Intense Exercise & Potential Heart Damage (aka Athlete's Heart)

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Dr. Andy Galpin
The science and practice of enhancing human performance for sport, play, and life. Welcome to Perform. I'm Dr. Andy Galpin. I'm a professor and scientist and the executive director of the Human Performance Center at Parker University. Today, we're going to start with a story. In fact, it's a ghost story with a character by the name of Caballo Blanco. For those Spanish-speaking friends out there, that, of course, means the "white horse."

And Caballo, as he was referred to, terrorized and mystified the Copper Canyon range of the Sierra Madre in Northern Mexico for the better part of 20 years. Many years later, of course, the story would be unraveled as an individual by the name of Micah True, which was also in fact not his real name. But as the lore goes, Micah or Caballo lived most of his life in this desolate Canyon region, and he would live running between 30 and 60 miles from village to village, gathering up resources, and then running back to his hut.

And no one really knew what the heck he was doing. Micah himself was running somewhere between 160 to 170 miles per week for nearly two decades. And again, keep in mind, he was doing this in an area where he had no access to running shoes, medical care, or really any advanced technology, nutrition, or hydration strategies, so simply surviving by running large amounts every day for decades on end.

Micah's story was made famous by the best selling book Born to Run. You can read more about it there. But really, he became one of the most central and legendary figures in the ultramarathon circles. This launched, of course, an entire generation of ultra runners. But where the story is of use today is the fact that Micah himself, while running those, again, 150 to 170 miles per week for decades, eventually end up dying at age 58 during a very routine 12-mile jog.

And if you're from the running community or world at all, you probably know this exact story. If you don't know of Micah himself, you know the bigger plot here. And that is this juxtaposition or paradox between really incredibly, highly cardiovascularly fit individuals, oftentimes runners, who then end up paradoxically dying in mid to early age of cardiovascular disease. And so if you looked at the title of this show and were kind of wondering what it meant, this is really exactly what I'm talking about.

It's this phenomenon of trying to understand, well, how are these people who are elite in this

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physiological area also at extremely high risk? The story itself is really as old as we've understood cardiovascular physiology. And so when we actually get into what's happening here, we don't know Caballo's story itself. It took them several days to actually find him. He was lost in the middle of a canyon somewhere, and by the time they got to him, it was too difficult to perform a proper autopsy.

But the prevailing wisdom here is he likely died of something called left ventricular cardiomyopathy. I'll get into what all that means here very momentarily. Where this has gotten more difficult is then cardiologists and other medical professionals have tried to inspire and help the world in the sense of preventative care here. We've also led to confusion and concern about really what is this relationship between exercise, extreme exercise, and cardiovascular health.

And so I think the best way to think about this is to split it into two broad categories. The first is the classic case of Caballo, what I just described. These are the folks who are typically between 30 to 50 or so years old. It's generally men, though it can occasionally happen in women, who have this paradoxical extreme cardiovascular fitness, and then they suddenly die in the middle of a race or an activity from cardiovascular disease. You also then have what people are probably potentially more familiar with, and that is the young athlete. So this is your, again, typically 12 to 20-year-old who has no apparent signs and symptoms and then unfortunately passes away during physical activity.

I'm bringing this up today for the unfortunate news, the fact that I have personal experience with this. A friend of mine, Mike Osuna, recently lost his 15-year-old son Lucas to a similar event. And so I think it's really important to me to cover this topic because some of this is preventative. We can do something about this. At the same time, we're causing unnecessary confusion in it.

And so what I really wanted to cover today with this athlete heart idea is, what do we really know about this situation? Why does it happen? What's causing it? How do we prevent it? What do we look for? And so we can separate the fact from the fiction as much as we know the information. I'll skip to the end here. We don't know everything right now. We certainly know plenty of things that I think are very actionable and helpful, but we have way more to learn.

And so I believe it's personally important for all of us to understand, what do we look for again? What is preventable? How do we do that? And then we can hedge people against the two major pitfalls, one being unnecessary risk. So when are we putting ourselves in a situation where we probably should reconsider that, or at least understand the true risk that we're in and not be overconfident. The other side of that equation is the opposite, where we're inducing unnecessary fear of exercise or running on people when it is completely unnecessary.

I think once people have that information, they can make whatever choice they would like. That's

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not up to me to decide. That is entirely up to you. But I believe it would be helpful for us to truly understand what do we know scientifically, what do we know clinically right now about this really interesting paradox of the athlete's heart.

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In 1899, we see this term "athlete heart" officially coined by a Swedish physician. What he started noticing was cross-country skiers had these enlarged or larger than normal hearts. Did not know if this was something they were born with, that's what allowed them to be good skiers or endurance athletes, or something that they accrued as a result of their exercise. Well, about a decade or so later, since people were noticing this was happening mostly in runners, not because of anything special to running, but just because these were the events people were doing, and so "athlete's heart" got turned into "runner's heart."

You'll still hear this today. If you ever hear those phrases, "athlete heart" or "runner's heart," you can think of them as basically interchangeable. I will, in short moment, tell you what the true scientific term for this phenomenon is, but in pop culture, runner's heart, athlete's heart, it's basically the same thing here.

And where we really first get our entry point to the scientific literature is in 1910 with a guy named Clarence Damar, who eventually won the Boston Marathon like seven or eight times, was famously standing at the start line of, I think, one of the Olympic games. And a crowd of physicians were standing next to him begging him to not run the race. I think at that time, and I

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don't know the exact details of the story, they knew he had some sort of irregular heartbeat or heart murmur, and they were all concerned that he was going to die, and they were begging him to not do this race.

And he runs the race and nothing happens. In fact, he lived until he was about 70, eventually dying of cancer, I think. And so now everyone's really confused. They're like, oh, my gosh. We thought we had our ringer here and this guy runs for decades and decades, wins tons of medals, and has no issue whatsoever. But they were able to autopsy him after he eventually died and they found that he had exceptionally large coronary arteries, and the heart itself was large. And so now they had their first description of a longitudinal study.

They had a real athlete whom they knew did real cardiovascular exercise, and then they got his heart in a good spot after he died, and they started to confirm, yes, indeed, he had these enlarged areas, but it wasn't detrimental. Before we go any further, though, I need to give you a little bit of a detour and help you understand some basic physiology and anatomy of the heart, because that's going to help you understand what is happening in response to exercise, what's happening in response to other situations that are maladaptive, like chronic high blood pressure. But we got to know a little bit of the physiology to understand what's going on here, and we also need to know this when we get to the end of the discussion today so you know what to look out for and how to ask for tests and interpret them when you get that information.

OK, so how does a heart really work? It's got four specific chambers-- two on top and two below. The two on top are called the atria. The two on the bottom are called the ventricles. And so the way blood flows through your heart is it starts in your right atria. It drops down to the right ventricle, which is directly below it. The right ventricle then squeezes and contracts and pumps blood from the ventricle to the lungs. It heads into the lungs to pick up oxygen, deliver its waste products, and et cetera, and then it comes back from the lungs and goes into the left atria. Just like the right side, it goes from the left atria, drops down to the left ventricle, and now when the left ventricle squeezes, instead of sending the blood to the lungs, it sends it to the rest of the body.

The left ventricle is probably 80% or 90% of the discussion of athlete's heart, because that's the place that has to overcome pressure that comes either with things like chronic high blood pressure or exercise. And what you're going to see unravel here is it's actually incredibly difficult, if not impossible, for us to determine positive versus-- again, I'll keep using the term "maladaptive" or "bad adaptation" in the heart, looking simply at what's called morphology. So morphology being what does the structure look like? What's the anatomy?

Because the adaptation itself, we don't know how it got there. And so there's something special and unique to not just the eventual result, but what caused the adaptation to occur in the first

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place. So when it leaves the left ventricle, it's going to go out via the aorta. And this is our first major artery. I love this, and I remember learning it when I was an undergraduate. But when things go away from the heart, they are arteries. When they come back to the heart, they are veins.

Now, one thing people mess up a lot is they assume all arteries mean they are oxygenated, and all veins mean that they are not oxygenated or deoxygenated. That's not necessarily always true, and the special cases when it's not true don't matter right now. But I think it's better to understand these things as away from the heart, "Away" with an "A," is an artery, with an "A." Coming back to the heart is a vein.

In the middle are the capillaries. And so I know I'm going to go through this in painful detail, but its very important to understand because we're going to look at the different aspects of adaptation here momentarily. When it leaves the left ventricle, it goes out that aorta. This is a giant artery that goes to the rest of your body. It then distributes all over your body depending on where the blood needs to go. And when it gets to the tissue that it wants to get into, whether we're talking about skeletal muscle or your organs or what have you, it's going to be switched then from arteries into capillaries.

And there's a bunch of other small things here I'm glossing. Over what we call the place of fuel and nutrient exchange is in those capillaries. And so you can just imagine, in this case, oxygen coming into a muscle bed. It goes into a capillary. The oxygen then goes into the muscle. Waste products then leave the muscle and go back into the capillary, go out the muscle bed, and then come back up to the heart via the veins.

So the aorta is our major one, and in the case of heart disease, chronic or otherwise, this is a major place where we have issues. But some of the other ones that are important to pay attention to are things like your carotid arteries. These, of course, are the arterioles or arteries that send blood flow to your head, brain, and neck. Other ones you've probably heard of are the coronary arteries. Now, I can say you probably heard of them because that's what I mentioned a few moments ago.

