How & Why to Strengthen Your Heart & Cardiovascular Fitness

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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 Andy Galpin, a professor of kinesiology in the Center for Sport Performance at Cal State Fullerton. In today's episode, we're going to be talking about the heart.

And I'd like to start with a very simple question. And that is, why do you breathe? Now, that may have caught you off guard. And so I'll let you think about it for a quick second. Why is it that you breathe?

The first couple of answers probably rushing to your head are something like, well, if I don't breathe, I'll die. And yes, that's true, but why? Why is it that if you don't breathe, you'll die? With that prompt, and you're now probably thinking about, well, I've got to get oxygen into my system, because oxygen is needed as a fuel for metabolism, to produce energy and to keep my cells in heart and brain alive.

Well, it's not exactly the right answer. Of course, oxygen is critically important, and you will die without it. But there are many other things going on that determine how you breathe, why you breathe, how often you breathe, and why that's vital to both your health and performance.

Given that the focus of this show is to discuss the science and physiology of maximizing performance, I think it's pretty prudent of us to then spend a little bit of time learning more about how and why your heart functions. In order to do that, we're going to cover what I call the three Is, the first being Investigate, another way of saying, how do I understand and analyze whether or not my heart is functioning at the highest level possible?

The second I is Interpretation. How do I value those numbers? Is that great, terrible, amazing, best in world history, et cetera? And then the third one is Intervene, which is a way to say, what do I do about it? How do I improve various markers? How do I reduce others so that I can maximize my overall functionality and performance of my cardiovascular tissue, or in other words, your heart?

In order to do that, we're going to have to expand our conversation past just the heart itself. This is going to include things like respiratory rate. In fact, I opened up the conversation here by asking you why you breathe.

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And so we're going to take a look at not only the cardiac function itself, say, your resting heart rate, maximum heart rate, cardiac output, VO2 max, and things like that, but we'll also get into other important and relative metrics like your heart rate variability, your respiratory rate, CO2 tolerance, and other things that you need to understand to fully appreciate and then therefore improve function of your cardiovascular system.

Before we get started with all that, though, we need to take a quick step back and go through really what the heart is, how it functions, what it's made of. And that will then give us insights and understanding about how to measure it, interpret it, and then therefore, improve it.

Now, before we go too much further, I'd like to take a quick break and thank our sponsors because they make this show possible. Not only are they on this list because they offer great products and services, but because I actually personally love them and use them myself. Today's episode is brought to you by Vitality Blueprint. Vitality is the world's only comprehensive bloodwork company built specifically for high performance.

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Today's episode is also brought to you by Rhone. Rhone is a premium activewear company that is easily my favorite in the world. Few things have aggravated my wife more than my clothing choices. Let's be honest, fashion is not exactly my strong suit, partially because I have just no sense of style, but also because I'm neurotic about texture and feel.

So honestly, no hyperbole here. I bet she has a 90%-plus return rate on items that she's bought

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me in an attempt to salvage my public appearance. But holy cow, did our lives change when we found Rhone recently. There's not a single brand I've tried over the years that has the same quality, feel, and fit that Rhone has absolutely mastered.

Rhone uses best-in-class materials to allow their products to be lightweight and breathable, but still durable enough to last even when you're training super hard. I remember the first time my wife wore it for a cycling workout. And she, who, let's just say, is well-versed in the activewear world, was genuinely surprised by how soft and stretchy but supportive the material was.

The Rhone women's collection actually just launched and is already filled with great reviews online. The Rhone Base Training Shorts are brilliant as well. They fit perfectly and have the right amount of stretch to allow total freedom of movement. They also have pockets, which I like, and zippered pockets that are sneaky helpful for holding hotel keys and things like that.

The Atmosphere Tee and Commuter Business shirts completely blew my mind. I do not understand how they made this material. I'll never go back. Trust me here, friends. Just stunning craftsmanship all the way around.

The wife literally said, wow, when I threw the first blue shirt on. Finding clothes that meet my texture and feel demands and her aesthetic requirements have plagued us for more than a decade, but not anymore, thanks to Rhone.

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OK, the way I'd like to get started is actually with an apology. You see, I'll admit and I'll tell you forthright that skeletal muscle is my favorite. Cardiac is a distant second. And I probably spent too many years not giving the heart its due.

And that's honestly because I came from what would be typically called as an anaerobic sport background. You see, I was much more interested in things like football, a little bit of basketball, baseball, and things that require not a lot of endurance, but a lot of power. And so I didn't really necessarily appreciate. In fact, I directly said that the heart was not nearly as important as your skeletal muscle.

I've come since to learn that that was the wrong approach. And I'll tell you more why about, even if you're into those types of activities, you should care deeply about the functionality of your heart and how that can absolutely improve your performance even in situations and scenarios like that.

OK, so as a quick reminder here, remember, your body has three main types of muscles--

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smooth, cardiac, and skeletal. Now, there's a number of structural and functional differences between these three. And just very quickly, smooth muscle lacks contractile properties.

And so some of the things we're going to get into later, the microanatomy of smooth muscle, it doesn't have, and so it lacks the ability to contract. It can isometrically hold in place. And so this is really something you don't have cognitive control over. It's the stuff that regulates your background physiology, digestion, things like that.

Cardiac tissue, again, when I say that, think the heart. And skeletal muscle, think everything else, so the muscles you can actively control, whether they be small muscles like in your fingers, eyes, or toes, large muscles like your hamstrings or glutes, spinal erectors, and things like that. So everything else is a skeletal muscle.

Now, there's a lot of similarities between skeletal and cardiac muscle, which I'll talk about a little bit later. But there's also some major differences. And that actually is going to explain a lot about how you need to approach these, interpret, diagnose, and then actually train these things differently.

And so I didn't appreciate that earlier in my career. I gave all of the credit to skeletal muscle and didn't understand how important and vital something like my respiratory rate is in terms of performance as well as tracking and monitoring ongoing progress, and then particularly, signs of things like nonfunctional overreaching or overtraining or general fatigue. So I hope that suffices as a small apology for all of you heart experts and aficionados out there.

OK, so let me wind the story back just a little bit so I can set the stage appropriately, and you'll understand why I felt the way I did. Coming out of high school, I knew I was interested in sport performances. So I actually wanted to go to college to learn more about the physiology and science of performance. But those programs really didn't exist.

And so I remember being taken on recruiting visits, and they would ask about your academic interests. And I would say that, and they'd say, well, we have an athletic training program, which is really injury prevention and treatment and management and stuff like that. Or we've got premed, and I didn't want to do that.

And really, the only exercise physiology programs involved exercise. But it was really more public health, disease prevention, treatment management, and stuff like that. And so I never really found a home academically, at least initially.

So I remember going through school. And again, while the exercise was a part of that, it was really from the perspective of, oh yeah, athletes do that. And then there's exercise. You should park your car at the end of the parking lot. You should get more steps in, and you can go

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upstairs. And it was that public health message, which is awesome stuff and incredibly important. It just wasn't my passion.

So I remember almost feeling like I didn't really have much of a home academically. And I would learn stuff, and I was excited about learning the human body. And that stuff fascinated me and still does.

So from the cardiovascular perspective, I just really didn't care that much about that stuff until we got into doing things like testing the VO2 max. I'll tell you what that is a little bit later, and we'll walk through it. But that got my attention, because it's like, hey, this is a maximal exercise test. And it was something we could do for athletics to see who's the most fit, who had the best endurance.

And if you look at the research on sport performance, there are some clear associations. In fact, some of them are very highly tied to success in sports and your VO2 max. Now, classically, you would think of something maybe like an endurance runner, a marathon runner per se. And while the VO2 max is not the only thing at all that predicts performance, clearly, it is higher in those individuals relative to athletes and, say, baseball or golf or something like that.

So some sports, it mattered a lot. In others, it didn't. And there was a way that we could assess and test and identify performance. And it all made sense to me, and I grasped it.

But what I never did was make that connection across to basic physiology. And I don't blame myself because no one else did, either. Now, what's funny about that is it really didn't come into my purview until really close to 2010 or so. And I was fortunate enough as a graduate student to have a gentleman by the name of Jonathan Myers, a legendary physiologist out of the University, actually, of Stanford.

And he came and visited our laboratory, and he gave a wonderful talk about the relationship between VO2 max and mortality. And I was stunned. And I'll give some actual studies later. But you're talking about research and papers that used 10,000 subjects, 100,000 subjects, just massive databases. And they were finding incredibly strong predictions of your VO2 max and how long you're going to live.

And my eyes just exploded. And I went, that's it. Oh my gosh. Being healthy, performing physically at your best is almost the same thing. So now I got really excited about this metric and said, hey, man, I want to know what this stuff looks like. Is Jonathan the only guy that found this out?

Well, learning more about the history of exercise physiology and going back, and I realized we actually had known this since the late 1980s. So there's another legendary physiologist who,

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unfortunately, very recently passed away, named Steven Blair. And he spent the vast majority of his career running these giant studies.

The first one, most iconic one came out in a journal called JAMA, so Journal of American Medical Association, one of the preeminent journals in all of science, in physiology and medicine, in 1989. And in that initial study, he was really the first one that said, hey, when we look at VO2 max and we compare that to, say, smoking or cardiovascular disease, it's as strong, if not a stronger predictor, of how long you're going to live than any of these other metrics.

And then actually, if you look, you will see study after study, and you can pull up meta-analyses. And that has really caught, actually, attention in the lexicon in the last, say, five or so years. People have really jumped on board. And it's really warmed my heart, actually, for that to happen, because I felt like it was something that us in the exercise scientist world, strength and conditioning folks, and again, scientists of exercise have been screaming from the top of our lungs for 20 years, and no one really paid attention to or cared about.

And then people found this stuff out and started talking about it as if it was a new finding. And again, in our world, we're saying, oh my gosh, we've been telling you this for 20-plus years. So that's OK. It's a free pass. I'll give you that. I apologize to you. I'll accept your apology for ignoring us exercise scientists for so long.

But I think it really highlights another theme of this entire show, which is the importance of understanding what maximum performance looks like. If you want to be a better athlete, that's great. It's my personal interest, but that doesn't have to be yours.

But the value that creates to the rest of society is unmatched. VO2 max is one of those examples. I will share with you many, many more of those in other episodes. But that is, to me, one of the best examples of when we stop looking at health and performance differently and start looking at it as, hey, if your physiology performs at the highest level possible, you're going to be healthy, right?

Bill Bowerman-- "If you have a body, you're an athlete." And so I just want your physiology to be functioning at the highest level it can. You can then choose to use those skills however you'd like, to be better at playing golf or basketball or pickleball or riding mountain bikes. I don't really care. Whether you want to have more energy, more recovery, better sleep throughout the day, something like a VO2 max is going to be intricately involved in all of those things.

Now, for those of you that absolutely love numbers, I'll give you some. But please don't get too specific and particular about these couple of studies I'm going to go over. Think of them just as really highlights of the overall field.

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Depending on which population is studied in a certain setting or database, these numbers will vary slightly. But again, this is going to represent what you would generally find across dozens, if not hundreds, of similar studies that looked at VO2 max and overall health and wellness.

A quick point of clarification. When we say fitness, scientifically, we're referring to VO2 max. In the actual strength, conditioning, and performance settings, you might have a different definition of it, and that's absolutely fine. But scientifically, those terms are pretty synonymous. So fitness means we've tested your VO2 max in almost every scientific situation.

