





Understanding the athlete's heart

Insights from echocardiography for life-changing diagnoses

Before they step onto the pitch, dive into the pool, or start pedaling, many of your favorite Olympians and professional players undergo critical cardiovascular screening. The main focus is helping these athletes maximize their performance, health, and safety. That includes preventing sudden cardiac death (SCD), the leading cause of death among young competitive athletes.¹ Crucial to this evaluation is distinguishing between normal cardiovascular adaptation to high-level exercise or 'athlete's heart' and true pathology, which places athletes at a higher risk for SCD with intense exercise. With so much at stake, some top sports cardiologists are relying on echocardiography for definitive answers.



One advocate is Prof. Sanjay Sharma, Professor of Cardiology at St. George's Hospital in London, and head of the largest sports cardiology unit in the UK. He is also a renowned expert in SCD in the young, athlete's heart, and heart muscle diseases. Professor Sharma's extensive research in this area has been pivotal in providing insights for echocardiographic assessment and critical clinical decisions.

St. George's Hospital has a busy echo lab and performs more than 30,000 echoes and 25,000 stress tests a year. Through the hospital's Inherited Cardiac Diseases and Sport Cardiology Unit, Dr. Sharma evaluates and treats hundreds of professional athletes, including Olympians, British Premier Football and Rugby League players, and members of the Lawn Tennis Association. When there's evidence of a potential cardiac abnormality during the screening process, many athletes will undergo an echo for evaluation and risk assessment.

We sat down with Prof. Sharma ahead of the Paris Olympics to learn more about identifying athlete's heart versus cardiomyopathies, and why echocardiography is a valuable tool in making a life-saving diagnosis.

What sparked your interest in sports cardiology and how has it evolved over the years?

Prof. Sharma: I became interested in sports cardiology in 1997 after learning about the occasional football player suddenly dying on the pitch from a condition called hypertrophic cardiomyopathy. It's a disorder that is characterized by abnormal thickening of the heart muscle. Exercising regularly can also cause the heart muscle to thicken, so I started asking myself, how does one tell the difference between physiological increases in left ventricular wall thickness and hypertrophic cardiomyopathy?

I knew that Saint George's Hospital had a very large cardiomyopathy center and I contacted Professor William McKenna about conducting research. I then made connections with the Lawn Tennis Association, triathlon, pentathlon, and it really grew from there.

You are medical director of the London Marathon and work with many professional athletes, including Olympians for Team GB and Premier League players. What are your roles?

Prof. Sharma: I've been the medical director for the London Marathon since 2017. My main job is really to enroll about 300 doctors of an appropriate skill mix to manage participants. We ensure that the race is safe and provide prompt care to get people running again or transferred to an urgent care unit.

I also work very heavily with the Football Association and look after about 14 football clubs, including 10 Premier League clubs. I've been working with Olympians for two to three decades, including athletes competing in Paris. Whether you take triathlon or Team GB rowing, they're managed by the English Institute of Sport.

My main job with all these sporting organizations is to ensure that we have identified athletes who may be at risk of an exercise-related sudden cardiac death. That means screening those hearts to make sure they do not have any overt electrical and structural abnormalities that could result in a cardiac arrest.

What is your main objective as a sports cardiologist?

Prof. Sharma: *The main role of sports cardiology is to identify an athlete who may be harbouring a potentially serious cardiac condition, and then to provide some form of safe exercise prescription, even if we cannot cure some of the problems. We try to find the underlying cause, treat it, and preferably cure it, like Wolff-Parkinson-White syndrome or anomalous coronary artery.*

If we cannot cure, it's our job to perform risk stratification tests to identify whether some athletes may be safe enough to continue competing, despite potentially serious conditions. In some cases, an athlete must retire because the condition is quite serious. In those people, we try to provide a safe exercise prescription, which they can continue to perform without risking their lives.

Cardiac sudden death is the leading cause of death in elite athletes. Can you provide more insights about the risk?

Prof. Sharma: *I'm pleased to inform you that sudden cardiac death in young sportsmen is relatively uncommon. If we look at the overall risk of sudden death is about one in 50,000. We know that males are more prone to death than females. For males specifically, it's about one in 37,000. Athletes of African or Afro-Caribbean origin are more prone to deaths compared to white athletes. For black males participating in British football, it's about one in 4,000. If we look at black males participating in basketball in the U.S., it's also around one in 4-5,000.²*

We also know that sports involving dynamic start-stop nature, such as basketball and football are particularly at risk.