Now, these are special and unique because the coronary arteries and veins are the ones that actually deliver oxygen to the heart itself. People gloss over this one. I know I did forever. In fact, I remember being an undergraduate learning this and be like, oh, my god, I never thought about this in my life. We always describe the heart as being necessary to pump blood throughout the rest of the body, but we also have to have blood flow to the heart itself. Remember, it is a muscle very similar to skeletal muscle. I covered this in detail in an episode in season one. We'll put a link to that episode in our show notes so you can go check that out.

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But the heart itself needs to contract. It has myosin and actin and ATP requirements, and it has to have carbohydrates and all those other things. So you have to have blood supply to the heart. So we need to have those coronary arteries and veins that pump into the heart itself. From the venous side, the equivalent to the aorta are two separate ones that are called the inferior and superior vena cava. So those are the main ones that bring blood supply back to it.

I glossed over a lot of gross anatomy there, but that gets you a rough idea of what's happening. This matters because the left ventricle, unlike the right ventricle, has to supply the entire body. In other words, when the right atrium or think about the top right-hand corner, the top four of your quadrant, the right atrium just has to drop blood into the right ventricle, which is right below it. It's also got gravity on its side.

And so what actually separates all these chambers are a bunch of valves that we won't discuss today. But basically, just open up the valve, and if you had to, let the blood drop from the atria to the ventricle. But the right ventricle has to then pump it actively to get it all the way into the lungs. But the lungs are pretty close to the heart. In fact, sometimes they're above it or below it. And so I just kind of have to move things a few inches, really, horizontally.

But the left ventricle has to, in one single pump, get it out the aorta all the way through the entire circulatory system, and then back up to the heart against gravity. And so the contractile strength needed in that left ventricular pump is unlike any of the other three chambers. This is also why, when you look at your heart and when we draw cartoons, we see this nice, symmetrical, vertically aligned heart. But in reality, it's often-- well, it's almost always tilted to the right because the left ventricle is much larger than any other of the four chambers because of exercise-induced adaptations.

What? Well, that's what we would call it in my world. In this particular case, you have to have more force, more output because of what I just mentioned. And so that muscle grows larger. This is why you're going to see this term, "hypertrophy," all over this literature. Hypertrophy is muscle growth. And you keep thinking, like, why are we mentioning this when we're talking about heart disease? Because it is a direct result of both exercise adaptation as well as heart disease.

That muscle will grow when it is chronically stressed or acutely stressed. You're starting to see the relationship here. It's really confusing to figure out. I can look at your quadriceps muscle groups, pick any one of them, and if that muscle is larger afterwards than it was before, there's almost no chance that a disease is happening. There are some extremely rare ones, but almost always, I can simply look at a before and after picture and say that person is healthier.

When we do that at the heart, that is not true. So hypertrophy has a different connotation in the heart than it has anywhere else, hence this term, "enlarged heart." Now we've got a basic

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understanding of gross anatomy and morphology. We've got to go back a little bit in our stories to a gentleman named Paul Dudley White. He was at Harvard, one of the pioneers of preventative cardiology. In other words, a guy really ahead of his time, 100 years ago, advocating for exercise as a way to prevent cardiac disease. Phenomenal work, awesome.

What he started to unravel was the fact that athletes or exercisers were experiencing something called bradycardia. So remember, tachycardia is an elevated heart rate. Bradycardia is a lower heart rate. Remember, put yourself in their shoes 100 years ago. We did not know that a lower resting heart rate was normal or good or bad, because the vast majority of time, if you're a physician and you see somebody that has a higher or lower than normal resting heart rate, something bad is happening.

And so the community did not know if this was the case. But because of Paul Dudley White's work, we started to realize that this lower heart rate, or bradycardia cardia was not pathological-- again, "pathological" meaning it's bad. There's a disease occurring or something similar. And so the narrative started to shift.

Now, in combination with a couple of his research partners, Lewis Wolff and John Parkinson, they were the ones that really promoted and, of course, got the naming rights to something called Wolff-Parkinson-White syndrome, WPW. We're going to come back and talk about this more later, but almost all of you I would be willing to guarantee are familiar with Wolff-Parkinson-White even if you don't recognize the name. More on that later.

Fast forward about three more decades. In the 1960s and '70s, our story is darn near complete because we have, again, another boom in running. First one, the Olympics are reintroduced. The second one came in the 1960s, and '70s. Marathoning and running in general exploded. I've covered this in other episodes. I actually-- this, to me, is one of the most fascinating if not most critical time periods in all of exercise science, and I would argue exercise physiology and modern health, 1960s and '70s, but nonetheless.

We started just getting research on this. There was interesting data sets coming from big groups of athletes out of Italy. And we started to see what I've already described-- left ventricular hypertrophy and enlarged chamber size in all these elite athletes, but no diseases. They weren't dying. We're also getting a host of what's called sudden cardiac death in young athletes, where we're seeing hypertrophic cardiomyopathy. "Hypertrophic," again, being "large;" "cardio" meaning "heart;" "myo" meaning "muscle;" "pathy" meaning disease. So "enlarged heart resulting in disease."

And so now we are smack in the middle of controversy. At the same time, we're getting data on athletes who are extremely healthy with larger hearts and then seeing young athletes with larger

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hearts dying. What the hell is going on? Hypertrophic cardiomyopathy, or I'm going to use the acronym HCM for that, is the clinical disease. Effectively what we're doing is we're labeling the exact same morphology differently. If it resulted in early disease, we call it HCM. If it didn't, we call it "exercise-induced adaptation." But nonetheless.

And so the dual thread here of normal adaptation versus what we would call on the field mimicry of disease begins. And this is, again, that classic case of sports or athletics or exercise mimicking disease without the pathology. Where this gave it the last final modern twist was in 2012. There was a conference in San Francisco called the American College of Sports Medicine. I was at this event. I remember this, and we're talking barely just over a decade ago.

And what happens at conferences is it's supposed to be an opportunity for scientists to share insights that are beyond their research papers. There's no point of going to these things and just giving a talk on the paper that you wrote, because everyone can read that. And so oftentimes, we generally give people a little bit of liberty here. Oh, share us some unpublished data. Let us know what you're working on, some insights you think are happening. And then, you know, next year or the year after that, when the paper comes out, we'll read it and we assume you're going to make some mistakes.

Well, this happened in 2012. A group-- I think they're out of South Carolina-- of epidemiologists had an unbelievably cool presentation. They had data from a place in Dallas called Cooper's Clinic. And so they had data sets from 1970 all the way to about 2000, about 30 years. And that included 50,000 people who had done cardiovascular tests with them. Now, of those 50,000, they had about 14,000 runners.

Now, I don't know if these were competitive athletes or just the occasional jogger, but these are basically people who self-identified as I run for exercise. So that's amazing. I mean, just think about this-- 14,000 people with cardiovascular tests who run. This is incredible. And the average follow up for these people was about 15 years. So not only do you have their baseline test, but you can see them before and after 15 years of this exercise, which, again, gives us incredible insights into what happened in response to the exercise because we have a pre and post on each individual person.

Well, not surprisingly, the first data set here was the fact that those runners had about a 20% increase in life expectancy than the non-runners. No surprise here, right, folks? Exercise is good. It will keep you alive much longer.

But where things got twisted-- and I actually want to be clear here. I'm not blaming the authors here because I don't remember the presentation being like this. My framing at the time was different than what actually happened later. But the reality of it is they also shared insights that

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said, hey, there seems to be this protective effect of runners up to about 20 miles per week, such that people that are running up to 20 miles in a given week are having an increase in lifespan. But if you run more than 20 miles per week, we potentially have problems. In fact, what you're seeing is those people had no increased risk of survival or no increased likelihood of survival, rather, than the people who didn't exercise at all.

So you can imagine yourself in an inverted U such that you start running, and you're increasing your likelihood of survival. You're doing better. But once you get to that 20 miles per week, more target. If you continue to run more miles past that on average, your benefit goes down, which is almost indirectly or directly, saying more than 20 miles per week is actually disadvantageous. It's dangerous, as another way to interpret this.

And you saw an explosion of popular media. I won't name the articles or the magazines themselves, but this took off everywhere. And if you've been in this field long enough, like I have, or many of the people that came before me, of course, you've seen this wave come and go. It seems to be every three or four years. We get an explosion of the, is exercise actually bad for you? Is too much exercise bad? Is it actually dangerous? And we're in that moment right now, just in the last-- part of the reason I'm doing this episode now is I saw another set of articles come out in the last month and I'm like, oh, my gosh.

And it's the same story every time. It's rarely ever updated, and I think you can actually cover the entire thing in one set, and in the next few years when this comes up again, barring any new information, you're probably going to see the same story repeated over and over. So it's frustrating because what happened in this was you actually had a few cardiologists who are extremely famous publishing in journals like the Mayo Clinic Proceedings, which are highly reputable in the medical community, of course, saying that exercise is inflammatory to the heart, causes things called fibrosis and scar tissue, and is not only deleterious, but dangerous.

But here's the reality. Those authors, when they finally published that paper a couple of years later in the Journal of American College of Cardiology, did not-- and I will repeat-- did not conclude that running was dangerous or disadvantageous. What they actually concluded was most, if not all running exercise is excellent for your health. Past 20 miles per week doesn't seem to be more advantageous, though. But it didn't hurt anybody, either. And I'll give you more data on this point directly.

But this is very, very, very, very different than how those data were portrayed two years ago and especially by other folks in attendance at that meeting, and then people that wrote about it a little bit later. I'll reiterate something I said at the beginning. I want to share the data as best as I can interpret. I think that is a fair way to justify it. And then you can decide if that risk is worth it or not. That's not up to me, but I think it is up to scientists to make sure that the data are being

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portrayed accurately and appropriately, and that the conclusions match the actual results. And in this particular case, I'm going to make a strong argument here in one moment that that was not the appropriate interpretation of those data and we got to be careful with jumping to conclusions.