So let's start off with that first seminal Steven Blair paper from 1989, in JAMA, in that they had about 10,000 men and about 3,000 women or so. And what's actually interesting about this study and many others like it, they typically follow the individuals for years. I believe, in this actual study, it was something like nine years. And within that, several hundred people actually died.

And so it's a bit morbid, I understand, but it makes the science incredibly compelling, because we can look at a number of people, wait for several of them to die, and then come back and say, what actually was different between those people who died at baseline versus those who didn't die, again, at baseline and after that? And so we can get really strong insights about what predicted death.

Now, what they found in this initial study-- and this is directly from the paper itself-- was after age adjustment-- so again, they would factor in their age and say, let's take that out of the equation. So after age-adjusted, all-cause mortality, meaning died for any reason, declined directly across fitness levels-- so as you reduce your fitness, you increase your all-cause mortality risk.

And it went from a number of what is referred to as 64, so 64 deaths per 10,000 people. That was the highest rate there. It reduced from that to about 18.6. And so again, if you're looking at that saying, all right, if I go from the least fit category to the most fit, my risk goes from 64 deaths per 10,000 people down to 18 deaths per 10,000 people.

If that part is confused, you just run the 18 versus the 64. So another way to think about that is if my risk of dying is 18 and now, all of a sudden, goes up to 64, it's a huge increase in your risk of dying. And nobody wants that.

Similar story for the women. The numbers there actually went from the risk per 10,000 was 39.5 and reduced all the way to 8.5. And so again, clear evidence that this thing was happening.

And what's also interesting here, just because someone will ask, I'm sure, this was true once they factored out things, again, as I mentioned, age but also smoking habits, cholesterol levels, systolic blood pressure, fasting blood glucose levels, parental history of coronary artery disease,

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and then follow-up and other metrics. And so what they're basically saying is even if you take those things into account, you still see this massive reduction in health when we have a reduction in cardiovascular fitness.

Now, I realize following numbers like that is sometimes difficult if you're only listening to this in the audio version. So we will have this paper in the show notes. The actual title of the paper is "Physical fitness and all-cause mortality-- a prospective study of healthy men and women." And again, first author, Steven Blair from 1989.

So if you cruise on to table 2 of that paper, you're going to see that they actually ran the analysis and split up the men and women into quintiles. So this would mean the lowest 20% of fitness, the next 20, next 20, next 20, next 20. So take everyone across the spectrum, lowest to highest. And split them up, top 20%, et cetera, et cetera, all the way down.

And what I will read off to you is the relative risk. And again, this is risk of dying as we go from the most fit 20 percentile to the next most fit, to the middle 20%, to the second to last 20%, all the way to the bottom 20%. That's a way to view this.

So if you start at the highest level of fitness, and we put that as just a number of 1.0. So this is saying, OK, you're at a 1.0. If I go from the top 20 percentile to the next 20 percentile-- so think of this as 60% to 80%, if you will-- my risk goes from 1 to 1.7. So this is a 17% increase in risk.

If I go to the next one down, it's gone from 1 to 1.7 to 1.46. The next after that, 1.36. And then here's where it explodes. So again, think of this as if you are somewhere between the 20th to 40th percentile, 100 being the best, 0 being the absolute worst, so just being the second to last category, your risk is 1.37.

You go from that category to the bottom 20th percentile, so just one category below. Your risk goes from 1.37 to 3.44. And this is why people will highlight you don't have to necessarily be the fittest on the planet from a health and cardiovascular risk perspective, but you cannot be the lowest.

The magnitude of improvement you see from going from the least fit people around to just the second least fit is almost half to three times the risk reduction, so massive improvements. You'll see the exact same thing in the women in the study.

So to not run you through everything, but we're really talking about improvements as you go from the healthiest or most fit, going all the way down to the bottom 20th percentile, that risk factor is 2.42. And then the lowest goes from 2.42 all the way up to 4.65.

So similar message between the men and the women. Just being the bottom of that category is

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incredibly dangerous and problematic for your health. So if you can just do a little bit to bump up one level, that's going to do a lot for you.

Now, again, there's a ton of studies you could pull from here. The numbers, again, I don't want you to be super specific on that because they will differ depending upon the population. A little bit of context on that.

I grabbed another study for you, also in JAMA, far more recently, called the "Association of Cardiorespiratory Fitness With Long-term Mortality Among Adults Undergoing Exercise Treadmill Testing." And this is actually going to tell you a similar story, but wanted to show you how even the studies that are a little bit different are going to have the same take-home message.

In this particular analysis, now they've got over 122,000 patients. So OK, great. Maybe there was something unique about Blair and his little population of 15,000 people or so. What about if we 10x that number roughly? Do we see the same basic results? And the answer is effectively yes.

So in this, almost 14,000 people died throughout the course of this study. So we're getting same kind of idea, pretty healthy people. Some are going to die. But what does it really look like in terms of the folks that stayed alive and those that did not?

I'll zoom you all the way down to the end to not make it so painstaking as the previous one, but similar stuff here. In fact, it's even more jarring because they're able to do more in-depth analysis here of some of those other cofactors, which is what I want to highlight.

So directly from the paper, again, here, the increase in all-cause mortality is associated with reduced cardiorespiratory fitness, which was comparable to or greater than traditional clinical risk factors such as coronary artery disease, smoking, and diabetes.

Now, I am certainly not trying to tell you that as long as you're in shape, that it's OK if you smoke or do anything else. Again, just from this one particular study, really profound there. The cardiovascular fitness, again, VO2 max, was more predictive than traditional risk factors like coronary artery disease, smoking, and diabetes. So I'll put numbers behind this because it gets even more interesting.

The cardiorespiratory fitness is inversely associated with long-term mortality and not observed to be an upper limit. What that also means is there doesn't seem to be any reduction in the benefit by continuing to increase your VO2 max. So in other words, the higher your VO2 max goes, the more it seems to preserve all-cause mortality risk.

So in this study, in fact, you'd see the same thing if you looked at almost any other study in this

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area. There seems to be no upper limit. And so there's just really not a rationale of saying, well, I'm good enough here. I'm OK. This is enough. And if I get any better, it won't really help that much.

You actually do see that in sport performance. So a classic example here is in the sport of mixed martial arts. If you'd examine the VO2 maxes of athletes in that, you would see that it's on an average of about 55 milliliters per kilogram per minute. And if you get past that, the benefits of performance continue to go up but slightly.

And once you really start getting past north of 65, it seems to be really no more association between improved performance. By that, I mean winning fights. Doesn't mean it is detrimental, of course, or not advantageous to be in better fitness prior to a mixed martial arts fight.

But what it's saying the rate of increase in performance against the rate of increase in VO2 max starts to taper off. We don't see a similar thing with cardiovascular health.

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OK, so hopefully, I've made my point here about the importance of VO2 max. But in case I haven't, just one more final study that I thought was of interest here to bring this point home

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even more. Actually, this is one of Jonathan Myer's more recent papers, called "Cardiorespiratory Fitness and Mortality Risk Across the Spectrum of Age, Race, and Sex," published in the last couple of years here.

This is actually in 750,000 US veterans between the ages of 30 and 95. And I like this paper because the sample size is enormous. Again, it takes into account things like race as well as age. Look at that spectrum, almost a 65-year spectrum.

And within that, 175,000 people or so died. So if it held up against 15,000, then it held up against 150,000, and now it's holding up against 750,000, I just don't know how much more evidence one would need to see to believe not only in this as an actual finding, but the relative risk ratio seems to be lining up across all these studies as pretty similar.

And so what they found in this-- again, same idea. They found no reduced benefit of extreme fitness. In other words, the higher the VO2 max, just the higher the risk reduction. There seemed to be no upper limit there.

And in addition, what they found was a couple of metrics. So if you take into account what are called the comorbidities, and so you look at things like diabetes. Diabetes, in this particular study, took their risk factor from 1 to 1.34. So that's a big deal.

However, going from the highest fitness level to the next highest fitness level represented an increased risk of 1.66. So again, I'm not saying that diabetes is OK or anything like that. Again, I'm not a medical doctor. And I don't do really anything with disease.

But just look how staggering this is. And in fact, if you run this all the way out, examples in this paper-- and here, I'm looking at figure 2, by the way, in case you want to go look yourself-- you're talking about the addition of age represented a 1.06 increase in risk factor, hypertension, smoking, arterial fibrillation, cancer. All these things are plotted. And you can see how much they increased risk. In all of those, the highest one was chronic kidney disease, which represented 1.49.

When you look into the VO2 max numbers, the lowest risk factor was 1.39. And then it just escalated from there, to 1.66 to 2.1 to 2.9, again, with the least fit people, having a 4x higher risk of mortality. That's how important your heart is.

And so it's hard for me to make a cogent argument that even as athletes who are interested in, say, again, dunking a basketball or these anaerobic, high-power, low-fatigue sports very difficult to say your heart is not playing a big role in your global health, and that that isn't going to limit your performance somehow.

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So at this point, if I haven't convinced you of the importance of your VO2 max, I don't think I can. So let's go ahead and move on regardless. Now, you're probably interested to know, how do I assess that? How do I value that and then do something about it? And we're going to cover that a little bit later.

I promise I'll give you a full breakdown of how to know whether your VO2 max is good, based on whether you're male or female, your age, and where that puts you in the categories and percentiles. We'll cover all those data and have, of course, plenty of links directly to tables in the show notes.

But I think before we do that, we actually need to talk more about what makes the cardiac tissue so special and unique. I'm going to talk a lot about skeletal muscle in other episodes. And so what I want to do here is really focus on what is unique and special about the muscle fibers in the heart, as this is going to explain a lot about how we interpret it, what we do about it, and how, actually, there's more things to pay attention to than just your VO2 max.

To get us started here, I'd like to actually ask you a question. That is, have you ever thought about why your heart never gets sore? I mean, as I said at the beginning, you've got three types of muscle, right? Smooth muscle, which doesn't have contractile properties, and it's not important or relevant to force production or human movement.

You've got skeletal muscle, which is everything else. It's your arms, legs, neck, shoulders, things like that, and then cardiac muscle, your heart. And you know when you exercise really hard or do something unique and unique novel, train over a larger range of motion, do more eccentric work, and all these other things, your muscles get sore.

But why does your heart ever get sore? If you went out right now and you haven't exercised in years and you ran a VO2 max test, you would get extremely tired. But you would not wake up the next day with a sore heart. Your intercostals or your ribs or your low back or something might be sore, but not your heart.

Well, why is that? Well, actually, the answer to that tells us a lot about how we should assess the functionality of our cardiovascular system as well as how we need to think about training it differently than we train skeletal muscle. You see, it always comes back to physiology, right?

So there's a reason we're going to walk you through how the heart is set up, the structure of the fibers, why it contracts the way it does, because again, this gives us insights into why we need to totally change our mindset about how we're going to train and improve it relative to how we talked about and we'll talk about training our skeletal muscle.

So the heart is made up of really four unique areas. And we call these chambers. It's got two at

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the top, called your atria, your left and right, and two at the bottom, called your ventricles. And really, the idea is you take blood from the atria. You squeeze and contract the atria. That pushes blood into the ventricles.

The ventricles then squeeze, and that pushes blood out of your heart and into your system. There's a lot more detail in there, but that's close enough for now.

Of primary interest is the left ventricle. That's actually the reason why, when you see a heart, it isn't that perfect symmetrical shape that you envision when your five-year-old daughter draws it. It's actually slightly tilted to the left a little bit.