What are some of the cardiovascular conditions that can be linked to deaths in competitive athletes?

Prof. Sharma: *Deaths in young sportspeople (under 35 years old) are due to a diverse spectrum of cardiovascular diseases, which could have an inherited congenital or an acquired basis. The most common structural abnormality implicated in exercise-related sudden cardiac death is cardiomyopathy. Hypertrophic cardiomyopathy is the leading cause of death in the United States, whereas arrhythmogenic cardiomyopathy is the leading cause in Italy.*

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Our own experience here at St. George's Hospital conducting autopsies on athletes who have succumbed during exercise, suggest that 40% of athletes actually do not have any underlying cause. When we investigate their relatives, such as their parents, we find that 40% of those individuals have an inherited electrical abnormality, such as Long QT syndrome or Brugada

syndrome. Other structural causes of death include anomalous coronary arteries and valvular heart diseases. All these structural abnormalities can often be identified with echocardiography.

Why is an athlete's heart different and how does that factor into assessment?

Prof. Sharma: *People who exercise intensively for about four hours a day undergo a constellation of structural and functional changes within the heart to permit the generation of a large cardiac output for a prolonged period. That means that if you go from rest to exercise, your cardiac output increases from 5 L/per minute to between 25 and 30 L/per minute. If you're only doing this for a few minutes, the heart doesn't have to adapt. But if you're doing it for hours and hours, the heart must grow to pump a large stroke volume per beat. The heart increases in thickness by about 10 to 20% and increases in cavity size by around 10%.*

We also find that our athletes have enhanced left ventricular filling, so indices of diastolic function are supranormal. What you find with athletes with a very big heart and a very slow heart rate is that the heart fills hugely during diastole. It means the heart doesn't have to move very much to pump an adequate stroke volume to maintain a resting cardiac output of 5 L/per minute. As a result, a significant proportion of our athletes have a baseline low left ventricular ejection fraction, which can sometimes be muddled up for an early form of dilated cardiomyopathy.

Consequently, when we're faced with an elite athlete with an enlarged heart that looks as if it's just slightly bigger than what we normally expect, our differential diagnosis is between physiological cardiac enlargement at an extreme or a cardiomyopathy.

You have published extensive research on the differentiation between athlete's heart and dilated cardiomyopathy. What should clinicians consider when making assessments?

Prof. Sharma: *It's important to remember that 50% of athletes, particularly males, have a left ventricular cavity size that exceeds the upper limits for the general population. Approximately 40% of male athletes have a basal right ventricle diameter (RVD1) that exceeds that of the general population. It's also important to remember that around 12% of endurance male athletes have a borderline low left ventricular ejection fraction. Although exercise causes left ventricular hypertrophy, it's very uncommon to see a left ventricular wall thickness of more than 14 mm in a white athlete and more than 16 mm in the black athletes.*

My advice is that when you see a big ventricle that looks a bit lazy, please don't jump to a diagnosis of dilated cardiomyopathy or arrhythmogenic cardiomyopathy. Similarly, if you see a left ventricular wall thickness of more than 14 mm in a white athlete or more than 16 mm in a black athlete, do not attribute that to physiological left ventricular hypertrophy. That is hypertrophic cardiomyopathy until proven otherwise.

An erroneous diagnosis can have very serious consequences. It could jeopardize a young life if they continue to compete, or it could cost an athlete physically, psychologically, and financially if they have to give up the sport. This is why it's important that assessments are done in a very systematic way in an expert setting.

How is echocardiography part of that assessment and what are the advantages?

Prof. Sharma: *Most deaths in sport are due to a structural abnormality. Echo is an invaluable tool in identifying these abnormalities.¹ We use echo to first ascertain whether someone's got physiological changes that are compatible with athletic training. It also helps us differentiate or diagnose hypertrophic cardiomyopathy, dilated cardiomyopathy, and arrhythmogenic cardiomyopathy. It can also assess the aortic root in someone who may have aortopathy or even visualize the origins of the coronary arteries on the short axis using the aorta.*

Why is stress echo a powerful tool in sports cardiology?