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If these adaptations seem to be somewhat similar, where are they at specifically and what do the data tell us? Well, let's start by understanding if we do have cardiovascular disease, what does that actually look like? What part of the heart is failing and why? Because this will tell us our answer.

The very first stop in this train is, like I said earlier, and that's the coronary artery side of the equation. If the vessels that give your heart blood itself become blocked or damaged, you're going to have or likely to experience what's called a myocardial infarction. Again, "myo" meaning "heart," "cardial" being "tissue." Rather, it's a heart attack, is how you've heard this.

Very simple to understand. If you were to stop providing oxygen and nutrients to any of your

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skeletal muscle, they would stop working because we can't go through normal metabolic processes. The heart is the same. So if you have an oxygen block, your heart itself becomes hypoxic. The heart will stop contracting. This will cause necrosis or damage, or if severe enough, you will die because the heart will die, and then we don't pump anything throughout the whole world.

So the first thing we're always paying attention to is the coronary arteries themselves. Are they healthy? The second broad category here are what I'm going to call firing issues. And this is the electrical signal that causes contraction. And this can look a lot of different ways. I'll give you two rough ways to think about this.

But the first one is what's called cardiac arrest. This is simply your heart stops charging. It stops firing. It stops contracting. It's not the fact that you were necessarily blocked by oxygen delivery, but something got screwed up in the electrical conductivity that caused the problem. The most deadly here is called v-fib, ventricular fibrillation. So remember, the ventricles sit on the bottom. They squeeze, do the most work, and they push blood to the lungs or to the rest of the body.

V-fib is incredibly dangerous. In fact, it's-- I won't go as far as to say it's a death sentence, but it's pretty close. If you have v-fib, you're likely not going to be exercising. You're going to probably have a surgical intervention. Something's going to go on. This is really, really nasty. It's also very uncommon, but it is particularly difficult to deal with. The left ventricle does not pump, and well, you can finish the story there.

The fibrillation part is a way to think about misfiring. It is fluttering. So instead of that left ventricle beating strong, relaxing, refilling with blood, beating strong again, it's kind of sputtering. You can imagine your car kind of like [IMITATING SPUTTERING ENGINE], shooting some smoke out the back occasionally. Well, what happens with that is, number one, you don't get a strong contraction and so blood doesn't get out. That's problem number one.

The other problem is you have things like because the valve system is working such that we open up a valve from the atria to let blood into the ventricle, then we close that valve. Why? Because if it wasn't closed and the left ventricle pumped, you would actually just be shooting blood right back into the atrium. And so you have to have this open, close, open, close, open, close to create pressure in the system and not shoot blood the wrong direction.

Well, v-fib kind of defeats that purpose because things are firing at the wrong time, and so you're kind of shooting blood into chambers the wrong direction, which again, further reduces blood going out and so forth. So it's a real problem. If you've heard of things like a heart murmur or an irregular heartbeat, oftentimes they're describing this fibrillation.

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I'll reiterate. If it's a ventricular or v-fib, that irregular heartbeat is very dangerous. If it's a-fib, or atrial fibrillation, it is much more common, still serious-- it can absolutely result in death-- but it's way less likely to be problematic. In fact, many people will have this and either not have any concern, not ever know it, or it will be checked out, be cleared by a cardiologist, and you'll return to sport.

If you've ever seen an athlete where-- and this happens every single year. They show up to training camp at the beginning of the season, and then all of a sudden they're sidelined for several weeks because something going on with their heart, and then they're back in the game. A lot of the times, it's because someone picked up an a-fib. They thoroughly checked it out and they said, OK, you're fine, and now you're back in the game.

And so mostly-- I won't say "benign" is the appropriate term here, but they're not nearly as dangerous and you can live a full competitive, athletic life with a-fib and be just fine. And I'll walk you through the data on that specific point a little bit later. But where these do become dangerous is similar to what we just discussed with v-fib, but in the fact that if your atria is not completely pumping blood into the ventricles, that blood can pool. When you pool blood, it tends to clot. If that clot is then moved to the ventricle, it is pushed out into the system. A clot can develop anywhere, which, of course, especially if it happens in your brain, will lead to a stroke, and that's why it can become extremely dangerous.

So those are the problems we're typically dealing with. Why do these things happen? Well, the first stop is genetics. And so I know that a lot of the discussion around sudden cardiac death or athletes that die tends to be like, well, it was something genetic. And that is, in large part, true. But the genetic side of the equation can cause issues with firing.

That's the Wolff-Parkinson-White thing I talked about earlier. Remember Professor White? I told you we'd come back to that. Effectively, what Wolff-Parkinson-White is is a genetic disease that causes a-fib. OK, now, it's more complicated than that. I don't mean to diminish that topic, but that's really what we're looking at. And I remember in the 1990s, people were terrified. This is why everyone started talking about Wolff-Parkinson-White.

But then we actually started to realize, well, again, it can be deadly, and certainly people have died from it. Most of the time, people are fine. It's OK. It's not great, but it is manageable. There are many, many, many of these, you know, genetic dispositions, especially with firing sequences. I don't have time to cover them all. But one I do want to point out is called LDS. This is Loeys-Dietz Syndrome. And this is exactly what happened, unfortunately, to my friend Mike's son Lucas. And so I wanted to bring that up because I know they are raising funds for that, and I'm going to put a link in the show notes for the fundraiser they're doing for that.

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But LDS is a situation that causes connective tissue issues, particularly in the heart, blood vessels and other surrounding areas, and this leads to what are called premature atrial aneurysms. And so effectively, we have issues in the vasculature around it. Young, lean, healthy, Louis himself passed away while swimming. He was actually doing a cool down after practice. Even though they tried to revive him and perform CPR because there's an aneurysm there, there was just nothing they could do about it.

So I wanted to make sure I called that out for Mike and Lucas. And again, I would encourage you to consider donating to that cause, of course, to provide more research there. I will personally be making a donation to that as well, have already. But I wanted to call that out for those folks.

On top of that, going past the genetic side of the equation, another one that happens is that HCM. You could have a potential issue with the firing side of the equation. It is also known that some people are simply born with an enlarged heart-- again, hypertrophic cardiomyopathy. This is problematic because it is the most common cause of sudden cardiac death in young individuals. I'll go over some of the numbers and how frequently it is a little bit later, but this is not necessarily a firing issue. It's simply the fact that you have an enlarged heart.

And the enlargement comes not from the left ventricle being bigger, but because it actually often almost always or frequently comes with a reduction of chamber size. So imagine the left ventricle itself. You've got a thick wall. Well, when you strength train, you gain and you grow muscle. That's muscle hypertrophy. Same thing happening here.

But in this particular case, the hypertrophy occurs internally. So the chamber itself, the area that can hold blood, gets smaller. And so then when you exert, you go through high physical exertion, this becomes a problem because you can't match your output. The ventricle doesn't have enough time to refill with blood to pump again. And this leads to that, again, sudden cardiac death.

So while we can't prevent, at this point, any of these genetic abnormalities, we can do things about it in terms of screening, and in the case of HCM, there are some medications that are possible, which we'll get into a little bit later. We need to differentiate that genetic-based concern from chronic heart disease. So this is maladaptations or pathology that occurred as a result of a lifetime, not as a result of a genetic predisposition.

What we're talking about here, and the analogy or the example I've used several times and will continue to use are things like chronic high blood pressure. What this can do is it mimics exercise, but instead of giving you a little bit of exercise for a few minutes or hours a day, your aorta are on resistance every single minute of every single day for years on end. If I told you you

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were going to do that for one of your skeletal muscles, you'd think, well, that's probably going to be bad for the tissue.

Well, the same thing happens with the heart. It's bad for that as well. So this is, in fact, what high blood pressure is. Because we have either a number of different issues that cause the aorta to either stiffen or become what we call less compliant, more rigid, or the pressure itself is too difficult to overcome. That left ventricle has to squeeze so incredibly hard to get the blood out for every pump, the vascular itself can become damaged.

So you just imagine the aorta being a fillable hose, a rubbery hose. Well, instead of being a hose where when you put more pressure on it, it expands, and then when you reduce the pressure, it will go back to its normal size. Now that turns into a brittle concrete. And so the pressure in the system is higher because the vessels themselves, the vasculature, does not move. And in fact, if you do it too much and there's too much turbulence, that's kind of like the blood smashing and slamming up against those walls, you can actually cause issues with that if not rupture them entirely.

And so because we lack compliance there, this also induces scar tissue in the left ventricle. This is technically called fibrosis. It's not exactly scar tissue, but this is an equivalent for our conversation today. Just like if you overuse skeletal muscle like this, you wouldn't be surprised to develop some scar tissue there. The exact same thing happens in the left ventricle. And so the true problems here are, again, lack of vascular compliance. You also now have an increase in scar tissue or fibrosis. This induces that left ventricular hypertrophy specifically a reduction in chamber size. It's an inward growth. You also then, because of all that, that compiles to reduce contractile strength.

The heart is physically weaker. The individual muscle fibers themselves do not contract with enough force to push enough blood out, you become weaker, and now you're actually in a cycle of problem. All this combines to reduce blood flow and make life more challenging.

So why this entire topic becomes so difficult is because the people that are suffering from these issues I just mentioned, outside of the chronic high blood pressure, it's typically asymptomatic. And in fact, the ones that are dealing with the athlete heart or the runner's heart generally have no symptoms and no other comorbidities.