And that's because the left ventricle itself is larger than the right ventricle, primarily because the right ventricle just needs to pump blood to the other side of the heart. But the left ventricle pumps it out of your heart in throughout the entire rest of your body, down to the tip of your toes, and then all the way back up into your heart. So it has to have enough force to have all of that blood moving up against gravity, fighting through muscular contractions to get blood all the way to return.

Now, you have some ways that you can help that blood return along the way. But primarily, that's what the left ventricle has to be able to do. And so because it is asked to have a higher function, in other words, produce more force, it actually is larger.

There is an association at all times between muscle size and muscle strength, though that is not linear. And we'll discuss that in other episodes. And so globally, the left ventricle is larger.

What's also unique about the heart is that the way that the muscle fibers themselves are made up. And so you see your heart, like any muscle, is just a composite of many hundreds, if not thousands, of individual muscle fibers. And we will talk again about the nature of those in the skeletal muscle episodes.

But for now, we need to think that they are actually quite different. And so while you think of muscle, your biceps muscles or hamstrings muscles or quadriceps muscles, they are meant to have specific functionality. A term that we're going to use in muscle science all the time is structure equals function. So the structure, the way that it is built, equals the functionality.

So as a quick example, your hamstring muscles are primarily meant for explosive movements, to run, sprint, jump, stuff like that. And so the way that they are built, the way that they contract and oriented and attached to the bone are different than, say, your spinal erectors, your low back muscles that are meant to just keep you up in vertical all day. They're not really meant to be exploded or contract with a lot of force. They want to be on and contracted mildly to keep you vertical and erect with that nice, great posture.

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When we go to the cardiac side, then we start thinking, OK, what is the actual need and demand of the heart? And so while we want to be able to turn skeletal muscle on and off a lot and to have really specific and precise movement, that's not the role of the heart. In fact, we need to hedge towards something else. We just want the heart to contract.

We don't need it to contract in different ways. We don't need high precision. We need a full contraction. And in fact, more importantly, we need to hedge against the possibility of not having a contraction.

If your hamstrings don't fire appropriately, or you think your glutes are turned off, or they're not as strong as you'd like, that's not going to really change your ability to live. If your heart fails to contract even one time, you have serious problems. If it fails to do that for just a couple of minutes, you're dead.

And so the demand is quite different. It needs to be very consistent, and it needs to basically do the same thing every time. And it needs to have fail-safes. So some problem exists. It can still contract.

And so the nature of the fibers in your heart are quite different. In muscle, they are very, very long. So you'll see them up to 5 to 6 inches in length of a single muscle fiber in, say, your quadriceps.

They are quite short and thick in the heart. The diameter cross-sectional area is roughly the same. You're talking about something like 4,000 to 5,000 micrometers squared in terms of a cross-sectional area. But the length is very, very short. Now, you're talking about something like 0.1 centimeters in length.

And the reason we want that or the reason that actually is happening is because the fibers themselves are what are called single nucleated. And so this differs significantly from skeletal muscle that has thousands of nuclei in the cell. The nuclei, as a quick reminder, are the place in which you hold your DNA.

It is the control center of the cell. It determines how the cell responds to external stimuli, recovers, repairs, goes through protein synthesis, or adds more mitochondria, deletes them, or whatever the case may be. This is being run by the nuclei.

And so by having more of them in skeletal muscle, it allows it to be extremely plastic and adaptable and responsive to exercise or interventions or lack of exercise or anything else going on. I don't need that in cardiac tissue. In fact, I don't need it to be growing and shrinking and dying really quickly. What I needed to be doing is extremely consistent with both its activation, so its contraction, and the force applied in that contraction.

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So the fact that you've got a single nuclei in the cardiac tissue tells you its primary role is not actually adaptation. In fact, depending on the study you look at, you're going to see that the muscle fibers in your heart are going to turn over somewhere between 50% to maybe up to 70% throughout your lifetime, meaning many of the fibers in your heart that you have as a child, especially past puberty, are going to be there the rest of your life. There isn't a huge turnover.

Now, that differs considerably if you look at something like the skin. That's probably going to turn over. You will have all new skin cells every 30 to 50 days or something like that. Red blood cells, maybe more like every 120 days. And skeletal muscle can actually have a lifespan of maybe a decade or something like that, maybe a little bit longer.

But your heart tissue is going to very rarely turn over. It's not meant to be hyperplastic. That does not mean it doesn't respond and adapt and change to stimuli, like high blood pressure, like exercise. It absolutely does. But it happens much slower. That's not the primary job.

So the fibers themselves are shorter. They are nice and thick, and they have a single nuclei. But they have a couple of actual special unique advantages that skeletal muscle does not have.

For example, they are connected to each other through what are called intercalated disks. Now, these are specific and unique to cardiac tissue. And what it actually allows to happen is for there to be what's called gap junctions. So there's almost little entry points from one of the fibers to the next one.

And what that does is it gives the ability for an action potential, which is the electrical voltage that goes into the fiber that causes it to contract. It allows that voltage to leak from one fiber to the next. You wouldn't want this in your skeletal muscle, because that means when you contract one fiber or set of fibers, you might accidentally contract other ones. Not good.

Remember, we want high precision and control of movement in skeletal muscle. With cardiac tissue, we just want it all to go. And so the fact that we have these open gates through these intercalated disks and through these gap junctions, that says, hey, if for some reason, we struggle to get intervention or activation of an action potential, as long as we get it into one of the cells, it'll be able to leak into the rest of them as well. So in this case, we want to hedge guaranteed contraction over control.

Now, in a similar point, if you go to skeletal muscle, it exists in what are called motor units. So you might have several hundred to even many thousands of muscle fibers, all innervated or controlled by one basic nerve is the way to think about that. This allows you, again, to upregulate how many of your muscle fibers in your muscle are contracting at a given time by turning on or off more total motor units.

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The heart doesn't have any. There is no motor unit in the heart. We don't want to have the consequences of what if a nerve fails or is blocked or dies, and now we can't contract those fibers. And so in fact, the heart is not dependent upon nervous system activation to contract.

Now, I'll say that again. The heart does not require any nervous system activation to contract. And this explains exactly why you can do really awesome and interesting things like in the movie Indiana Jones, Temple of Doom, where the gentleman reaches into the guy's heart, and he pulls it directly out of it. And he stares at that man's heart that's in his hand, and it still continues to beat.

This happens because, again, unlike skeletal muscle, which requires nervous system activation, the cardiac tissue does not. It has its own intricate rate and can spontaneously produce the electricity needed to contract independent of the nervous system.

Now, that does not mean the nervous system does not have a role in your heart. It absolutely does, and we're going to talk a lot about that. In fact, it's incredibly important to understand that as a way to monitor global fatigue, readiness, performance, and overall nervous system activation.

Another thing that differentiates the skeletal from the cardiac tissue is how and how long they contract. In skeletal muscle, we actually want the ability to do what's called summation to reach tetany. And so what happens is the muscle fibers in, say, your biceps brachii will contract with that electrical potential. And then actually, almost before it gets all the way back to baseline, it will contract again. And it'll contract again and contract again.

And so those mini contractions start to stack on top of each other or summate. And in fact, if you do that long enough, you can reach what is called full tetany. Think of this as a muscle cramp. So this is the muscle fibers themselves contracting permanently instead of doing this on-off, on-off rhythm.

Cardiac tissue doesn't do that. And I think you could probably imagine why. It would be a very bad thing for you to reach tetany of your heart.

Remember, when we first started talking about the anatomy of the heart, the primary job of the heart is to move blood from the atria, or the top of the heart, to the ventricles in the bottom, and then move that out to the body. So if this thing were to reach tetany, blood wouldn't move anywhere. You wouldn't be able to circulate any blood throughout your body, and of course, you would die.

So while it's OK to have a cramp in your calf and it's painful, and it's annoying, and it's all those things, having a cramp in your heart would be far worse. And so your body hedges against that.

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And what it says is, all right, if I have this extremely fast, what's called refractory time in skeletal muscle-- it's the ability to contract multiple times within a single muscle fiber-- I want to extend the time of contraction in the cardiac tissue so that I don't have that repeat in summation.

So in addition to not wanting tetany, you also need to allow time for blood to fill up the ventricles. Remember this. We're going to come back to this later in the episode when we talk about determinants of VO2 max, what to improve in some of these other numbers, and why that relates to your resting heart rate, your maximum heart rate, why that's not trainable, why there's no difference in maximum heart rate between highly fit people and unfit people, and things like that.

So the ability of your heart to fill back up with blood is critical. So it's got to contract, allow enough time for blood to fill back into the atria or ventricle, and then contract again. So big, long, smooth contractions, not a lot of plasticity in the tissue itself. We want to hedge against having lots of fine motor control. We want consistency over specificity here.

So another way to build on top of that is going back to what I said a second ago of, how does it produce a contraction independent of the nervous system? I gave you, potentially, a little bit crude example from the Indiana Jones movie. But another way to think about this is, how can my heart beat if I'm unconscious, if I've got the brain turned offline?

Well, it will continue to do that because it has this intrinsic rate. You've got four what are called pacemakers in your heart. And the one I want to cover and talk about the most is the SA node. So the sinoatrial node, this is in the right atria. And it controls, for the most part, your heart rate.

Now, you've got other ones like the AV node, purkinje fibers, and bundles of His, and things like that. But those are really backup systems. So in case the SA node fails, it'll go to the next one, go to the next one, and all the way down there.

So we've got various fail-safes that give us the ability to say, all right, if we have a problem, we're still going to get contraction, because remember, all we've got to do is get one chunk to fire, and it'll spread through those gap junctions and get everything else to contract in the appropriate fashion.

And so we want to have that in position. This is also why, if you have something like a heart attack and several of your tissue in your heart die, you can still survive because you can get contraction of everything else. But it complicates the process, because we start to lose electrical impulse through the parts of the tissue that are dead.

Now, the SA node itself is actually a bit of a marvel. You could think about this and actually remember in school being told that we have no idea. It's one of the modern mysteries of the

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world of how the SA node intrinsically develops this space.

Well, that's not exactly true. I think my teacher either didn't know the answer or was just trying to hype me up a little bit. We know a lot more about what controls. In fact, there's a number of things that go into that.

It does have a little bit of wonder. I don't want to steal that. We don't know exactly how or why this thing beats the way it does, why it's similar between almost every human, and how it can spontaneously create these action potentials.

That is regulated by a number of things, including various endocrine or paracrine, these hormones that are circulating in your system, blood pressure, the strength of your contraction, the amount of blood that comes back into your heart called preload and various other factors. So it's actually a fairly complicated milieu that go into it.

I'm actually still OK with you thinking of it as this modern mystery, that it has just this magical property where it contracts and causes electrical stimulation and action potentials out of nothing. I'm cool with that too.

What we do know more about, though, is how this regulates the rest of your body. So when we talk about skeletal muscle, we know, specifically, there's a neurotransmitter called acetylcholine that is required for muscle activation. So the reality of it is your nerves are actually not directly attached to skeletal muscle. There's a little space in between them.

What happens is acetylcholine is on the presynaptic nerve. So this is the nerve that comes in there. It gets released into this little space in between, actually attaches to little ligand gates on the muscle itself. They open up. They let sodium into the tissue, and they cause a whole series of electrical things.

We call this an electrical to a chemical, back to an electrical signal, the way you transfer an electrical signal down your nerves into a chemical signal back into an electrical signal that allows muscle contraction. So once again, acetylcholine is the primary neurotransmitter that excites or activates skeletal muscle.