Prof. Sharma: *When we try to differentiate between physiological cardiac enlargement and dilated cardiomyopathy in athletes who have big ventricles with borderline low left ventricular ejection fractions, we put them through a plethora of investigations, including biomarkers, ECG, baseline echo, stress echo, and MRI scans. We found that stress echo was by far the most superior tool in differentiating between dilated cardiomyopathy and athlete's heart.²*

An increase in left ventricular ejection fraction by more than 10% from baseline to peak exercise, or a peak left ventricular ejection fraction above 63% are very good discriminators for athlete's heart. Therefore, if you can increase your left ventricular ejection fraction above 10% from baseline to peak or increase your ejection fraction above 63%, that would be more in favor of physiological adaptation and allow you to safely tell the athlete to continue.

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In addition to differentiating between athlete's heart and dilated cardiomyopathy, stress echo has a very important role in diagnosing obstructive coronary artery disease, ischemia from myocardial bridges or anomalous coronary arteries.² It can also identify dynamic left ventricular outflow tract obstruction in athletes who may have mild left ventricular hypertrophy with an elongated anterior mitral valve leaflet that may not show itself at baseline echo but demonstrate clear obstruction during peak exercise.

Putting athletes to the test

Focus on British Rower George Bourne



An echocardiogram and stress echo provided the answers professional rower George Bourne was hoping for. Prof. Sanjay Sharma was able to determine that the 26-year-old British rower, who was vying for the Paris Olympics, did not have a structural heart disease or other pathology that could possibly end his career.

“I came here to have my heart tested after some altitude training and getting sick with Covid. I was worried because I heard that other athletes on the team had real complications, and I was having similar symptoms to them,” Bourne explained.

Bourne trains several times a day, six days a week. He acknowledged that it’s a gruelling training schedule, but

says it was only after the echo screening at St. George’s that he could see the physical effects of intense exercise on his heart.

“Obviously we work hard, and we measure our heart rates, yet coming here to an environment like this and with all this technology, it’s really cool to know how everything [in your heart] is working. And it’s really reassuring to know I was getting the best tests possible,” says Bourne.

While Bourne was nervous about receiving the results, he was also grateful to have the comprehensive testing available, including stress echocardiography, to rule out serious conditions.

“After all the medical tests, Professor Sharma determined it was a reaction to Covid and gave me the ‘all clear’ to start rowing again. I was relieved, but if I hadn’t got the good news, it is still a much nicer way to find out here on this bed, rather than in a more dangerous environment,” he says.

Unfortunately, Bourne missed a spot on the Olympic Team after coming up short at the final hurdle in the Men’s Single Skulls at the World Rowing Final. But the determined athlete has no plans to stop competing. He is back on the water and training harder than ever (under the guidance of Prof. Sharma, of course).

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In addition to diagnostics, how can echocardiography be used to estimate risk for cardiac conditions?

Prof. Sharma: *Echo plays a greater role than just as a screening tool. It also provides important risk assessment in many conditions. For example, let's take hypertrophic cardiomyopathy. If we look at the risk factors for hypertrophic cardiomyopathy, these include the actual maximal left ventricular wall thickness, the presence or absence of dynamic left ventricular outflow tract obstruction, or the presence of a very large left atrial diameter. All these parameters can be incorporated into the ESC risk model for hypertrophic cardiomyopathy.*

If we take an athlete who may have a low ejection fraction, we know that individuals who have dilated cardiomyopathy who have very low ejection fraction tend to do worse than individuals who've got normal ejection fractions. We also know that individuals who've got aortic roots that are approaching 50 mm are more likely to rupture their aortic root or dissect compared to individuals with a lower aortic.

What are the benefits of echocardiography over other modalities?

Prof. Sharma: *The most obvious benefits of echocardiography over other modalities, such as an ECG or an exercise stress test, is that it identifies structural heart disease. Although an ECG is an invaluable tool for someone like me, as is an exercise stress test in identifying electrical faults and exercise-induced arrhythmias, the only way we can actually diagnose structural heart disease is through an imaging test. Echocardiography is probably the most practical and pragmatic imaging test we've got available to us around the world.*

What technology is advancing the echocardiography field and allowing for more confident care?

Prof. Sharma: *There have been several advances in the technology of echocardiography machines, including GE HealthCare's Vivid machines. There have been improvements in tissue harmonics, which means that we can characterize the endocardium of the left and right ventricle and make much better measurements of the left ventricular wall thickness, the left ventricular volumes.*

And hence, we can provide a more accurate estimation of the left ventricular ejection fraction. In this respect, the 3D technology of the Vivid machines allows us to give a much better estimation of the ejection fraction.