They don't drink. They don't smoke. They're not overweight. They're typically lean. They're physically active. They stress manage. They do all the things right and they still deal with this issue, and so this is why we often call this stuff "the silent killer." Oftentimes, if you have multiple comorbidities, you're not surprised to learn you've got cardiovascular or heart disease. You know you're overweight, you smoke, so on and so forth.

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But in this case, you don't know that because you're probably doing all, if not most of the right lifestyle things but you're still dealing with this concern. And so the term we use to describe this now, instead of calling it "athlete's heart" scientifically, is EICR-- Exercise-Induced Cardiovascular Remodeling. And this is an important term because we're saying the remodeling or the change in size and function is exercise-induced. That front flag there of "exercise-induced" is our way to distinguish the same end result because it came from exercise is now not pathological but potentially even advantageous.

And so it's really difficult to identify, looking at this stuff, as I mentioned, just as a screen, if you were to run in and get, say, some imaging of your heart, you may not necessarily see what's going on here. In fact, there was some data that I came across showing that even if you were to run an ECG, you probably would miss 50% of these issues because there's not one single place to go and because you don't necessarily know if this is maladaptive or not.

And when it comes to HCA, I told you I'd share with you some numbers earlier, and here you go. About 1 in 500 people have HCM, but very few will be dead from it and the vast majority will never even know that they have it. And so typically, in this show, I like to cover it in the format of the three I's-- the first I being "Investigate," which is how do I measure it, second being, how do I "Interpret" those results, and the third eye being how I "Intervene," what do I do about it?

I'm going to switch that order a little bit, and I'm going to cover the second I, Interpretation, first. And then I'll get into Investigation, or what do I measure, what tests do I run a little bit later. And that's because you really-- honestly, we've been covering Interpret for the vast majority of our conversation already. And so I just want to finish polishing that part off, and then I'll share with you what tests you can run and what you can do about it a little bit after that.

So let's start that second I right now. In the preparation for today, I read a lot of papers and found a ton of excellent resources. And I'm going to put the vast majority in the show notes, and that's because it's really very technical. I just didn't want to spend the time covering the millimeters of thickness of the left ventricle and things like that, where you just don't really know what that means. But I do want to include all those in the show notes.

But the Exercise-Induced Cardiac Remodeling, EICR, has three main components-- the first being structural, we've described a number of these things already; the second being functional, how does the heart work; and the third being electrical. And we can go through this pretty quickly because we've laid the foundation already. Overall summary here.

From a structural side, we're going to hone in mostly on that left ventricle, and we're looking at the overall size or mass. We're looking at the dilation or the chamber size. This is described a bunch of different ways, but that's what we're referring to, so how big the ventricle is itself, how

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much space it has to fill up with blood. And the third one is called supernormal diastole. This is basically saying when we have exercise-induced heart, cardiovascular, changes, the ventricles themselves get an excessive ability to relax.

Now we actually see the same thing happening in skeletal muscle. One of the fastest ways that you can increase your strength is when you start to actually learn how to relax your muscles faster in between contractions. I know this sounds counterintuitive. Go check out our muscle episodes there. But the same thing happens here in the heart. The faster your arteries and ventricles can relax in between beats, and the more they can distend, the more blood you can fill them back up with, which means more blood can come out of them.

And so that's something we really want to pay attention to, that left ventricular both filling space as well as relaxation speed, if you want to think about it that way. So those are the big structural ones. From a functional perspective, it really is honestly kind of this simple. If the adaptation induces greater stroke volume, it's almost always positive. If it induces reduced or less stroke volume, it's almost always negative and you could basically full stop there.

Now what's stroke volume? Every time your heart pumps, it kicks out a certain amount of blood. The amount of blood that comes out of that is the volume of the stroke or the heartbeat. That is what we're talking about. So the more blood that comes out, your stroke volume, however it got there, it's almost always assured to be a good thing. Or if it reduces it, it's generally bad. And this makes so much sense once we think about how the left ventricle works.

If the net result is you pumping more blood out per pump, it's probably good. If it's the opposite, it makes sense that that's a bad thing. Think about this from the perspective of VO2 max. Everyone is excited and talking about how you need to maximize this to live as long as you can. Well, we need to realize and understand the fact that one of the major determinants of your VO2 max is your stroke volume. You can go look up the full equation, and I've described it elsewhere.

But for right now, if you maximize or increase your stroke volume, you're going to increase your VO2 max. And in fact, one would argue pretty well that that is the single biggest way to improve your VO2 max, is to increase that stroke volume. And that's because things like your heart rate, which is another part of VO2 max equation, is fairly fixed. You really can't increase your maximal heart rate. Your age, your sex, and genetics and things like that are going to tell you where you're at there.

In fact, your max heart rate is only going to go down as you age. But your stroke volume is extremely plastic and we have, gosh, 60-plus years of research on stroke volume increasing with exercise. It's incredibly robust. It does not take running. It does not take aerobic exercise, high intensity, low intensity, blah, blah, blah. It's nonspecific responder, but a very, very fast and

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eloquent responder to exercise. So that's our place of improvement.

What ends up happening there is you get two things. You get an increase in ventricular filling, so the ventricles fill up with more blood. More blood in there means there's more blood to pump. You also have what's called lower end diastolic volume, which just simply means after the end of the pump, you have less total volume. A fancy way of saying-- or similar, I guess, of describing a phenomenon called ejection fraction.

We don't need to go into the details here, but just think about it. If you had-- I'll just say 10 units of blood in your ventricle and you pumped out five, your ejection fraction would be 50. In other words, of all that was possible there, how much was left after the beat? If you can improve that, and so now your ejection fraction, you only have two or three units left, you pumped out seven or eight, then again, you can do the math there. That makes general sense.

So more on all those potentially in a future episode. But that's basically what we're talking about. So we described the different structural changes. The functional or the outcome changes are the ones I just highlighted. And the third, electrical, are things like an increase in vagal tone. So remember the vagus nerve goes directly to your heart. When you ramp this thing up, you lower your heart rate. And this is one of the things that happens with chronic endurance exercise that allows your heart rate to lower is because the fact you don't have to pump as often because you're getting out more blood per pump.

The mechanism here is, again, an increase in vagal tone. Now, the vagus nerve is what regulates this drive between your sympathetic and parasympathetic system. And so what also happens here, because you have a lower or increased vagal tone, you have a more relaxed heart, which allows it to feel more. It's also then what causes the problems with things like a-fib. So I'm jumping ahead a little bit, but since we're right here, if you have a-- just think of it as like a more relaxed and zen heart.

And so remember, those valves are supposed to be shutting tightly after the pump. Well, when you're more relaxed and you're chilling, they don't have as much urgency to shut and they don't slam all the way. So when you have these nice big, hard contractions, a little bit of blood kind of shoots back into the atria that causes this heart murmur. That's effectively what we think is happening with exercise-induced a-fib, is you actually get such an increase in vagal tone and such an increase in overall plasticity of the heart. In this case, it dilates more that we just get this little bit of extra fluttering and flickering there.

And so it's at this point not necessarily knowing how dangerous that is, if it is exercise-induced. It's also not known if this just happens to be noticed more in people who exercise, because they are-- not always but oftentimes a little bit more in tune with what's happening in their body, so

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they might feel this a little bit more. So you can kind of blame this a little bit on your heart being a little bit too relaxed. Maybe we should ease up a little bit on the vagal tone there, pay a little bit more attention, but it also may not be pathological.

Highlighting everything right there, worthy of a little bit more discussion of each one of those three areas in detail right here. So let's start with the left ventricle. I've mentioned this stuff already, so we can recap this. But remember, if it enhances stroke volume and it enhances exercise performance, it's probably good.

We talked about that chamber dilation. Remember, this is the inside getting larger. A lot of research from large cohorts of athletes-- one in particular that's used quite often here is a group of Italian athletes. They've done similar things with athletes here in the US, and in general, we're hearing about the same things in all these papers. But a large percentage-- in fact, I've seen some studies that have even indicated up to 80% to 90% of endurance athletes are going to have an increase in chamber size.

So I think it's fair to say this is a very common adaptation that you'll see routinely. If you want exact numbers there, you're going to-- depending on the paper, something about, you know, 50%, 5-0 percent, will have an increase in left ventricle diameter of more than 55 millimeters. If you're a cardiologist, that will make sense to you. If you're not, fine. And then you're somewhere between 10% and 20% will be even greater than 60 millimeters.

So those numbers don't matter to you, fine, move on. You get the point. If they do, interesting tidbit. In terms of the left ventricular size or the midwall thickening, if you will, it doesn't necessarily happen that often in strength training athletes. And in fact, I'll share with you why exactly this happens in a second. So we're mostly talking about endurance, aerobic, or cardiovascular training-- that's why we call it "cardiovascular training." And most of the time when we see this we do see an increase in that wall thickness-- you know, 12 to kind of 13, 14 millimeters is the range there.

You start getting past 15, you might want to do some additional testing. But those are kind of where the numbers generally lie there. A number of other things may pop up that are worthy of note here, things like asymmetrical hypertrophy is a particular problem. So whether this is excessively larger left ventricular size relative to right size; fibrosis, as I've mentioned; contractile reserve or ejection fraction being poor, and there's a whole host of things you can look at, again, if you're in the cardiology side that you'd want to pay attention to.