But shocking enough, if you put acetylcholine on to the heart, it slows it down. Yeah, it does the exact opposite. And so you have a number of nerves that are coming in. Probably the most famous is the vagus nerve. Now, this is a V-A-G-U-S, not a V-E-G-A-S, like the city.

So the vagus nerve and several others are what are known as parasympathetic drivers. And so the autonomic nervous system is split up into two large branches. The first one is the parasympathetic. This is rest and digest. This is relaxed, sleepy, depressed, chilled, all those

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things over there.

The other side of the equation-- and it is more complicated than this. But this is all we need to know for right now-- is the sympathetic. This is fight-or-flight. This is freeze. This is action, anxiety, aware, aroused, and all kinds of things like that. We want both of these. They are critically important for everyday life.

We need these for high performance. We need these for health. We need these to just be alive. And so we want to be able to fluctuate back and forth between these two states appropriately. They are not on/off switch. They are more like a gradient or a toggle. They're a dimmer switch more so than they are, again, flipped on or flipped off.

So what happens is the intrinsic rate of that SA node is probably higher than your resting heart rate. In fact, it probably wants to beat more like 100 to 120 beats per minute. Most people's resting heart rate is more like 60 to 80 beats per minute. So you have this vagus nerve that is constantly applying this drip of acetylcholine to naturally slow your heart rate down.

Now, this is actually a really cool mechanism, because what allows you to do is if you want to increase your heart rate, the very first thing you have to do is not necessarily turn on sympathetic drive. It's just to reduce parasympathetic drive.

Another way to say that is imagine you're driving downhill. Say you're in San Francisco or some place that has a ton of hills. And you're going at 60 miles per hour. And you decide you want to go faster.

Well, the initial instinct is to maybe hit the accelerator or hit the gas. Think of that as the sympathetic nervous system. But you don't actually have to do that. The first step is just to make sure your foot isn't on the brake, the parasympathetic nervous system.

So what's happening is at all times, when you're driving, the vagus nerve is slowly keeping its foot just a little bit on that brake to keep you relaxed. Now, it's doing that again so that if you want to go faster really quickly, all we have to do, rather than giving out additional resources, like epinephrine or adrenaline, all I actually have to do is stop us from slowing you down.

It's one of those classic double negatives, right? So if I inhibit the inhibitor, I can actually go faster. So if I remove my foot from that break, my heart rate will increase to, again, somewhere in that 100 to 120 beats per minute range, plus or minus here, without us doing anything.

If I want to continue to accelerate past that, so now I'm going down that hill. I was going 60 miles an hour. I've removed my foot from the brake. Now I'm going 80 miles an hour or 100 miles an hour. But that's not fast enough. I want to go 150.

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Now I can hit the accelerator. Now I can push down on the sympathetic nervous system, increase adrenaline, turn on faster rate, and pump my heart even more to produce more work, more energy, or whatever I'm trying to accomplish.

Great. Now we've got that down. Let's go back and answer our question. Why doesn't the heart get sore? Well, let's think about it. What are the reasons that cause skeletal muscle to get sore?

Remember, all skeletal muscle with the exception of one-- I wonder if you know which one that is, by the way. All skeletal muscles, with the exception of one, is connected to bone via tendons. And so when we contract muscle, pulls on the connective tissue, pulls on the bone to get you movement.

Our cardiovascular, our heart is not that. It is not connected to bone. That's not the point. We're not trying to cause movement. It is really just connected to itself.

So because of that, we can't ask it to go over any additional range of motion. So that factor gets thrown out. The only thing we can possibly do is put more blood back into the heart, which puts it on an eccentric stretch. That's our only mode here.

Now, eccentric exercise does lead to excessive soreness if done especially heavy or in a novel fashion with traditional exercise. And so eccentric exercise is something to pay attention to. But the fact is we don't have the ability to overload the heart more than the maximum amount of blood we already have in our system. So there's no novelty we can add to it that it's not already used to.

By the way, to answer your question, what's the only muscle not directly attached to bone? I'll give you a hint. You can see it on me right now. And if you were my five-year-old, you would love to show it to me all the time. It's your tongue. Pretty cool, right?

All right, going back to business here. So it's not range of motion. It's not the eccentric training. Other things that cause soreness are higher intensity. Not really applicable here. Again, if you're used to contracting at a maximum heart rate, we're not going to be able to go past that.

More volume, well, we could do that. But more volume tends to mean more exercise over more range of motion. Your heart beats all day. It is not subject to that much change in volume. If you looked at the total amount of heart beats that you go through throughout the day, a little bit of exercise is not changing that volume too much. So it's really difficult to add much volume relative to the standard or baseline there.

And so as you just continue to go down all the other factors that influence muscle soreness, and you see they don't really apply to muscle. Again, that's not his primary role. And so while you

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may get fatigued from exercise, especially endurance-based exercise, the heart itself is not really subject to fatigue.

In fact, the heart rarely gets tired. It has far more mitochondria in it than skeletal muscle. We used to refer to this as the ultimate slow-twitch muscle. It is not meant for force of contraction.

Going back to motor units, we actually can't alter force of contraction in the muscle fibers themselves in the heart. We can only do it by changing the stretch on the tissue. Same thing in your skeletal muscle.

But in that case, you've got both options. Change stretch or strange contractile properties. We really can't change the contractile properties in the heart, especially acutely.

What we can do is put it on more stretch. This means more blood back into the system. Again, preload, we'll talk about that a little bit later. Afterload is another way. But if we put more back into it, we put the muscle on a bigger stretch. And this allows it to then respond.

I'm thinking about a rubber band. If I pull it a little bit, it snaps back. If I pull it a lot, it snaps back harder. That's all we can really do. But it has not meant to be regulating force up and down. We don't even have motor units. It's an all or none thing, ideally.

And so we've got a lot of mitochondria in there. We are phenomenal at aerobic metabolism. Again, specifically, within the contractile properties, we're not talking about aerobic metabolism of your entire system or heart. We're talking about the capillaries surrounding the heart itself, the ability to get blood into the tissue of the heart, not the blood actually in the chambers that you're using to send the rest of your body.

Remember, your heart has its own blood supply, not the stuff it's trying to give out to everybody else. Think of this as Halloween, where you're sitting in a house and you've got this giant bucket of candy. And the candy you're giving out to the rest of the world, you're not eating that candy as well. You've got another supply of candy in your back pocket and in your house, and you're pulling out of that candy, if you will.

OK, so the heart itself is meant to be incredibly robust against fatigue, against damage, against soreness, and against changing any of its inherent contractile-- or ionotropic is what the cardiovascular folks would probably call it-- properties. That said, it does respond somewhat similar to skeletal muscle with exercise adaptations.

So just like in skeletal muscle, where you can add quality-- contraction force and speed and power-- and you can add quantity-- muscle size-- the same thing actually happens in the heart. The heart can get physically stronger. This would result in you pumping out more blood per

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pump.

Again, the fibers themselves won't necessarily change their inert properties, but the heart can contract with more force. We'll talk about that a little bit later. That's going to be referred to as ejection fraction and stroke volume.

It can also get larger. And in this particular case, typically, what you'll see in response to exercise or healthy lifestyle behavioral changes, the enlargement in your heart, you'll see, will be primarily in the left ventricle. Again, this is the one that's going to have to deal with the pressure of the aorta, getting that blood out to the rest of your body.

And what will ideally happen is the amount or size of that chamber, so the inside, the space that can be filled by blood will either stay the same or even get slightly bigger. But what you'll basically do is you'll pack on tissue to the outside of the ventricle. So it gets bigger, allows it to produce more force, but it doesn't compromise the size of the chamber.

So again, think about the left ventricle as a balloon. If that balloon gets smaller and you can fill less blood in it, that's going to be a problem. We don't want it to necessarily be extremely large, either. And so if the back end grows but the chamber size, the balloon size stays the same, then we're going to be able to contract with more force and not compromise our total blood flow.

If you achieved an adaptation like that and it allowed you to pump out more blood per pump, that number is called stroke volume, so the volume of blood that comes out per stroke or per contraction. The percentage of the blood that gets emptied out of that ventricle is called your ejection fraction.

So let's just say there was 100 milliliters of blood in your left ventricle. And you contracted, and 50% or 50 milliliters was left in the ventricular. Your ejection fraction would be 50. And so we would like to see high ejection fractions so that we're not wasting our time contracting and blood still sitting in ventricle.

If you improve either of those things-- and I'm going to really focus mostly on stroke volume here-- that will allow your heart rate to drop. And so one of the classic adaptations we see of any type of physical training, but think more specifically endurance-type training, is a reduction or a drop in resting heart rate.

See, at rest, right now, as you're sitting here listening, the amount of oxygen required is the same whether you're fit or unfit. It doesn't necessarily matter. There's a minimal amount of oxygen based on your body size and other factors that don't really matter your fitness level. And so we call that your cardiac output.

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So what that is, is it's the stroke volume multiplied by your heart rate. So how much is coming out per pump and how many times are you pumping in a given minute? You multiply those together, and you get a cardiac output.

Let's just say that number is 5 liters per minute. It's a very standard resting cardiac output. If you are fit and we improve your stroke volume, since the total demand-- again, the back end of this equation is still 5 liters per minute. But I've increased one of the numbers. That allows me to decrease the other number.

And so your heart rate, as I said, the SA node is paying attention to many things. And one of them is that preload. So how much blood is coming back in and how much is going back out and various other factors?

So if I'm getting, say, this 100 milliliters of blood out per pump, I don't have to pump as often. So increase the acetylcholine drive, slow the heart rate down, and let's chill out. In fact, we'll cover some of these numbers later about what a good heart rate is, what the best we've ever seen, why you don't want to be too high or too low, and stuff like that. But that's basically what's happening.

And so we can identify whether or not we're struggling in either the stroke volume portion, the cardiac output side of the equation, or heart rate, just based on that understanding of how the heart works. That will then tell us what style and type of training we need to do to make the most efficient improvements, and not only our heart rate, but more importantly, our VO2 max.

Now, your heart rate, again, how many beats per minute you're using at rest or during exercise, is incredibly telling, as I mentioned earlier. It doesn't actually change, though, in response to exercise training at its maximum. The only really thing that matters in this particular case is your age. And we know that maximum heart rate goes down as you get older, but it doesn't alter that much with fitness.

And so what it can tell you, though, are things like your heart rate variability. And so let's just use the example of a heart rate of 60 beats per minute. So one would think and assume that if my heart rate is 60 beats per minute, that means I'm having 60 beats in 60 seconds. That would be 1 beat per second.

And if you calculated the total amount of beats you had over the course of the minute, you would in fact, achieve 60. That's what that number means. But it doesn't necessarily mean it's on the exact same rhythm.

So it would not be like on a metronome. Your heart would not be beating every second on the second. There is a variability in the space between heartbeats. So while, again, you would

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achieve the same number by the end of the minute, in this case, 60, it might do two or three fast ones in a row, have a little bit of to pause, have a little bit of to pause, a fast one, five fast ones, et cetera. So there's a variability in that rate.

Now, it's not very long. It's actually so small that you won't even be able to perceive it. But we can measure this with a number of different technologies. This is called heart rate variability. You may have heard of it before. It's been around for over 60 years. And there's extensive evidence and research on this.

Originally, most of the work there, again, came from these disease and health models. HRV has been associated with cardiovascular health, mortality, mental health, depression, anxiety. But more importantly for me was when HRV started coming along for things like athlete readiness, recovery, sleep, and performance.

And so as always the case, physiology is physiology, friends. If it's dysfunctional, it's dysfunctional. If you're leading that to long-term health implications, if that's leading to short-term performance detriments, it's really the same thing, right?