We've got speckle tracking that gives us information on the global left ventricular longitudinal strain. And then there is tissue Doppler, which allows us to ascertain diastolic function in a much better way that we used to be able to do 20 years ago.

Is Speckle Tracking Strain imaging important in your practice?

Prof. Sharma: *There is a lot of emphasis placed on ejection fractions and strain values. It's important to remember that there is a diverse spectrum of ejection fractions that one sees in an athlete, but in general, athletes have lower ejection fractions compared to non-athletes. Our male athletes usually have a left ventricular ejection fraction below 60%. It's very difficult to comment on strain because just like our endurance athletes have low left ventricular ejection fractions, a significant proportion of our athletes also have a low global left ventricular longitudinal strain.*

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I think we need to revisit the distribution of ejection fractions and also publish knowledge on a large number of athletes about the value and the cutoff values for a pathological global ventricular longitudinal strain.

Sports cardiology is evolving. What other areas of the field need to be addressed with more research?

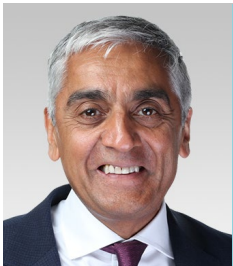
Prof. Sharma: *There are several knowledge gaps in sports cardiology that we need to cement. For example, I think we need more information about the impact of ethnicity on sport. Almost everything we know about sports cardiology is derived from the white population, but deaths in sport are more common in individuals of*

African or Afro-Caribbean origin. Asia probably contains the most athletes in the world, but we don't have data on that population.

We also need to look at the impact of exercise on the acceleration of cardiomyopathy. We know that arrhythmogenic cardiomyopathy is made worse by exercise, but we don't have that data for hypertrophic cardiomyopathy.

What's the best part about your job and working with elite athletes?

Prof. Sharma: *It's wonderful to work with individuals that push themselves to the limits and demonstrate to us that the human body is capable of so much more than we ever imagined 20 or 30 years ago. It's just great to see that aspiration and desire, not just to do well for themselves, but to make the nation proud. ■*



Prof. Sanjay Sharma is Professor of Cardiology and lead of the Inherited Cardiomyopathies and Sports Cardiology Unit at St. George's Hospital in London. He is also the Medical Director for Virgin London Marathon, Consultant Cardiologist for the charitable organization Cardiac Risk in the Young (CRY), and Cardiologist for the English Institute of Sport, British Rugby League, and the British Lawn Tennis Association. Prof. Sharma was also lead cardiologist of the London Olympics for several events.

With more than 500 publications, Prof. Sharma has an international reputation in cardiovascular adaptation in athletes, sudden cardiac death in the young, and heart muscle diseases. His research has been key in characterizing the impact of age, sex, and ethnicity on cardiovascular adaptation to exercise and in the identification of non-invasive methods of differentiating electrocardiographic and echocardiographic manifestations of cardiomyopathy and ion channel disorders from those representing 'athlete's heart'.

- 1 Wasfy MM, Hutter AM, Weiner RB. Sudden Cardiac Death in Athletes. *Methodist DeBakey Cardiovasc J.* 2016 Apr-Jun;12(2):76-80. doi: 10.14797/mdcj-12-2-76. PMID: 27486488; PMCID: PMC4969030.
- 2 Millar LM, Fanton Z, Finocchiaro G, Sanchez-Fernandez G, Dhutia H, Malhotra A, Merghani A, Papadakis M, Behr ER, Bunce N, Oxborough D, Reed M, O'Driscoll J, Tome Esteban MT, D'Silva A, Carr-White G, Webb J, Sharma R, Sharma S. Differentiation between athlete's heart and dilated cardiomyopathy in athletic individuals. *Heart.* 2020 Jul;106(14):1059-1065. doi: 10.1136/heartjnl-2019-316147. Epub 2020 Apr 27. PMID: 32341137.

Prof. Sanjay Sharma is a paid consultant for GE HealthCare and was compensated for his participation in this testimonial/case study. The statements by Prof. Sanjay Sharma described here are based on his own opinions and on results that were achieved in his unique setting. Since there is no "typical" hospital/clinical setting and many variables exist, i.e. hospital size, case mix, staff expertise, etc. there can be no guarantee that others will achieve the same results.

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