But the point that I'm making here is something we've said several times now, the fact that left ventricular size alone can't differentiate the EICR from cardiomyopathy, unfortunately. But it can leave us clues and it can tell us where we want to go from there. Some terms you're going to

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hear here routinely-- and I know, again, we're going to get a little bit technical here, so stay with me. And I'm bringing this up, though, because these are common and exercise science but they have a different meaning in cardiology.

"Eccentric" versus "concentric." When you say those in exercise, they mean something different. When you say them with a heart, "concentric remodeling" is generally the bad thing. Here's what they're referring to. Remember the high blood pressure example I've given earlier? If you have chronic high blood pressure, your left ventricle has to squeeze, concentric, like it's a squeezed contraction, all the time. This often results in that reduced chamber size.

However, when you're chronically exercising, because when you exercise, most of your arteries and veins dilate to increase blood flow, that actually doesn't cause the left ventricle to overcome higher blood pressure acutely. What does happen, though, is you're pushing way more blood out, and so you have a lot more blood coming back in. This is called preload or venous returns, the amount of blood returning from the veins going back and slamming into that right atria.

That all slams into there. So what you're actually seeing is pressure coming from blood pushing outward on the chamber. So instead of the chamber having to contract to push the blood out to the body, it's filling up. Just imagine me dumping a bunch of water into a balloon, and the balloon expands inside because of pressure from the water pushing inside pushing outwards. That's why they call it eccentric. It's extending. It's not causing the fibers there to contract and shorten. It's actually causing the chambers to lengthen or widen because of internal pressure because so much blood flow is smashing back into the arteries and ventricles.

This is why that, quote unquote, "eccentric" stimuli tends to result in a greater chamber size. Folks, this is as SAID principle as it gets-- Specific Adaptation to Imposed Demand. The demand is different between these two things, so while they both result in your, quote, unquote, "heart getting bigger," they're getting bigger differently based specifically on the way that they are being stressed. And this is why I felt appropriate to take the time to walk you through both this anatomy and a little bit of this technical jargon, because it actually hopefully makes this less confusing and is now like, well, why is this-- well, this is why.

It's not actually identical. Even though it feels identical, it is quite different and so we have a different physiological response. Now we could do the exact same thing here with actual exercise. So again, in the heart, they call it isotonic exercise. This is your endurance stuff. This is your running, cycling, swimming.

When you do that stuff, I mean, think about what has to happen. You have to have a lot of volume of blood sustained for a long period of time. And you don't have a lot of what's called peripheral resistance. In fact, oftentimes it's less. You've dilated vessels. You're not squeezing a

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ton of muscle, right? When you squeeze muscle that squeezes the blood vessels, that makes it harder to pump blood.

You're not doing that a ton with this classic endurance style of exercise. And so what you generally are commonly see in response to this isotonic cardiovascular exercise is a fairly even distribution of dilation between all four cardiac chambers. So the atria get bigger and ventricles get bigger. This is a balanced hypertrophy, if you will.

Now there are some differentiators there within subgroups of exercise. Like, for example, there's some literature on rowers who tend to develop a little more eccentric left ventricular hypertrophy just due to more pressure, where distance runners are going to be looking more like eccentric left ventricular hypertrophy with normal left ventricular mass. So it gets larger, but not-- if that blew past you, don't worry about it. But if you're in those groups of athletes, you might have found that interesting.

This differs from strength training or resistance exercise, where we would generally call this, again, isometric. Again, not at the level of the skeletal muscle. It's isometric for the heart. This is lifting, this is wrestling, things like that. Well, now it's the exact opposite of what I just described. You have a huge increase in peripheral resistance because, as I just mentioned-- in fact, if you look at some of the literature, you'll see blood pressure during a maximal effort deadlift be as high as 450 over 300 or even higher potentially, which basically means you're full blood occlusion. No blood is pumping anywhere because all of the muscles or most of them on your body are squeezing so hard blood can't move.

So it's a different stressor to your heart, but it's still a positive stressor. And so you combine this with a little bit of a pressure overload, this is when you tend to see that mid left ventricular wall thickening, but you don't see chamber dilation. We're not getting a ton of preload back. We're now causing a lot of blood to be moving a ton when we're strength training, again, in general. So we're not seeing a ton of blood returning. We're not seeing that increasing diameter size.

And so if one were to want to maximize their cardiovascular health, this is why, in general, strength training, speed training, power training are probably not our best bet to maximize cardiovascular health. They have some benefits, but to really see those increases, improvements in chamber size, which is, again, as we described, a really positive thing, we're probably going to have to do some sustained effort at some point.

So now that we've got that understood, we can kind of move on and talk about some of the things that will determine how much of that adaptation occurs and how much does not. I've mentioned really quickly earlier-- and I just honestly don't have enough data to go off of here, but there does seem to be sex specific differences in the amount of exercise-induced cardiac

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remodeling that occurs. There are some excellent studies done with several hundred elite athletes, and it just doesn't seem to be moving as much in females as it does in men. Though it does move, and in the show notes, you can look at the papers I'm talking about and look at the specifics there, if that's of interest.

But there's also things to consider, like ancestry and genetics. And this is actually where the field is moving. But it's really early. I've seen a number of people jump, I think, to what are our unfair conclusions at this point. But I can tell you right now, we are really almost in the dark ages here.

Here's what I'm getting at. I'll share some more differences towards the end of today, but what we have seen now is most of the research that has even looked into genetics are trying to identify single-point mutations. And while it does seem that much of these cardiomyopathies maybe have a single place of error, it's not consistent. And so unfortunately, we can't run a single genetic test. We can't look at a single nucleotide polymorphism and tell you you have one of these issues.

I'm saying that because I've seen this now purported several places, and people are selling these things, and it doesn't match with the literature. It's just not consistent. We've also seen a little sprinkling of this being different between folks that are, quote, "white" versus folks that are, quote, "Black." And I'm saying it that way because that's really all the literature does.

You'll run studies like this. They'll often use questionnaires and they'll say, are you white, are you Black? But as we all know at this point, there are a enormous number of differences between all white people and all Black people. So we need to break these into subgroups because that almost tells us nothing. And so this is actually where I hope the field will move to, is saying, let's move to more appropriate and more specific ways to categorize folks to see are there subpopulations that are at a higher risk of either the genetic predispositions or the exercise-induced or otherwise.

So we have some information there, but again, we don't have a crystal clear understanding of what that means because of, I'll just say, suboptimal or lack of precision with even how we categorize different ancestries and genetic groups for this particular topic. Third thing to consider here, and this is the big one that we can get into, and this is what we call training exposure. And this should be fairly intuitive.

Depending on how long you train, how hard you train, how often you train, this will determine how much adaptation actually occurs. I don't have exact numbers to give you, but I'll do my best here. What it looks like is happening is that initial cardiac remodeling is happening in that concentric phase. After that, the eccentric starts to occur. And this is, again what we're looking for with sustained exposure.

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The minimal thresholds, there seems to be a breaking point at about five hours of exercise per week. OK, so depending on what paper you're going to look at it, again, a little bit different, but somewhere in the neighborhood of four to hours seems to be the breaking point. So if you're doing less than that, you might experience cardiovascular adaptations-- bradycardia, lower heart rate, VO2 max go up.

But in terms of the structural remodeling-- ventricular size, so on and so forth-- you probably need to be accruing, again, in that four to five hours per week range consistently for many months, if not years, before the heart itself will start to change its actual size and structure. Past four to eight hours is when we start to see some of these other adaptations that we've described.

So again, remember here, we're not talking about functional changes, VO2 max, or stroke volume. Those happen really quickly. Those can actually happen with even minutes of exercise per day. In fact, I recently posted on social media some new data actually on not only aerobic or endurance exercise snacks, but there's some research now on strength training snacks. So these are things where you basically break out and do as many push-ups and sit-ups as you can, body weight, and then go back to exercise.

These things have been shown to improve VO2 max. Now a small amount in untrained people, but it highlights a point. You don't need hours and hours and hours of exercise to see an improvement in function, but you probably need several hours per week for a long time to see changes in anatomical or morphological positions of the actual heart itself.

That number probably is leaving a little bit higher in older individuals, and that would be consistent with everything else we see in exercise science. Probably just takes a little bit longer for those adaptations to occur, but it does seem to occur. So even if you are an older individual and you haven't been doing your cardiovascular training for some years or maybe longer, it's not too late. You can still do it, and we still would expect to see positive morphological changes in the body.

So I think that is a fair summary of what's happening in the left ventricle. I spent the most time there for the reasons I've described a little bit earlier. I will hit a couple of other things before we leave this position, but I'll go much quicker through them because there's less data for us to cover and it's a little bit less relevance.

Today's episode is brought to you by Truemed. Lately, I've been thinking about what actually drives performance. It really comes down to two things-- the quality of your training and how well you recover from it. And both sides of that equation matter, and the tools you use to train and the tools you use to recover-- high quality fitness equipment, sleep support, recovery tools-- can get expensive.

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So starting with the right ventricle, it's honestly pretty similar. But really, it's more difficult than the left ventricle because we don't have normative data. So while I was able to give you pretty good information about the left ventricle in terms of actual size, we don't know what that looks like from the right ventricle. And so we're going to lean heavily on what we understand from the left side at this point, and then actually just go ahead and move on to the atria.

There are really well-established adaptations here. Aerobic exercises, endurance side of the equation, these folks almost always have a larger left atria. And if we remember the anatomy from several moments ago, this should make plenty of sense. Some good studies have been done here. You're talking about research on several hundred, sometimes up to 500 or 600 athletes, and we really consistently see this finding this left atria volume just being much bigger than age and body weight matched controls. So it's very, very similar conclusions between the right atria and the left atria, and I think we know our story and so we'll go ahead and move on.