So understanding the role of HRV is something we're going to have to get into a lot later in future episodes. I would love to talk to you more about that. There's a lot of nuance and interesting things we can pull there, but globally, something we want to pay attention to.

So as we go into our next section here, where we cover those three Is-- how do I Investigate my current cardiovascular fitness? How do I Interpret how good that is? And then how do I Intervene? What do I do about it? I want to make the point that just looking at your VO2 max is not enough.

Just looking at your resting heart rate wouldn't be enough. You would also want to pay tremendous attention to your HRV and then various other factors like your respiratory rate. Again, I will do respiratory rate in a future episode. I would love to talk to you for many hours about that.

I would actually tell you right now a little bit of a spoiler alert. I think that is the most underappreciated of all these metrics. I think it should be considered a vital sign and is potentially the most important thing that you can measure for overall fitness and health.

And quite honestly, it's the thing I pay the most attention to on a day-to-day basis of all of these metrics. More on that later. You're going to have to wait for now, though.

I'd like to take a quick break and thank our sponsors. Today's episode is brought to you by Momentous. Momentous makes safe, high-quality supplements. Now, I naturally despise and

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frankly don't trust most supplement companies and for good reason.

In fact, I recently coauthored a review article that was published in 2023, in which we found that the amount of adulterations, which are accidental contaminants or deliberate spiking, like adding stimulants or anabolic or other agents which are not supposed to be in the product, or mislabeling, in which the supplement contains either a far greater or lower concentration than is being reported on the label, is shockingly high.

So I actually honestly spent literally years vetting both Momentous and their leadership team before deciding to officially partner with them last year. Bringing them on as a sponsor for the show was this a natural evolution of our partnership.

Now, I did all that because of their dedication to cleaning up the supplement industry. And I've seen them do that with their pocketbook. First, every single Momentous product is third-party tested, and that's expensive. And many are even further NSF Certified for Sport.

But more unique than that is their vision for the future of where this, frankly, dysfunctional industry should go. For example, they've recently started an initiative they call the Momentous Standard, which I can't wait to tell you more about down the road.

On top of this strong ethical company value, their products are also fantastic. They're used by the best of the best, including countless professional teams, Olympic committees, 31 of the 32 NFL teams, many members of the Department of Defense, and many more.

Now, none of you have to use supplements. And I genuinely hope you never feel pressured to do so if you don't want to. And supplements will never be more important than whole foods. And I've had great success with plenty of athletes and executive clients who do not want to use any supplements for whatever reason.

However, they can be beneficial to some. So if you're going to use them, I strongly encourage choosing a brand like Momentous, which, in my opinion, has the highest safety and quality standards in the world. If you'd like to give Momentous a try, go to livemomentous.com/perform to get 20% off your order. Again, that's livemomentous.com/perform to get 20% off.

Today's episode is also brought to you by LMNT. LMNT is an electrolyte drink mix that has an ideal ratio of sodium, potassium, and magnesium, but has no sugar. Electrolytes are critical to proper hydration, which I've been harping on for years. But you can't do that by only drinking water.

In fact, many of you will probably remember that I featured LMNT in my YouTube series on optimizing hydration all the way back in 2020, which is obviously long before this podcast even

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existed. So I've been a fan of LMNT for a long time.

In fact, astute listeners will also realize these hydration videos were the genesis of the Galpin hydration equation, named not by me, but by the legendary neuroscientist Dr. Andrew Huberman. I featured LMNT in these videos because their blend of 1,000 milligrams of sodium, 200 milligrams of potassium, and 60 milligrams magnesium really is different than any other electrolyte on the market and has great scientific support.

However, I want to be ultra clear. 1,000 milligrams of sodium is a lot, and not everyone needs that much. In fact, that may be way too much if you don't exercise or sweat much, already eat a lot of salt in your diet, say, from a lot of processed foods, or otherwise have sodium-related medical concerns.

I personally use the citrus and watermelon flavors a ton to prehydrate before heading out on a full day of training or a long hike or a hunt since I know I'll be losing a bunch of fluids and won't have many opportunities to take things with me.

LMNT has also just released a new line of canned Sparkling LMNT, which I am all about. If you'd like to try LMNT, you can go to drinklmnt.com/perform to claim a free LMNT sample pack with the purchase of any LMNT drink mix. Again, that's drinklmnt.com/perform to claim a free sample pack.

OK, we're now at the part of our conversation where I can answer the question we started off with, which is, why do you breathe? And we talked about how it's oxygen, and we need that. Well, oxygen is not really a fuel for metabolism. It's needed to go through aerobic metabolism, but the fuel is coming from your fat and carbohydrates.

I only need oxygen during aerobic metabolism, but I'm very effective at anaerobic, which means I can produce energy without the need of oxygen. But to finish those processes, I've got to have the oxygen around. And so it's a little bit of a twist here.

This is also explaining why even if you're an anaerobic athlete, you still care deeply about your aerobic system, because this is what allows you to recover, to completely metabolize your carbohydrates, to finish that process and restore yourself back to homeostasis. The faster you can do that, the faster you can repeat your anaerobic processes. You can recover. You can get back to training, get back to competition. The more you practice, the better you get, the better you perform.

And so what's actually happening is this. When you take a breath in and you inhale, you're bringing in, amongst other things, but primarily, oxygen. When you take a breath out, you're breathing out CO2. The oxygen you bring in is primarily there to regulate metabolic processes.

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But the CO2 you're exhaling is regulating your pH.

Now, there's a handful of things your body will regulate almost anything else. One of them is your pH. It does not like to mess with this. If you were to look at other markers, like, say, your blood glucose, you realize that that's highly variable.

It can be as low as 70 milligrams per deciliter, as high as 150 during exercise or something like that. And so you can see, it will double or maybe even triple the amount in the blood.

You would never do that with pH. It has an extremely tight window that it will not move out of. And that's because all the enzymes that are required for you to go through any metabolic process need to be in a certain pH range. If it gets out of that, it becomes too acidic or too alkalotic. They can't function. You can't create energy. You're going to die very, very quickly. So pH is insanely important to hold into a tight window.

And so what happens is you don't feel that air hunger or that desire to breathe because oxygen starts getting low. Remember, especially at rest or even during exercise, you can produce energy anaerobically. So when you start getting low on oxygen, you'll just switch to anaerobic metabolism. It's not necessarily a reason for you to panic, to stress, or to change your behavior.

Increases in CO2, though, will do that. And so remember, your muscle, whether it's using fat as a fuel or carbohydrates as a fuel, it's trying to generate a molecule called ATP. This is the energy currency in all of biology.

And it doesn't matter what you use that ATP for, by the way. It doesn't matter if we're talking about skeletal muscle, we're talking about cardiac muscle, or anything else. So whether you're using this for exercise, to power your brain to recover, to digest food, it's irrelevant here. We're going to use carbohydrates or fat as a fuel. We're going to make ATP. And then at the end, the final product of all metabolism is going to be water, CO2, and ATP.

So the CO2 concentration increases as metabolic rate increases. As a result of that, you start then moving CO2 from your tissue into the blood. Concentrations of CO2, then, and blood go up. You've got chemoreceptors in your brainstem and various other places that are going to be paying extreme attention to the amount of CO2 in your blood.

If CO2 gets really, really high, we call this hypercapnia. If it gets low, it's called hypocapnia. Remember those terms. So hypercapnia, increases in CO2 concentration, actually signal your red blood cells to drop the oxygen on them, making it easier for your muscle to extract and absorb the oxygen.

Effectively think about it this way. If CO2 is high in the blood, your body is under the assumption

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you're going through a lot of metabolism. So it's under the assumption that we want to use and need a lot of oxygen, so it reduces that affinity. This is called the Bohr effect.

If you get hypocapnic, again, too low of CO2, it does the opposite. Now, this is going to be counterintuitive, when we talk about things like CO2 tolerance and respiratory rate in future episodes, as to why you could potentially have problems with hyperventilation or overbreathing.

So what's happening in this context is those signals are being sent to your brain, and that is interpreting it as saying, CO2 is too high. Let's reduce that. The way you reduce CO2 concentrations in your blood is to exhale.

And so this would cause you to increase your respiratory rate and to start either mildly or excessively hyperventilating. And this is why, as you exercise, your respiratory rate, again, the amount of breaths you're taking, goes up. It is in part to increase and bring in oxygen, of course. But when we're doing it anaerobically, we're not using oxygen anyways.

So the real reason we're breathing so hard and we're panting and all that stuff as we're getting harder and harder of our exercise is because we're trying to dump and get rid of all that CO2 buildup. Remember, excess CO2 is altering pH. This is making us more acidic. This becomes an extreme problem.

So another way to think about this is when you inhale, that's actually a sympathetic driver. And so your heart rate increases during inhalation. When you exhale, it is parasympathetic, and it drops. So effectively, what's happening is your body is saying, oh, you're inhaling. We're assuming then you're bringing in oxygen. Let's get prepared to deliver this oxygen throughout the system.

When you're exhaling, it's the opposite. I don't want to be in a situation where I'm hyperventilating. I don't need to be breathing too much because if, again, that CO2 gets too low, instead of being acidic, we are now in respiratory alkalosis, so the opposite direction. We're too basic. And so it slows the heart rate down.

So every time you take a breath in, your heart rate jumps up a little bit. Every time you take a breath out, it goes down a little bit. So if I'm altering my respiratory rate, I'm then altering my heart rate. And this is why things like HRV are so intrinsically tied to things like respiratory rate.

I can't let us move off this point without saying one final thing. I know we want to get to our three Is here in one second. But a lot of people are aware. And in the coaching world, people use HRV very often. And there's a lot of data to support this. There's a lot of critical information we can get for assessing, say, exercise volume, fatigue readiness, and things like that. Tons of value there.

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But I don't think enough people are paying attention to respiratory rate. This is really highlighted in a paper that just came out in the last few months. And so I'd like to bring this to your attention.

What they did is looked at college age students, and they simply measured their respiratory rate. And one of the things that they found that's interesting is for every breath per minute that increase-- so if a respiratory rate went from 15 breaths per minute to 16 breaths per minute-- they increased their likelihood of experiencing stress by 1.25x.

And what I found particularly interesting about this is they found that irrespective of changes in things like HRV, total hours of sleep, sleep efficiency, sleep onset, and various other things that are typically the metrics used to measure overall stress and autonomic nervous system functionality and things like that.

And so what we're going over here is not to say that HRV or sleep are not good metrics to take. They clearly are. It's just that you're going to find things in the respiratory rate that you're not necessarily going to see in other places that give you great clues about overall stress. So strongly encourage you to pay attention to respiratory rate. And we'll talk about that plenty in the future.

So at this point, we've now got a much better understanding of why our cardiovascular system matters to both performance and health. We know a little bit more about how it contracts and some of the unique properties that exist within it, that differentiates it from some of our other tissue, like skeletal muscle. And then, of course, we've learned why we breathe and how that relegates many other functions like our sleep, recovery, and of course, overall performance.

Using all that, we can now discuss the three Is, which are, how do I Investigate, how do I Interpret, and then how do I Intervene on improving my cardiovascular fitness? Let's start with the first I, Investigate. Now, depending on the metric you're interested in, something like a heart rate can be done with no technology whatsoever. You can simply put your fingers up to your neck, count your heart rate, divide that by the time domain, and get your heart rate. Easy example there.

