll call them early adapters.
	01:10:12	That's a marketing thing. Early adapters. There's some people who will buy the latest high fi equipment, which costs 5000 pounds, and then in about six months, it'll cost about 20 quid. There's always people who will be at the front of the curve, majority sit somewhere in the middle and there's some people will never change no matter what. And so you need the early adaption? Things will change slowly.
	01:10:40	___ Ivor, and I hate to jump into the thing at the moment, but I'm going to have to say I'm going to have to go in about two minutes. So, it's been a fantastic discussion. I think I've learned a bit myself here. So my learning would be, you know, we can always listen to a gentleman and exchange views and I think I'm delighted to be a part of this. I think civilized discussion about

		this and shared views and hopefully… I think we're all mostly on the same page about most things still in almost everywhere where we're agreeing badly. My concern is, is in the world as we have it now, I'm a bit worried about people going off in CAC scanning and then not being dealt with correctly. But that world changes, the dynamic changes and the balance of benefit ___ changes as well. But, you know, I'm not not anti scanning per se, as you know, as we've discussed. So I think that's my final word on the matter.
Scott	01:11:47	And I have to say that, it should never happen that someone gets a CAC score and then they go straight into the cath lab. And this isn't ___. That should not be a reason for an interventional cardiologist to make money from your arteries. And you need to know, and in the UK what would happen is they would either have a CTCA to see whether we can actually see whether there's any ___ combination of hard and soft plaque. We can even potentially see FFR to look whether it's ischemia there. But even that is a bit difficult around the boundaries you need it to be, but it really definitely ischemic to non ischemic. And then you should be having some form of stress test to reassure. You should be seen a cardiologist. You should be encouraged to adapt new lifestyle changes, but you should certainly not be getting in a cath lab because I've heard bad stories about that. So that's what I would probably think ___ with Malcolm's opinion on this, ___ should be used as an individualized way to recognize your coronary phenotype, rather than a blanket screening thing ___ who needs it.
Ivor Cummins	01:13:07	And I guess what I'd say though, we might do another little segment to fully close this out. But my thoughts on it would be that we need to give the power back to the patient to take personal responsibility. I'd be focused most on the people who are willing to do that, and can do that. The ones who are not, they'll at least get some medications or they may be go through the process, Scott, as you said, to get proper follow up, and proper treatment appropriate as opposed to the bad stuff. And really the right to know for me is that you have a right to know that there is a scan that can actually tell you properly compared to all those junk risk factors. You have a right to know about the scan and to make your own decision. And if you walk willy nilly in and don't know what you're doing, okay, that can create worry, but what I'm seeing are growing army of people who are finding high scores and ___ and okay with it, and they're taking action. And we know there's so much action that can be taken. So I think that's where I would focus more on, that everyone

		knows about it and has a choice. And right now no one knows about it.
	01:14:16	And one last thing, actually, Malcolm, to your point, the early adapters, and I know those that the marketing curve with the chasm, the early adapters who go out there and find out all the own truths we've been told, and who take action and research, they can become very vocal, and they can become very influential also. So I think it behooves us to get the message out on the scan and the root causes and create a growing army of people who are now aware, and they will ultimately change all of the bad things about the system we're talking about. But unless we get it out there, we'll be left with all the bad things, I guess, Malcolm that you referred to.
Scott	01:14:59	This is the point where you put in the, is it a Few Good Men, Jack nicholson and Tom Cruise when he says, "You can't handle the stress." Is that what you're going to…
Ivor Cummins	01:15:08	That's exactly it. If people can…
Malcolm	01:15:12	It's a bit of a thing. I understand saying there's only two things are infinite: the universe and human stupidity tonight and I'm not so sure about the universe.
Scott	01:15:19	Well, the universe is made up of protons, neutrons and morons, is that right?
Malcolm	01:15:24	That's right.
Ivor Cummins	01:15:26	Okay, well, at least none of us three by any means are in the latter category, that's for sure. We're very well aware. Hey, thanks a lot, guys.
Scott	01:15:36	Thanks very much.
Malcolm	01:15:37	Thank you.