Other stuff to pay attention to that I will also go quickly over are the things that I've mentioned before with the ejection fraction. So some papers have shown up to 90% of endurance athletes will have either the same or an elevated or increased ejection fraction. So a reminder, this means you're getting more blood out of the left ventricle per pump. That's a good thing. We don't need to waste our time pumping it and leaving half the blood still in the ventricle.

And so an elevated ejection fraction is generally good. But at the same time, you actually then see about 10% of athletes that have lower than expected ejection fraction, lower than 50%, if you will. So something to pay attention to. You're going to expect positive results there, but if you don't see or you see actually lower than standard, this may be something you want to look into for further investigation there. It could be completely irrelevant. It could be a sign of early

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cardiomyopathy, or it could be actually some other thing that we just don't have any idea about, especially if we know that the net result is increase in exercise function, overall cardiovascular fitness, we need to take all these things into context before making a clinical evaluation.

All right. Some other stuff to pay attention to here. Some data suggest that this ejection fraction is actually particularly genetically driven. I don't know how good that stuff is at this point in terms of quality of evaluation, but I did see some stuff there, and so I wanted to flag it as something to potentially pay attention to. Probably more specifically, here, we're looking at a different number of polymorphisms that have been identified in these kind of large genetic testing pools.

To me, when I read those papers, I kind of walked away going, maybe? I don't know. OK, I see something here, but this was a too-early signal for me, personally. If you read it, maybe you feel differently, but I'll leave that up to you. So I'm waiting for more evidence on that particular point before I draw any further conclusions, but something to pay attention to.

I want to highlight a couple of really landmark studies in this area. There's a series of them called the ProHeart Studies. And just to summarize them really, really quickly here, one of them in particular had 280 or so participants. These are early, you know, 18, 19-year-olds. But what's interesting is they're elite athletes. So you're talking about mostly males, but these are national and international-level endurance athletes. I think the average VO2 max here was 63 or 64, and these folks were exercising 15 to 16 hours per week.

And when you looked at that, you started seeing things like a large percentage of them, somewhere up to 15% to 16%, had that reduced ejection fraction either in either the left ventricle or the right ventricle. And then some other smaller percentages had maybe reduced ejection fraction in the right side only or the left side only, or some of them had both.

And so the overall findings were, again, like I described earlier, the fact that while the majority of endurance athletes have an improvement in ejection fraction, a non small amount, sometimes up to 10%, actually see worse. But now several times have said this, we actually just don't know if that's bad or not. But we're going to call that good for our understanding of this part and move on to the next thing I want to discuss, which is kind of a way of saying, when it actually goes bad, what is really happening here?

As a reminder, exercise is almost always a good thing. But the real question on this particular topic is if we take exercise to an extreme, what could plausibly happen? What are the maladaptations? You can really boil them down into three particular things. The first is arrhythmia risk. That's that a-fib I'm talking about, "arrhythmia" being you're out of rhythm or out of sequence. So that's the firing or the electrical or the cyclical part becomes weird.

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Another one we have not discussed yet is called coronary artery calcification. So remember, the coronary arteries, the ones that pump to your heart, become calcified. If those things either burst or rupture, then we've got our myocardial infarction. And the third one, which we've mentioned several times now is this myocardial fibrosis or scarring tissue. So if we do have an exercise-induced problem that is not just genetically HCM or something else, these are the three big things that will actually happen.

And so there's a lot of debate on these particular topics and a lot to be said. I will say this-- the arrhythmia risk is there. It is very real. It is documented. A-fib will be seen very consistently. If you look at the research, it seems most prevalent in people that combine both high intensity and high volume for long periods of time. Again, you're talking years to decades. This should not stop anyone from doing mesocyclones or months of intensified training in both volume and duration.

What we're talking about here are the folks that do this consistently for years, if not decades. And I'll share with you what that actually means numbers wise here, right now. Really classic example, probably one of the more infamous studies in this area-- at a Sweden cross-country, skiers, there was 50,000 or so people in this database, and they found that of these cross-country racers, the people that are finishing at least in the top half of those groups, so generally the better skiers, the faster people, which is a way of interpreting-- you can kind of interpret that as probably people who are training more, a fair way to summarize that-- those individuals had a 1.3 times greater risk of having a-fib.

Other studies have found an even higher number. So you're talking about people in this particular database, when they sub and analyze this into people who have done this race more than five times, you saw an additional 1.3 risk on top of those other individuals. And so 1.3, is that a lot? Is that not a lot? Well, I'll leave you to decide that, but that is a number. And you will, again, see things like that really consistently across the literature.

And so why this becomes very interesting is the people that present with a-fib that are from databases like this have almost always zero other health risk factors that are known to cause a-fib. So in a normal person, if you see a-fib, you're almost always going to see either hypertension, apnea, you'll have some sort of valve disease or cardiomyopathy. But these people don't have that, and so it's presenting itself in a different way. This is why it's that silent killer, if it is, or this is why it's difficult to see.

I would say that if you were to circle up the experts in this field, it wouldn't be crazy to say that you have upwards of a five times higher risk of a-fib if you are engaging in a lot of high-intensity exercise over many, many years. That number would not-- some might disagree on that, but it wouldn't be crazy to put that out there.

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Why this is problematic is the risk of fibrosis. It's the risk of the scar tissue actually developing because, as we mentioned, when you actually do this stuff, you're actually doing some exercise adaptations that lead to more stroke volume. And so the functional outcome is actually higher, that vagal tone is higher, and it's the exact same thing that leads to the extra adaptation. So the thing that's giving you more ability to pump blood is the same thing that's also giving you the risk for a-fib, and so you see it really, really, really commonly.

And this is where the detractors here, the too much exercise is bad for you, are going to lay heavily, is on this set of data. And again, they're not wrong. You will see this really consistently. It is a real thing that happens.

But it's really a function of risk predisposition versus too much high-intensity exercise. One of the things we're starting to learn is it's probably not that combination of exercise. It's probably that combination of that exercise in a subset of people who have some genetic predisposition that we are not yet aware of. That's what seems to be happening.

And so hopefully, in some short time, we'll start to figure out more what that looks like that could then lend to development of potential screeners or something. But we don't know what that stuff is quite yet. So I don't want us to lose the plot. While there is a slight bump in a-fib in endurance athletes, it's also done in exchange for a massive drop in basically everything else. So your risk of mortality, your risk of heart disease itself, and almost anything else you throw at it, aerobic and endurance exercise is, on net, very, very positive.

So this is a matter of how are we twisting the story. If you want to focus and write an article just on a-fib, sure, we can do that accurately. But we're not seeing people dying from aerobic exercise. In fact, almost all the research will point to the opposite. So we got to be really clear with how we're describing this risk and what it means.

I will also reiterate on a-fib, the vast majority of the research is in men. The gist really is we just don't see it as much in women and there hasn't been enough research in female athletes. It tends to be a little bit less in them for the data that we have, but I don't think we have a fair, complete picture of that.

So there's a couple of papers-- again, I'll leave for you to read one that I think is very interesting called "Athlete's Heart or Heart at Risk? Cardiac Remodeling in Exercise-Induced Ventricular Arrhythmias in Elite Athletes." And I'm bringing this up because what we described at the beginning, I just talked about a-fib. But v-fib, ventricular arrhythmia, is a different thing entirely. If we see v-fib in athletes, it's probably not a good thing, and it's probably not exercise-induced.

Recent study was just published on this looking at over 2,500 athletes, and it does not seem, at

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this point, that v-fib is something that happens as an exercise adaptation. So when we do see this-- and this paper, again, was in really high-level athletes, I think Olympic level athletes-- it does not seem to be detrimental. So more in that particular area, but that's something we want to pay attention to.

We've had plenty of discussion about the fibs side of the equation, but I want to move on to something we have touched lightly, and that is the calcification stuff. If you're interested at all in the heart, you've probably have thought about, discussed, or heard this calcification idea. I won't go into all the pathophysiology at this point, but one thing you will consistently see is athletes very frequently have an elevated coronary artery calcification score.

What's really interesting, though, is how bad is it? Is it actually detrimental? Almost always, if we see calcification or plaque buildup of any kind in the coronary arteries, this is a problem. But we're seeing it a lot, and in athletes, we don't know if that really is the issue. So the pathology here is unclear.

It can happen for a lot of different reasons-- inflammation, I mentioned earlier this hemodynamics where the blood is just smashing into the arteries themselves and causing vascular tears and all kinds of other things like that, tons of other issues. But the maladaptive process of atherosclerosis in this particular example, is it benign? Is it vascular remodeling? Is it something else? It's really quite on the table.

In fact, I can share I've had plenty of discussion with a number of radiologists and cardiologists in the last three or four years on this, and those that have seen plenty of athletes come into their clinic, everyone's kind of confused here. Like, we really actually don't know what's going on. But calcification is such a scary thing, you almost always opt to just, let's treat it. Because until we know clearly it's not a problem with you, it's fair to say that's kind of risky to just leave it by itself.

And so at this point, I would default to the people who are saying, hey, look, I actually don't know if it's normal for athletes, but I'm not willing to run that risk. So let's use a statin or some other form of medication to address that specific issue.

So really, I think the most compelling research on this, another Cooper Clinic study, 20-plus thousand-- I think it was actually closer to 26,000 people-- followed some folks for a couple of decades. And yeah, sure, found that those with higher calcification scores were associated with more heart attacks and other detrimental revascularization processes.