Most classic one we teach is start a stopwatch. Count how many times your heart beats in 15 seconds. Multiply that by 4. And then you'll understand how many beats you're taking per minute. You could also simply just measure it for 1 minute, count those numbers, and that's fine. But in reality, most of you probably have some sort of fitness technology, app, or watch or something of that sort that's going to tell you that number already.

As far as things like HRV and respiratory rate, we're going to have to cover those in their own individual episodes, as you've got a lot of options, and there's some context there. I will tell you,

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again, most fitness technologies will give you some insight of that, whether directly on the app or something you can get if you dive in to the data little bit further.

HRV is really challenging, though, because there's a lot of ways to measure. It's not standardized, and there's just a lot more context we have time to get into right now. So unfortunately, we're going to have to take a little bit of a pass on that.

Respiratory rate is actually quite simple. You probably want to focus at least initially on respiratory rate overnight rather than during the day, but both are acceptable as well. And again, probably already coming in, any of the technologies that you may be using to track your sleep, recovery, or performance or anything else.

So I'd like to focus most of our attention here on the cardiovascular stuff that we spent most of our time in our conversation with and get into some of those details right now. The gold standard to measure your VO2 max is going to be in a laboratory with a metabolic cart. You can get this stuff in various equations. You can use any of your fitness technologies.

I will strongly encourage you, though, that if you care about this number, if it's possible, and it may not be, spend a couple hundred dollars and get this actually tested in a laboratory. The data are quite clear at this point. I have not yet seen really any standard over-the-counter fitness technology that gets an accurate number of your VO2 max when the number gets high, and particularly for people who are already fairly fit.

If your VO2 max is really low, it might give you a decent number. But for folks that are moderate to highly trained, it's just really challenging to get an appropriate estimate from a watch or a ring or things like that. Perhaps those will improve in the future. In fact, I quite expect them to. But as of now, the margin of error is a little bit too high for me to be comfortable with when you care about accuracy.

If you're trying to just get a global sense, they're fine. In fact, if you want to do that, you can use any number of absolutely free estimate equations. Examples of this would include something like a two-minute step test where you would take your heart rate, step up and down on a very small box, say, 12 inches or so.

You do that continuously for two minutes and then measure your heart rate at the end. You can enter that score into an equation and get an estimate of your VO2 max. Those are scientifically validated methodologies.

The 12-minute test ends up being something like a mile and a half run. So in fact, you could do this if you'd like as well. Run a mile and a half as fast as you can. Take that time, enter it into equation, and get an estimate.

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If you have that time as well as your heart rate, you can enter it in as well and get a more accurate picture. And so again, all of these are close. We call these submaximal estimates because they are that. They are not the direct measure. So I would encourage you, again, if at all possible, to actually go into a laboratory and get this measured.

In addition, if you do something like that, you can get a bunch of other metrics you can't get with some of these estimate equations, like what percentage of fat versus carbohydrate you're using, your anaerobic and lactate thresholds, your maximal ventilation, so how much total air you can bring in and out, and a bunch of other stuff that we talked about on the show and we'll get into in a second.

Again, if you don't have access to any of that, that's fine. Use any of those other free or extremely low-cost options, and you'll get yourself pretty close.

Our next I is Interpretation. So sticking with heart rate and VO2 max, as I mentioned a little bit earlier, resting heart rate will go down as you improve fitness. But your maximum heart rate will not really change. In fact, if anything, it will be reduced because you're a little bit older.

And so to state it one more time, there's no real association between highly fit people and their maximum heart rate and unfit people. So it's not a metric that we should be overly concerned about of, where is your maximum heart rate? It is relevant to, again, your stroke volume. But the heart rate itself is not going to tell you that much. So not something to be concerned about.

Your resting heart rate, though, or your heart rate at any given intensity is very important. So if you're going to be running, say, at a standard pace, say 7 miles per hour, and your heart rate at 7 miles per hour was 150 beats per minute. And now, a couple of months later, your heart rate at the exact same speed is now 115 beats per minute.

That would represent a significant improvement in cardiovascular fitness. Stroke volume would be much higher. And because of that, that allows your heart rate to come down.

Resting heart rate is a similar story. Giving you a little bit of context here-- and the reason this is top of mind is because there's a hummingbird that lives in my backyard. And my wife and kids look at it every single day, and they get so excited. They call her mom's little helper. They named it Squirt, actually. And so they get really excited when this little hummingbird is flying back there because it's super fast.

Hummingbirds have a resting heart rate of something like 1,200 beats per minute. It's absolutely insane. So you're talking about putting an order of magnitude on top of almost a human maximum heart rate. You contrast that to a larger animal like a giraffe, an elephant, or a blue whale, and you're talking about a heart rate of something like 5 to 6 beats per minute.

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And so humans, of course, are somewhere in between. If you were technically to google this, you might see something like a normal resting heart rate is 60 to 100 beats per minute. I am here to tell you, I cannot fathom a situation in which somebody's resting heart rate is over 80 beats per minute, and they are healthy. And I absolutely would not think that that would happen as somebody who is performing at their maximum.

In fact, I even will tell you-- this is off the record. This is not the science. This is me and my professional opinion-- even a resting heart rate of 60 or so, particularly in a man, that's catching my eye. I would like to see most folks probably in the 40s to 50, somewhere in the range. Again, you might be fine at 60, but getting much above 60 is quite a bit high.

I can also personally tell you I've worked with plenty of athletes, specifically in this case, several UFC fighters, whose resting heart rates were in the low 40s, if not high 30s, so 37, 38, 40, things like this. And so that's the lowest I have personally seen.

That said, there are classic stories of plenty of endurance athletes-- you're talking about elite cyclists and cross-country skiers and marathon runners and such-- who were in the low 30s. The lowest I'm aware of is the legendary cyclist Miguel Induráin having a resting heart rate of 28 beats per minute. As far as I can tell, that's the lowest ever reported in the scientific literature, though, please, if you've seen any lower, let me know.

I'm sure there are plenty of stories, of anecdotes of people and personal training records and stuff who think they're lower. But if you've ever seen anything verified scientifically, I would love to see that.

Interpreting your VO2 max is more interesting, in my opinion, because there's a lot of components to it. And so in order to truly understand this, let's talk about how we calculate VO2 max to begin with. The easiest way, in my opinion, is to think about VO2 max equals your cardiac output multiplied by what's called your A-VO2 difference.

Now, as I've stated, cardiac output is simply your stroke volume multiplied by your heart rate. So if we were to combine this entire thing, we would say, your heart rate multiplied by your stroke volume, which says, OK, how much blood am I getting out per pump? How many pumps am I getting?

And I multiply that by what's called your A-VO2 difference. Now, the A stands for Arterial. V stands for Venous, and the O2 is oxygen. So what literally this means is, what's the difference in oxygen concentrations between the arterial side and the venous side? Remember, arteries generally go away from your heart, which means they're going to exercising tissue, and veins come back.

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And so what we're really looking at is saying, OK, how much oxygen is in the blood when it leaves the heart? This is going to be the highest concentration of oxygen possible. And how much is in the blood when it comes back, having passed through muscle? This then directly tells you how much oxygen your tissue extracted in the process.

So to give you some numbers here to make this easy. These are not accurate, just representing the math here. If you had 100 molecules of oxygen that left your heart and went into your quadriceps, and then once it's gone through the capillaries that surround all the muscles and tissues and fibers in your quadriceps, and it went in as 100, and then it came back out the other side and went back to the heart and lungs to be oxygenated, if it went in at 100 and came out as 75, the difference between the arterial and the venous side is 100 minus 75, which would give you a score of 25.

Now, what that means-- and again, I'm using those round numbers to make this simple-- you've extracted now 25% of the oxygen that came into your system. You only got 25% of it out. That's not a very good score. You want that number because it's a multiplier to be as high as possible. So if 100 goes in, I don't want 75 coming out. I want 0 coming out.

Let's say maybe you got 10 out. So now 100 went in. 10 came out. You extracted 90% of the oxygen that was available to you, to bring it into muscle and use it for everything we talked about earlier. And so now your A-VO2 difference is 90 much higher than 25. So we get to multiply that by our cardiac output, which brings our VO2 max even higher.

The easiest way to think about VO2 max is to use what's called the Fick equation. VO2 max, according to the Fick equation, is your heart rate, in other words, how many beats per minute, multiplied by your stroke volume, how much blood coming out per pump, multiplied by what's called your A-VO2 difference.

To be perfectly honest with you, the A-VO2 difference numbers are difficult to convey over audio only. So I'm going to spend most of our time on the other side of the equation. When it comes time to interpreting your VO2 max numbers, there's a lot of charts and papers you can use. We'll provide some of those in the show notes for you. I want to give you some numbers, though, to give you rough context.

Typically, we think about VO2 max in what's called a relative term. Now, scientists and people that are more advanced in this field might like to use the absolute versions. Depending on the scenario, that might make sense. But for now, let's just stick to the relative.

What that means is, how many milliliters of liters of oxygen are you using per kilogram of body weight per minute? So you'll see them expressed as things like your VO2 max is 50 milliliters per

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kilogram per minute, so 50 milliliters of oxygen per kilogram of body weight per minute.

You can think about VO2 max almost on a scale of 0 to 100. And so the average person that would, say, walk out of my classroom, somewhat moderately trained, male or female, in the neighborhood of 170 pounds or 70 kilos, something like that is probably going to have a VO2 max around 35 to 45 milliliters per kilogram per minute, something in that range.

If you fall below 18 milliliters per kilogram per minute, you're probably crossing below the threshold of what we call independence. For women, that's about 15 or 16, which means it's very difficult to live independently and by yourself because your fitness is so low. Going through basic activities of daily living become challenging.

So what I'd like to share with you is not only the normative values, but also the highest we've ever seen in terms of VO2 max, cardiac output, and stroke volume. Now, there's so much data on VO2 max, we can actually break this down by age and by sex in, really, specific numbers. So if you know your exact age, you can go ahead and look these up in these charts. But I'll give you a couple, again, just to give you ballpark.

Let's say you were a female between 40 and 49 years old, and your VO2 max was 28. That would put you as what we consider to be below average. If you wanted to go from below average to average, you'd have to go into the 32 to 36 milliliters per kilogram range.

If you wanted to go all the way to elite, tell me what's the best ever, you'd be needing to surpass the mark of 47 milliliters per kilogram per minute. Any of you that are maybe older, let's go ahead and jump way down.

Let's say you are 71 years old, again, talking about a 71-year-old female here, below 18. You're still above that line of independence, but it would still be considered low. You would really want to be looking at something like 22 to 24 to be considered above average, and then really over 36 to be considered lead.

Now, I know my friend Peter Attia likes to tell people he wants them to be considered elite in at least the decade before their actual age, if not two decades before. And that's a phenomenal way to think about it. So just as that example, if you were that incredibly ambitious and vigorous 71-year-old, elite for you would be 36 milliliters per kilogram per minute.

The decade before, again, 60 to 69, that would be 40. But if you wanted to be that extra person and get that double gold stamp from Peter, you'd want to be above 46. So the difference is going from 36 to 46 would give you that elite category for two decades younger than you.

For the men, it's a similar story. You just add a little bit of the numbers to it. So a male who is,

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say, between 50 and 59 years old, above average would be 36 to 40 milliliters per kilogram per minute. And you'd need to be above 50 to be considered elite at that age.

Now, in my personal opinion, I like to say there's no excuse to be under 50 unless you're over 50, meaning you have no reason to be having a VO2 max of lower than 50 unless you're over 50 years old. And even then 50 to 60, I don't want you anywhere near below 50. So that's a nice number to go after.

We'll also talk about in a second how changeable that is and how much it responds to various types of training. And so it's going to give you a little bit of hope. You have some room to move there, and it will respond to your training.

And so there's some light at the end of the tunnel if you're looking at those numbers. And you've had a VO2 max done recently. And you're thinking, oh my gosh, I am way below what Andy said there. That's fine. You still have within your capabilities to change that.

Now, while I said that those are technically elite and would probably put you in the 99th percentile, they aren't necessarily the highest we've ever seen. In fact, it is very common to be much, much higher than the scores I just described.

So as always, I'd love to share with you what the best in the world are. It's important for us to reset our standards and to recognize and challenge what we think is possible and to not accept just being in the 99th percentile. Let's see what is absolutely possible in human physiology. And those numbers and scores go far higher than that 55 and 60 I just described.

So for many, many years in history, the highest documented VO2 max that we would acknowledge scientifically was from an Austrian cross-country skier. Now, this study was actually published about four years prior to him then winning a gold medal in the Olympics. And so he was obviously a very high profile and highly successful athlete.

He came in-- if you know these values. If not, that's OK-- with a hemoglobin concentration above around 16.8, which is outrageous. Most folks are going to be 14 or 15 or something like that. Hemoglobin is the molecule that carries oxygen around in your red blood cells. So innately, he's got a huge ability to do that.

His VO2 max was reported to be 90.6 milliliters per kilogram per minute, which is incredible. So that actually stood around for a very long time. And people thought that that's basically it. Again, there had been talks of people in the 92s, 93s, and stuff like that, but nothing had really been independently verified. Whether you think that's important or not, hard to know, right?

Until a few years later-- and this is one of the most miraculous and coolest stories I ever

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remember being a part of. In fact, I remember when this happened live, and it was fun for me getting prepared for the show to go back and read the updates on this individual, because it was such a stark and massive change.

It was one of those LeBron James or Tiger Woods moments for the endurance and exercise physiology worlds. The guy that came on the scene was an absolute phenom.

What I'm talking about, of course, is the story of the legendary Norwegian cyclist Oskar Svendsen. Oskar came on as an 18-year-old. And I remember hearing these murmurs coming, and people were saying, some kid, some 18-year-old just hit 100 on a VO2 max score. And everyone was, like, no way.

Again, we've heard all these stories before, may or may not be true. And everyone was basically, like, OK, prove it. Prove it. You've got to do it in a verified way. We got to send some independent scientists out there.

In fact, they sent the manufacturers for the metabolic cart company out to the facility that says, you need to be here. You need to verify this thing is accurate. Nobody really believed it. Not the first time we had heard stories like that.

And so of course, actually, the team out there that was coaching him connected with a guy named Mike Joyner. Mike is a legendary exercise physiologist at the Mayo Clinic, one of these people, actually, who's probably potentially published more in this area, did a lot when we were considering, in the early 2000, whether or not a two-hour marathon was even physiologically possible. Mike did a lot of these calculations, so really involved in the field.

They connected with Mike. They published this paper. And it turns out that they were actually able to verify that Oskar was able to hit a VO2 max of 96.7 milliliters per kilogram per minute. If you're more familiar with absolute terms, that would be an absolute of 7.4 liters per minute, just phenomenal, phenomenal record.

What's also really interesting about this story is he actually retired just a few years later, at the age of 21 or 22, I think. He had some success in competitive cycling, but perhaps not as much as one would think, given his VO2 max was so extraordinary.

I think this is also an interesting message, right? It tells you this is one of the reasons why we love sport. It's just because you have some physiological parameter. You're tall or have some skill. That doesn't necessarily mean you're always going to win. Endurance events are based on more than just VO2 max. And sports in general have a lot of things going on in them besides just physiology.

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I love physiology. I'm obsessed with it. But it's also why I love watching sports because you never know what's actually going to happen.

The world record for VO2 max for females is also phenomenally impressive. This belongs to, of course, the iconic Paula Radcliffe. She had a VO2 max, reportedly, of about 75 milliliters per kilogram per minute.

If you're unfamiliar with Paula, multiple-time world record holder in the marathon. Again, iconic is maybe even not enough to describe how successful and talented Paula was. She reportedly ran somewhere in the distance of 140 to 160 miles per week.

And this is actually really cool because this is a document that you can go look up. She had a very well-respected and known exercise physiologist, Andy Jones. He was actually one of the gentlemen, probably, most responsible for bringing beetroot juice and arginine and things like that onto the scene from a supplementation and nutrition perspective.

But Andy worked with her for, I believe, almost 20 years and was able to document her training and her performance and metrics and things like that, so was able to take her through a bunch of world records, and so has this stuff as something you can go download and take a look at.

Before we move into our final category of intervention, I think it's important to give you some context. I realize many of you are probably familiar with a vertical jump test, a 40-yard dash, maybe your one rep max bench press. Those numbers make sense. Perhaps these ones, they have less context for you. It's hard to grasp how impressive they are outside of seeing how much bigger or higher these numbers are for world champions relative to the rest of us. The numbers don't make a lot of sense.

So what I've done is I've made a couple of quick calculations here to give you a little bit of context of what it means physiologically to have a VO2 max this high, what that means in terms of how much blood you're pumping throughout your system, how efficient your muscles are at getting that in. And so just as a couple of examples, I want hit you with some fun numbers here.

As we talked about earlier, a standard cardiac output at rest is something like 5 liters per minute. And remember, cardiac output is heart rate multiplied by stroke volume. So if we assume a resting heart rate of, say, 60 beats per minute, and we wanted to get to that number of call it 5 liters per minute, that would mean your stroke volume would need to be somewhere in the area of about 80 to 90 milliliters.

Now, for some of you, depending on where you're at, milliliters make complete sense. Those here in America, maybe not. And so I've converted that. That's something in the neighborhood of just under 3 ounces. And so while you're sitting here resting, every time your heart is beating, it's

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kicking out about 3 ounces of blood every time.

Watch how high this number gets when we get to maximal exercise. In the case of these phenomenal athletes like Paula or Oskar, we don't actually know their stroke volume. But we can run some quick calculations and get a pretty close estimate.

Oskar would have had to be in the neighborhood of about 225 milliliters at his max to reach the cardiac output of around 40 to 45 liters, to give him a VO2 max in the 100 or so milliliters per kilogram per minute. Again, I know I'm moving from liters to milliliters, so run the math yourself if you want to challenge that number.

On the back end, as I said, A-VO2 difference is really complicated. So most people are probably in the neighborhood of about 70% extraction rate. So of all the oxygen going into tissue, they're able to get about 70 of it.

Higher trained athletes, though, are looking something more like 93%, 94%, 95%. And so the ability to extract, get it into tissue is just far higher than the average person in that 70% to 80% range.

Coming backwards into stroke volume. If we assume that he or she is in that neighborhood of 200 to 225 milliliters or so, this is what that math would look like. So if we'd said Oskar was-- call him 20 years old to make math a little bit easier. His predicted maximum heart rate would be about 200 beats per minute.

If you're not familiar with that equation, if you take 220, subtract your age, that gives you a very rough-- and please, this is just a rough estimate of your maximum heart rate. But a maximum heart rate of 200 beats per minute is maybe a little higher than what you would really see, but not out of the question.

So if we took 200 and multiplied that by 225, that stroke volume, so heart rate multiplied by the stroke volume, that would put us right near that 45 liters per minute mark. Again, that's the highest I've ever seen in terms of cardiac output. Perhaps there's some conditioning and endurance coaches out there that have seen higher. But that's phenomenally high.

In fact, again, many of the best-performing endurance athletes ever are still in 40, 42 liters per minute. And so Oskar would have had to, again, be able to get at least up to 200 beats per minute. And if not, if he could only, say, get to 190, his stroke volume would have had to be even higher than 225.

What that functionally represents, for those not familiar with the metric system, 45 liters per minute is just under 12 gallons of blood pumped per minute throughout your body. I'll say that

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one more time-- 12 gallons of blood being pumped throughout your entire body every single minute.

Another way to think about that. That 225 milliliters is around 7.5, 7.6 ounces. So if we go back to earlier, remember when I said you're kicking out, something like under 3 ounces per contraction, now you've overdoubled that number.

So you're giving half a bottle of water out per pump. But you're pumping three times per second. Remember, your heart rate is no longer at 60 beats per minute as in one beat per second. It's over triple that.

So you're beating 3.3 or so times per minute. So you're not getting out 7.6 ounces per second. You're getting that out per pump. But what you're really getting at is closer to 25 or 26 ounces per second.

A general water bottle is 12 to 16 ounces. A large one is 20. You're doing that entire thing every second that you're exercising and that your heart is beating. If you were then to extend that throughout the entire minute, it would mean you've pumped over 1,500 ounces in a single minute through your heart.

I don't know if and how any of that information actually helps your life. But me personally find it just endlessly fascinating to think about not only the performance side of this equation. How fast can I run a marathon and things like that? And that's really awesome and cool.

But what's the physiology behind it? What does my body have to do to enable something like that to occur? And thinking about the fact that, man, I'm going to have to pump 12 gallons of blood through my body per minute to be able to execute on something like that, to me, that's maybe even a bigger joy than seeing somebody perform a race at a certain time. Both equally impressive and fun, but love to see the physiology behind that.

To round this entire story out, let's move on to our third and final I, which is Intervention. In other words, what can you do about these things? How much do they change? And what do I have to do to see improvements in them?

If we work backwards through this VO2 max equation, as we've talked about, can we see improvements in our A-VO2 difference? Absolutely. How so? We're primarily looking for a couple of things.

One, increase in capillarization, so the amount of capillaries in our exercising muscle, and/or some sort of combination of improved mitochondrial size or content. If you do those things, you'll be better at extracting the oxygen that's coming in into the tissue as well as utilizing it to go

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through aerobic and anaerobic recovery metabolism.

Back off of that, we now have stroke volume. And in fact, one of the things that makes this interesting is as we go towards maximal exercise and start improving our stroke volume, we start to run into a little bit of a problem. You see, if our heart rate is too high, we don't have enough time to fill the ventricles and the arteries back up with blood. And so we start actually reducing our stroke volume.

And so this is one of the reasons why you would not actually want to have your heart rate continue to increase as a training adaptation. It's now at the point, somewhere around 200 beats per minute or so, where it's compromising what's called filling time. If you don't have enough time there, we can't get enough blood in.

So while you have extremely strong tissue and you can pump a lot of blood out of there per pump, your ejection fraction is massive. You're getting all the blood in the left ventricle out of there every single time you contract. You've got to have some physical time to actually fill it up. And so you will see adaptations in the heart tissue itself.

In fact, if you look at the actual size of the left ventricle, an average number to think about there is 150 grams or so in a nonathlete, where maybe upwards of 200 in an athlete is something that we see respond and is generally associated as a positive adaptation to exercise. And so we know we need to increase the strength of the left ventricle as a starting place. If we do that, that will allow or actually produce and result in an increase in stroke volume.

So what does that mean for training? Well, fundamentally, outside of things like exercise technique and timing and nutrition and all that other stuff, if we're just talking about the background physiology, we have two avenues or areas to push on to improve our VO2 max. We have our stroke volume and our A-VO2 difference.

So there's a lot of ways we go about improving both of them. I am of the opinion that you need to train across a wide spectrum of exercise intensities to optimize both factors. If you, in fact, look at classic training logs of endurance athletes, going back to even what we know about Oskar's training, they are typically going to spend something like 70% or so of their time at a low intensity.