But-- and I'm going to be talking out of both sides of my mouth here, because this is the story of this topic-- you also saw those people with lower all-cause mortality risk. So the interpretation of that would be probably likely to have a higher calcification score, likely to then have a higher risk

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of heart attack, but less likely to die from it.

I think if you have a different interpretation of the data at this point, I couldn't push back on you. I would say, OK, fine. We're kind of doing some jumps in logic there. I think it's fair, though, to say this does indicate that fitness, cardiovascular health will offset some of the anatomical changes that happen. Another way of saying that would be its potential you could interpret this to say your fitness allows you to get away with some anatomical changes that are otherwise problematic.

I'll reiterate this is a risk tolerance issue. You can interpret these how you'd like to do there. But that's really where the field is kind of arguing, I think, fairly at this point. The stuff I talked about earlier, I think, was an unfair way to position it from the ACSM meeting. This, I think, is a fair way to have controversy. It's a very fair argument back and forth here.

Ben Levine, really, really classic scientist in this area, has done lots of work, also published some compelling data here. And I think this is really interesting. I want to highlight it because their evidence actually suggested that higher levels of physical activity, being defined as more than 3,000 met minutes per week, which I'll describe a little bit later, these are associated with increased prevalence of this calcification score. But those are not associated with an increased all-cause or cardiovascular disease risk, even after a decade of follow up, even in the presence of what's called clinically significant coronary artery scores.

So what you're talking about here are people who are doing an average of 30 miles per week of running at about a 10-minute pace. They're not being harmed. They're not dying. They're there. So again, those are the data. You interpret that how you will.

And one more time, just to put the cherry on top, this seems to be a bigger problem for men than it is for women. Women certainly show typically no additional coronary artery problems in female athletes versus controls. So for some reason, it seems to be happening in men, if not exclusively, certainly more predominantly in them.

We will give some final thoughts on this calcification stuff at the end, but I want to move on right now to the myocardial fibrosis, again, this scarring of the tissue there. There's some new evidence that suggests this might be happening with high-intensity exercise, more so than lower-intensity exercise, which fits with the previous theme that the problems really happen when you combine high volume with high intensity for large portion.

That scarring process can cause a bunch of problems, from mechanical stress to inflammation, to ischemia, to oxidative injuries, and making the chambers more stiff and less compliant, and so forth. We see it happen in the atria, the right ventricle, the septum, the spot in the middle of the heart, and a bunch of different areas. But that said, I think this would be argued as-- of the three

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things we've just discussed, this is the area of most unclear data.

And I can give you a highlight of this from a very specific study that dove into this. And they examined, in their analysis, a whole bunch of veteran endurance athletes. So these folks were over 50 years old. They had cardiac fibrosis, and they were at about a 5x risk of non-benign ventricular arrhythmia. But that said, this group was averaging not only 12 hours per week, but they were doing it for 20-plus years and all of them competed about 20 times per year.

So you are seeing this increase in fibrosis in a large amount and increases in ventricular arrhythmias. But you're looking at a whole bunch of people that are doing really high amounts of exercise and tons and tons and tons of competitions. So I don't think it's fair to interpret the same thing would then be happening for the average Joe or even the serious Joe, because that's a high amount of exercise.

And I don't think it applies to even your chronic runners, because even if you are one of these individuals, that amount of exercise for that long is pretty rare. So you're talking, again, if you want to break it down this way, running for two hours a day, six days a week, for 20 years. There just aren't that many people who fall into that category. There are some. I know some. But it's just not that many.

You also contrast that directly with the substantial evidence that exercise-related survival benefits in extreme endurance athletes have also been documented. We've seen this story play out several times now, right? So we've got Scandinavian cohorts and lots of other groups of individuals, including some research that I did in Sweden, looking at 80 and 90-year-old cross-country skiers who started competing in national and Olympic and international races in the 1930s, and '40s and '50s, were still competing 50 to 60 years later, yeah, had all kinds of imaging issues, but their VO2 maxes were almost 40 on average as a group, which is obnoxiously high, and they're independent-living 85 to 90-plus-year-olds.

So even with these extreme endurance stuff, you may potentially see some of this fibrosis. But to summarize here, we see far more evidence for the exact opposite than we do for the damage. We see far more evidence for the fact that-- just look at the data on things like runners who do 40 miles per week have been shown to have upwards of 25% less likely to develop coronary heart disease, even relative to those who are running 13 miles a week. So there are data to show, hey, even if you go from the recommendation, 13 to 15 miles per week, which would be equivalent-- to clarify, that'd be equivalent to kind of normal exercise guidelines, not necessarily running that many miles per week, but that's the amount of exercise you should do. You double or triple that, you're probably dropping your risk of heart disease by 20 or more percent.

So we also have direct evidence. And if you look at a number of different studies that have

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looked at hundreds of thousands of people, if not more, there really seems to be no upper limit for VO2 max. So we aren't seeing inverted use such that when your VO2 max gets too high for too long, that you have an increased risk or even reduced benefit, we just don't seem to see that. So the higher the VO2 max, the higher the survival rate. And if that were not the case here, something would be popping up in flagging and we just aren't seeing that.

Survival rates-- again, studies, I'm thinking of some that are 100,000-plus people from Cleveland Clinic over a 15 or 20 year span. These are articles published in JAMA. And you're seeing, of 125,000 people, you know, 10,000 or 15,000 people are dying after 10 years, but the greatest risk is in the people who have the least fitness.

In fact, in those particular papers, when you subdivide and look at the people that are in the 98th percentile of fitness, so the upper, upper, upper limit, you're still talking like a 20% reduction in risk, mortality. And even those that are in the top 25%, so not even the elite of the elite, are also looking at 20% to 30% reductions in risk. And so when you look at the actual total death rates, we're not seeing the bodies.

So the most compelling research on these extreme exercisers, I think, that we can come up with. Really interesting stuff here. One paper I'm thinking of, the 2,000 men and women in it, average age, in the early 50s. These were folks that were-- and I'm going to say this correctly here-- that we're exercising 35 hours per week on average for on average of 28 years. So if I haven't convinced you yet, that's about as extreme as we can probably get.

It sounds a lot like Caballo Blanco. 10-year follow-ups on these people. There were 66 in this particular trial of those 2,000 that met this type of exercise range. At the end of the 10 years, two of the 66 had died and neither of those two died because of heart disease of any kind. And so, yes, that is 66 people out of an entire population, I get it. But that's the best data.

I mean, do you know how hard it is to find people who exercise 35 hours per week for 30 years and to have none of them dying of cardiovascular disease? Again, I'll let you infer and draw any conclusions you'd like from that, but I know what I think. In fact, I can summarize my thoughts on that. Very similar to what Dr. Paul Thompson said many years ago, and that this entire topic is, quote, "intellectually interesting, it is clinically worth knowing, but it is not worth worrying about."

So that being said, because it is clinically meaning and worthy of paying attention to, I do want to move into our last couple of sections here, which is Investigate. What should you actually measure if you are concerned, have reason to be concerned, or you just find it interesting? You can go about this a number of different ways.

I always like to start from the most affordable, most reasonable thing and work our way up. And

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so I'll start with this. I think it's reasonable to suggest targeted screening. If we look at the data as it stands now, the most susceptible to issues here are from the sports of rowing, running, cycling, swimming and skiing. If you're in one of those sports, we should-- it's reasonable to say maybe we are putting more resources into that as overall screening purposes.

You also have an increased risk of sudden cardiac death in specialized sports like basketball. I remember, as a kid, it seemed like it happened-- I think it was actually in back-to-back years. Reggie Lewis and Hank Gathers, like 1992, '93, both died of this SCD. And there's not a ton of information on sports past these endurance ones I just mentioned. But we are seeing things like this.

If you look at the rate of this occurring in Black, male college athletes, it's about 1 in 16,000. If you go past that and you look at male basketball players, it's 1 in 9,000. If you look at male Black basketball athletes, it's about 1 in 4,000 or so. So these rates aren't actually extremely high. But it's some signal. Maybe that's just an artifact. We have no idea. Is it something specific to basketball? Is it-- I actually don't have any idea.

But I think I'm bringing up here is to say it's reasonable to conclude there's potential accelerated risk in this group of individuals, so maybe we do some additional targeted screening prior to participation or every couple of years or something like that. And this screening can look a lot of different ways, but I think this is how we could start, because, as I'll share in a second, it's not feasible to run a full cardiometabolic panel on every single athlete, not in the US. And so maybe just targeted screening at those most at-risk groups, and right now, the only data we have on that would be these particular groups that I've just described.

A third criteria would then be literally any human being that has a family history of early heart disease. And I cannot emphasize this point enough-- if you have anybody in your immediate-- or I will even say secondary family-- who has died of heart disease earlier than age 55, I would deeply and strongly recommend that you get some sort of cardiovascular screening. Because remember, it's not going to present any symptoms. You're probably not going to have any of the comorbidities.

You can be exactly like my dear friend Joel Jamieson. I've spoken about him many times. He's a world-renowned expert in cardiovascular exercise, endurance training, and he's spoken about this story so I can share it here today. He himself does not smoke or drink or party. He sleeps well. He manages stress. He's lean. Loads of cardiovascular exercise. Probably has a VO2 max upwards of 55, if not higher.

A couple of years ago, he went and got screening done. I think he was 40 years old and found he had a 50% blockage in his widowmaker in his heart. That is as close to death sentence as

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you can possibly get. 50% blockage, zero sign symptoms. Once he thought about it, I think his mom had a stroke at age 60 or something like this. His dad died, I believe, early. His uncle died early. His, I think, brother has had a triple bypass before age 50.