What does that mean exactly? Hmm, it depends on the athlete. But you're probably talking about something like between 60% to 80%, maybe up to 82% of their heart rate peak. Most of their time is there. I'll explain why in a second.

Then you've got another additional, maybe, 20% to 25% of your time being spent at a moderate intensity, typically something like, again, 82% to 90% or so of your heart rate peak and then 3%

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to maybe 6% of the time at the remaining higher heart rate. So this is 92%, 93% or so plus.

The reason I'm giving you rough guidelines there is every scientific paper has those zones, if you will, a little bit differently. All kinds of different endurance coaches historically have set different landmarks. And so there's no exact numbers there.

And so as a very rough guideline, I think it is very safe to assume some split like that should be highly effective at improving your VO2 max. What does that mean in terms of exercises? Well, actually, it's entirely up to you. VO2 max is, remember, dependent upon how many milliliters of oxygen per kilogram of muscle per minute, which means the more muscles you utilize, the higher the VO2 max is.

If you were to go to get a VO2 max test done, and let's say you were not specifically trained on a bike, if you were to get that same exact test done on a bicycle versus a treadmill, where you're running versus cycling, the score on the treadmill is going to be about 10% or so higher than it is on the bike. And that's simply because there's a small increase in the amount of muscles involved when it comes to running versus cycling.

Now, if you are specifically trained on the bike and you cycle a lot, that may not actually be the case. And in fact, highly endurance trained cyclists, rather, will score higher on a VO2 max test on the bike than they will on a treadmill. But that really is now coming down to test specificity, efficiency, and other things that are-- that's not what we're trying to talk about here. And so generally, the more muscles involved, the more oxygen being utilized, the higher that VO2 max.

So when it comes to training, we want to think about the same thing. The exercise mode, I don't want to say it doesn't matter. It is relevant, but you have unlimited options. If you want a bike or swim or cycle or row, that's great.

If you don't like any of those traditional modalities and you want to use something like an assault bike or pull a sled, run uphill, drag something, those are also incredibly viable options. It's not the exercise per se that determines the adaptation. It's the application of the exercise.

The body works and physiology works on a principle called the SAID principle, S-A-I-D, which stands for Specific Adaptation to Imposed Demand. So you put a demand on a tissue to bring in and utilize oxygen at a high rate. It will adapt to that specific demand.

So challenging your muscles continuously to bring in and utilize oxygen at a rapid rate is all fundamentally that needs to happen for you to improve that. And so again, the mode of the exercise is not that big of a deal. If you are new to exercise, I would generally recommend you being careful of exercises that involve a lot of eccentric action, so jumping and landing, because you're going to get really sore really fast.

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But if not, feel free to choose whatever exercise modality or combination of them. Switch it up a little bit. Do some cycling. Do some running uphill. Jump in the pool. Really up to you. The intensity at which you do that is more like what I just explained.

As I apologize at the beginning of the program, I, earlier in my life, grossly underappreciated the cardiovascular system as a whole. And I certainly underappreciated the importance of low-intensity exercise.

I'll also be candid with you here. I am not as fond of zone 2 exercise as some other folks are. I certainly don't think it's bad. It is good for you. I just don't think you need to be that worried about what exact zone you're in.

You want to be something probably in that lower intensity, 60% to 80%-ish of your heart rate. I don't really care where your millimoles are. In any of those low intensities, you're going to be challenging the ability to bring in and utilize oxygen over a long period of time.

Look at any amount of research on that. It is very clear. Steady-state, lower-intensity exercise, especially over time, six months to a year, is generally going to improve VO2 max, probably upwards of 5% to 10% effective, depending on the person, the training history, and other contexts like that. So it's very, very effective and something I have absolutely incorporated more and more into both my life personally as well as my coaching practice. So really important to do that stuff.

On the other end of the equation, you can do things at an extremely high intensity for a short bout. Depending on the study you want to pull here, you can see things like high-intensity intervals. This could be a combination of 30 seconds of maximal exercise, resting 30 seconds, and repeating that anywhere between 4 and 12 times can equally improve VO2 max, if not greater, and more so than your steady-state exercise.

There's a lot more context that go into that. It's not necessarily meaning high intensity is better. There are some significant downsides and concerns with only doing high-intensity exercise, another thing I've changed my opinion on. And so I think we want to use high-intensity exercise. There's clear benefit there.

It's fundamentally different, though, than low-intensity exercise. So we're challenging a different part of the system, which is why I am going to argue you should be incorporating both most of the time. Doesn't have to be always in all of your training, but you wouldn't want to leave either one of these things entirely off if the pure goal here is to maximize VO2.

The reason is when you do something at a higher intensity, the point of failure in the tissue becomes different. So extending my ability to move at a lower or moderate intensity for a long

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period of time is challenging different aspects than it is when I ask it to introduce a tremendous amount of fatigue.

So I'm now into anaerobic metabolism when I'm going really hard and really fast. I can't use oxygen. So I'm building up a ton of byproducts. pH is being disturbed. Potential damage is happening. Other things are occurring. CO2 is getting extremely high.

And so enhancing my ability to deal with that is a similar thing in terms of increasing mitochondrial biogenesis. So more mitochondria, higher functioning mitochondria, larger mitochondria, increasing aerobic capacity, all of these same things occur. And so again, I don't want to make the argument that one higher intensity or low intensity is better than another. I think you should do both.

I will make the same argument for moderate intensity. While that isn't as specific and precise in terms of what it's challenging, it's reasonable to build some of that into your equation as well. Another thing you're going to find commonly in the research is a longer bout of intervals. This is described in a lot of different ways.

A good friend of mine and an expert in endurance physiology, Joel Jamieson, has talked a lot about High-Intensity Continuous Training, HICT. If you're not familiar with that stuff, I would encourage you to look it up. It's very, very effective. Lots of different things and tools we can pull out here.

One example would be something like what a classic runner would do is something more like one mile repeats. So run a mile as fast as you can. This is going to take most folks 6 to 8 or 9 minutes or so. However long it takes you to run that mile, rest that same amount of time. So it's a 1 to 1 work-to-rest ratio, so 6 minutes of running, 6 minutes of rest.

And then you repeat that again for a total of two or three or perhaps four repetitions. That's a very long workout. The average person would not be able to do that.

But those of you that are not average, that are good to high to strong performers listening right now, that's absolutely within your capabilities. And in fact, you've probably done it before. It doesn't have to be that extreme.

You could use shorter durations, say, 2 minutes, 3 minutes. 4 minutes is a very, very common one you'll find in research, so 4 minutes of all-out exercise, 4 minutes of recovery. Repeat it again two to four times.

What's critical to understand here is these work when you're actually achieving a maximum in that time domain. So you can't do 4 minutes at 70%, rest for 4 minutes, and do that again. That's

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going to burn you some calories and has other benefits of just making you feel better today and some other stuff like that. But in terms of VO2 max, that's probably not the most efficient thing you can do.

So to summarize all of that stuff, spend a good amount of time at a lower intensity. That's going to drive efficiency, a common adaptation there, since it's going to be the highest activity you can do to maximize utilizing fat for fuel. You're still going to be burning, primarily, carbohydrates. Don't get that confused. But that's the best way to burn some fat.

So this is typically associated with higher metabolic efficiency, getting better at using fat as a fuel source, and things like that. It's also easy to recover from. It's not going to change your autonomic nervous system that much.

So you typically don't see big drops in HRV scores. We don't really see as much overtraining or nonfunctional overreaching, elevations in respiratory heart rate, other signs of hunger, fatigue, not wanting to train, things like that. Doesn't really happen when we spend time at lower intensities.

Higher intensities are phenomenal, really, really, really time-efficient. But they've got consequences as well. They're going to be entirely or mostly anaerobic, which is OK, too, because you'll still use the aerobic side of the equation to recover from that. So it's super important.

But there's a price to be paid there. People can run into problems, and you're more likely to see issues with those metrics I just described. If you're doing too much intensity too often, especially if you're combining this with a normal, stressful life.

So you're doing this kind of exercise. Then you're going right back into your day job. You're having difficult meetings, even if they're exciting and happy meetings. You're thinking hard. You're working. You're getting back and forth. And you're in a long, high stress environment all day. Really, really challenging on the system to be in that high of a stress at all times.

So other ways you can mitigate that, we can talk about those in future episodes. But just wanted to say, while high-intensity exercise is very time-efficient, it's not necessarily a free pass, either. Low-intensity is not a free pass, either. It's going to leave things on the table that you're missing.

So to round all that up, again, I would recommend a combination of lower-intensity, moderate-Intensity, and high-intensity training. The mode of the exercise in terms of what you choose-- bicycle, kettlebells, circuit training-- it's entirely up to you, spin class, whatever you'd like to do.

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Frequency can be as high or as low as you'd like. There are plenty of studies showing the higher-intensity stuff done two to three times per week can improve VO2 max. But you can also do the lower-intensity stuff every day or combination. So really, you can modify this based on your lifestyle and what's going on.

And finally, rest intervals, they're not incredibly applicable here. In fact, we've already baked them in. If you're not doing intervals, then there is no rest interval. If you are, we typically look for something like a 1 to 1 work-to-rest ratio, but you're welcome to do 2 to 1, 1 to 2, or any combination of that.

If you train appropriately-- and of course, you've got all the other factors like nutrition and sleep and stress management under control-- it's not unrealistic to expect a 30% to 50% improvement in VO2 max after 6 to 12 months. You'll find plenty of studies that land in that ballpark. The rate of increase obviously goes down as you become more and more trained.

Now, candidly, you don't have the ability to improve your VO2 max probably as much as you do something like your strength, but you can improve it significantly nonetheless. You will find plenty of studies showing even a 10% to 20% increase in highly trained individuals after a year.

In untrained folks, that probably takes about half that time, so 10% to 20% improvement in four to six months or so. So if you know where you're at right now, you train appropriately, fairly consistently, again, those are reasonable numbers to expect after a half a year or so of training. And as we understand it, the biggest limiting factor at this point is probably the time needed to fill the ventricles back up with blood.

I know we covered a lot of ground in this episode. And I hope you had as much fun listening to it as I did talking about it. But before we walk out of here, let's quickly recap what we discussed.

Most importantly, we talked about why you actually breathe, how you can pull a heart out of a living animal, and it can continue to beat on its own, and why your heart, unlike the rest of your muscles, never get sore. Along the way, of course, we talked about what your heart is, why that tissue is special and unique, and how it functions.

We talked specifically about your VO2 max, how to test that score, how to know where you are in that spectrum of good, great, terrible, elite, and then really, what to do about it at the end. In covering that stuff, we also gave some hints about things like CO2 tolerance, how that influences sleep and recovery, respiratory rate, HRV, and a number of other factors that are not directly but highly associated with overall cardiovascular health.

If you were of my opinion, when I first started my foray into exercise physiology, and you didn't really give cardiovascular health and performance the credit it deserved, I hope that I've

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changed your mind a little bit and warmed you up to it. If you're the opposite direction coming in, being a champion of the cardiovascular system, I hope I just gave music to your ears and let you double or triple down on your joy and biases towards the heart and its importance in overall health and physical performance.

Thank you for joining me for today's episode. Our goal is to share exciting scientific insight that helps you perform at your absolute best. If the show resonates with you and you want to help ensure this information remains free and accessible to anyone in the world, there are a few ways that you can support.

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My handle is drandygalpin on both platforms. Thank you for listening. And never forget, in the famous words of Bill Bowerman, "If you have a body, you're an athlete."

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