And so he had every sign and signal in the world, and as I started off saying, probably the biggest signal we have here is familial or genetic inheritance. And so if you had had anybody even close to your immediate family die of this-- I, myself, I have. I've had multiple uncles, direct uncles and other ones, great uncles, and so I have I've gone through the screening myself, and my wife pressed me for about a decade to go do it, and I didn't because I'm like, I'm young, I'm healthy, I'm doing all the things. I'm fine.

And if you're leaving this podcast with one thing, I hope it would be that. That is a very poor attitude. It's probably a mistake to do that. If you've got this kind of early signal in your family, regardless of anything else, it's worth doing some assessments. And even in my case, I had to pay out of pocket for this. I mean, if you can at all afford it, it, in my opinion, is worth the risk.

Another thing you can do here that is completely free. The American Heart Association has a free screening program. It is a 12-step screening process. It's a questionnaire. You can take that or you can administer that to your friends or coworkers or whoever you're with. And it asks questions like, do you faint? Do you have chest discomfort while exercising?

In fact, what you'll see a lot really happen often is the symptoms look like asthma. And so you'll ask a lot of questions about, do you have asthma as a kid? Sometimes that's asthma, and sometimes that's actually cardiovascular stuff that you're seeing, because it is these heart murmurs and things that make you feel like you can't catch your breath. So those are things that I would recommend starting with.

Getting back to what I mentioned a second ago, which is what I personally did. Take this for what you will. I'm only bringing this up and sharing it so that you can have something tangible to grab on to. I'm not indicating this is the best or anything else. But I went through something called a coronary CT angiogram. So this is when we're imaging the heart itself in those coronary arteries.

So "CT angiogram" is the phrase you're looking at. Pretty easy, pretty simple. I think it was a 20 or 30-minute scan. That also came with a calcification score. I think hard cost is around $1,200. So that is a lot, but many people hopefully can afford something like that if you flag one of those things I described earlier.

There are other companies. Probably the most famous one is called Cleerly. This is an FDA-cleared machine that does a bunch of non-invasive stuff that looks at atherosclerosis and plaque and stenosis and likelihood of ischemia, and a bunch of other stuff using AI and a bunch

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of other learning models. I have not gone through Cleerly yet myself. I know many people who have.

I think that's a little bit more expensive, maybe close to $2,000 or $2,500. And again, I can't vouch for it other than what I just shared for you. But this is a potential option for those of you that are more at-risk, want maybe a more in-depth analysis, or just have particular interest in those areas.

So that said, there are some other things we can do right now from an intervene perspective. And this looks a lot like the following. The easiest and cheapest thing you can do is a classic D train method. So if you get some imaging done and your cardiologist or physician is concerned about abnormal size, the easiest protocol and the most common one is they'll just tell you to stop exercising for three months. Now, I know for some of you were like, oh, my god, that sounds horrible. Like, the worst possible thing you could ever hear is not exercise for three months.

But come on. You're potentially at risk of dying of sudden cardiac death. Three months of your life, to answer that question, I think we can handle it. And so the reason they're doing that is because that's really the only way we can establish cause and effect. If you don't train for three months and then you get imaging done again and you see a reduction in size there, this gives you some indication that the increase in size was exercise-induced.

But if you don't, so you don't see a drop in size after not exercising for three months, then it's most likely this is genetically driven and now you're probably in that pathological situation. Doesn't mean you stop exercising or can't, but we're at a different risk stratification and we have some potential different techniques and other solutions we need to come up with. So it's certainly not perfect, but I think it is a very reasonable thing that everyone can do, because all it requires you is just not exercise for three months.

And you can't do it for three weeks. You have to do it for a long enough time where morphological changes, anatomical changes will actually occur. That said, something like 20% of people-- athletes, rather, even if they stop exercising, won't see a reduction in size. In fact, there's one paper that found up to five years later, you're still at the same size. So it's not perfect, but it's something to try.

Another thing you can try that is also free is you can hedge towards that lower level of exercise. The threshold seems to be about five hours per week. So if you limit your exercise to an hour a day or so, if you want to, you could make an argument that you might be reducing risk if you have some genetic predisposition for this stuff. At this point, that seems to be the safe zone. So unless you've got one of the real, real struggles genetically, we seem to be OK. Risk an hour or so of exercise.

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And friends, don't be nuts about that number. Hour, 75 minutes-- like, plus or minus. We're trying to differentiate that from the 30 hours per week kind of thing. But that seems to be the number, around an hour of exercise or so per day, which in reality is probably about the most extent that most people do anyways. So most of us are probably fine there.

However, if you choose a different path, fine, for whatever reason, I think you just need to be proactive about your health monitoring. You can't just assume because you're lean, have a high VO2 max, you don't do all the other stuff, that you're going to be OK. You can't just assume also because your blood work looks good, you're OK.

This is something that happened to Joel. He monitored his blood every six months or so for decades. Cholesterol, all that stuff is being measured, and it all looked good. Not making any comments here about the relevance of cholesterol to heart disease or anything like that. I'm simply saying, just because those are good, it doesn't automatically mean your heart is fully good either. Again, back to these things being silent killers. We have to have multiple forms of analysis.

So I think we should wrap ourselves up here with a nice summary and conclusion of what we talked about. When we see these articles pop up and we get this idea of, quote, "too much exercise being bad," it irritates me. I think it's flat out wrong and it sends the wrong message. I understand what people are trying to do here, but the idea that too much exercise is inherently bad for humans is, I'll just say it kindly, most of the time, misguided.

It comes and goes every few years. It'll be back again, I'm sure. But what typically happens is, unfortunately, somebody dies of high profile. It makes headlines. A case study is published or something is put out in a medical journal. And it gets recycled. I have really enjoyed reading David Epstein's work here. There's another writer, Alex Hutchinson. Mike Joyner, who's renowned in this field, have all written extensively about that. You can go read all their articles. These are easy to read things. We'll put them in the show notes.

But these people have been really important voices to me in this field of centering us on, again, what do we know really and what do we not know? And so what do we know? In summary, the same mechanisms that confer benefit can also confer risk. The general mechanisms here we're looking at is cardiac enlargement, particularly the left ventricle, vascular compliance, so being really ability to expand and contract and not break and get damaged, as well as enhanced metabolic flexibility.

The things that are the risk are the arrhythmias, the a-fib in particular, and v-fib being very dangerous itself, potential coronary calcification increases, and myocardial fibrosis or the scarring. Those things are occurring. Is it worth the risk, then? You can probably tell by my tone

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of voice this entire thing, I think it is.

But that's ultimately-- I truly mean this-- up to you. If we look at this on aggregate, we know that longevity, all the way back to the classic London Bus studies to plenty of other ones you want to pick your poison from, VO2 max, cardiovascular fitness, a low resting heart rate, a high stroke volume-- you can pick your poison here-- it's almost always associated with longer health, both length and life quality.

We also know that, just honestly, as a human, VO2 max is one of the things that makes us special. We have a five times higher VO2 max than our closest primate relatives. It's a defining characteristic. You know, in the book I mentioned at the very beginning, Born to Run, he makes the strong argument that this is an inherent human property, that we are quite literally born to run, and we're not necessarily built for strength, but we're built for endurance.

So I think to dissuade people from something that is as natural as running specifically or even exercise in more general terms, I think it's misguided, as I've said before. We also have another line of evidence that I haven't gotten to yet, but I'm very familiar with and that is called cardiac rehab programs. So imagine taking people a few days after having some cardiovascular event and getting them back to exercising. In fact, this is the predominant thing that happens for rehabilitation program. You want to get the heart back to work.

So that alone should tell you this is a good thing. And I remember actually being at a large program-- one of the best programs in the country, honestly, for cardiac rehab, and then actively using as much high intensity exercise as possible in people really quickly after cardiovascular events because they're seeing better outcomes. So I think you tie all this stuff in together, and my conclusion is, look, we shouldn't negate the profound physiological and psychological benefits of exercise. There's so pronounced, they're so robust, it would be silly to throw that all away.

If you're of the type that likes to do the extreme exercise, in this case, tons of volume for a long period of time, it's probably reasonable to pay more attention. Get imaging done, get testing done, get multiple types of it. Don't just get an ECG and think you're clear. Don't just get blood work and think you're clear. You need a robust set of testing to really understand your actual true risk.

If you're exercising more than an hour per day on average, consider that right. Consider your family history. Very limited reason to firmly think this is bad for you. I hope you should not interpret the data that way. I don't think you should cut it back for no risk if you have no reason to do so.

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You also should acknowledge it could be risky for you. It is possible you're doing that. It's fairly rare, but it's not a promise I can tell you that you can exercise as much as you want, you'll never be at risk because that would not be consistent with the literature either. Lastly here, the testing itself is not simple. There is this promise in the field of genetic testing to cure all, solve all, but it's just, quite frankly, not there yet.

So I'm going to end today on my soapbox. This is why we have to study elite performers. We would not know the basic physiology of how the heart works if we weren't looking at people like Caballo Blanco, if we weren't studying these individuals who are pushing the limits of human ability. And does that come with some risk? Well, in the case of Caballo, it did. I don't know if he regrets it or if he ever would. Who knows? But that's not for us to judge. That's not for us to decide.

The exercise science field is critical and needs to be thought of as not something for sports, as something necessary for human health and understanding because, as I've said, always, and we'll say one more final time here today, friends, if you have a body, you are an athlete, like it or not.

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Thank you for listening and never forget, in the famous words of Bill Bowerman, "If you have a

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body, you are an athlete."

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