

# Starting Strength

## Heavy Lifting and Heart Health

by

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In September 2013, Dr. Jonathan Sullivan explained how the Valsalva Maneuver, a forceful attempted exhalation against a closed glottis, *does not* predispose lifters to hemorrhagic stroke, a potentially fatal event [1].

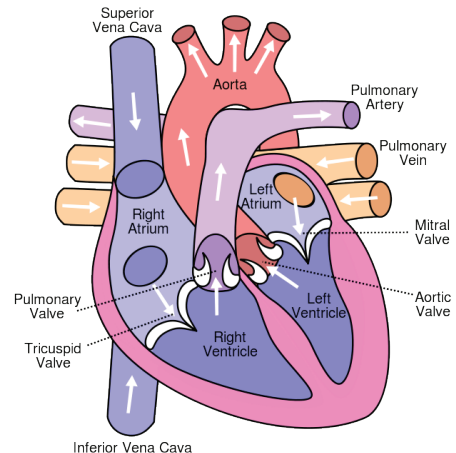
However, fears over barbell training and the Valsalva maneuver don't stop there. We frequently hear and answer people's concerns about resistance training and cardiovascular health in general, including claims that barbell training "doesn't do anything for the heart" and that "you'll give yourself a heart attack if you don't do some running, too." Since the heart is just a wee bit important to our health, and since no one is looking forward to their next heart attack, it's important to address these concerns.

### The Heart and Barbell Training

First, we have to understand a little about the heart and how it responds to barbell training.

Your heart has four chambers – two atria and two ventricles. The atria receive blood returning to the heart and the ventricles pump blood out to the rest of the body. The right ventricle pumps blood to the lungs for oxygenation, after which the blood flows to the left side of the heart. The left ventricle then pumps this oxygenated blood into the aorta and the entire body.

Each heartbeat is composed of two stages: systole and diastole. *Systole* describes the action of the ventricles squeezing to pump blood out of the heart, while *diastole* describes the relaxation of the ventricles, which draws in blood from the atria in preparation for the next contraction. This is why we normally represent blood pressure as two numbers (systolic and diastolic) with the systolic being the higher value. When I refer to "diastolic dysfunction," then,



Simple model of the heart including all four chambers along with the major veins, arteries, and valves discussed below. Shared through a creative commons license from Wikimedia Commons.

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I'm referring to a fault in the ventricle's ability to relax, expand, and fill with blood between cycles. For our purposes, "blood pressure" refers to the mean arterial pressure, the "average" blood pressure as measured in the peripheral arteries over one full cardiac cycle.

*Heart rate (HR)* describes how many times the heart beats in a minute and *stroke volume (SV)* describes how much blood the left ventricle pumps into circulation with each beat. *Cardiac output* is the product of HR and SV and describes how much blood the heart pumps out each minute. *Blood pressure* is equal to the cardiac output times the *systemic vascular resistance* (or *peripheral resistance*), so anything that increases HR, SV, or the resistance to flow in the body's blood vessels will increase blood pressure.

Before you begin training, your heart rate is maintained by natural pacemakers in the heart and is regulated by two primary control systems – the *sympathetic* and *parasympathetic nervous systems*. In this context, the sympathetic ("fight or flight") system ramps up heart rate and the parasympathetic ("eat, mate, and sleep") system slows it down.

Even before you start lifting, your brain will begin to stimulate the sympathetic nervous system. As you warm up, sensors in the muscles (*proprioceptors*), oxygen/CO<sub>2</sub> sensors (*chemoreceptors*), hormones, mineral concentrations in your blood, and other signals feed back to the sympathetic nervous system, raising heart rate to keep up with the body's demand for oxygen [2]. Your heart rate will fluctuate throughout a training session, rising during hard effort and falling during between-set rest periods, but if rests are kept to a reasonable length, it will remain elevated above normal for the entire session. A few enterprising lifters on the forums have used heart rate monitors during a standard novice linear progression and reported an average heart rate of around 120 BPM over a training session [3][4].

At the lowest loads (slow-moderate pace cardio, light prowler pushing, air squats, warmups), increased heart rate alone can handle the body's circulatory demands. As the effort increases, the muscles contract harder, increasing peripheral resistance. Since blood pressure is tied to both cardiac output and peripheral resistance, blood pressure can increase dramatically, and the left ventricle has to overcome this pressure to push blood out into circulation. This pressure is known as *afterload*. When the weight gets heavy enough that the lifter must hold his breath and brace down to protect the spine (the Valsalva maneuver), pressure in the thoracic cavity increases. This increases afterload even more. It also compresses the large veins that return blood back to the heart [5], thereby decreasing the *preload*, the blood available to the heart for pumping in systole. Under these conditions, studies have shown systolic blood pressures as high as 311 mmHg, about two and a half times normal [6].

How long the lifter holds the Valsalva is important. Dr. Sullivan's article went into detail here, but initially, there is only a rise in blood pressure and stroke volume while heart rate remains relatively steady. As the strain continues past about 5 seconds, the high thoracic pressure prevents venous blood from returning to the heart from the body (which you can see as bulging neck veins as the blood "backs up," unable to empty into the heart). With less blood to pump, stroke volume (how much blood is pumped out with each stroke) falls, which reduces total cardiac output and therefore blood pressure. This requires a compensatory increase in heart rate to maintain blood pressure near normal levels.

## **Heart Training: The Adaptation**

Based on that understanding, the traditional model of the heart's adaptation to training goes as follows:

In response to increased afterload, the heart's left ventricle (the main "pump" that pushes blood out to the body) will grow by adding sarcomeres *in parallel*, improving the heart's ability to

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pump harder. This increased thickness also helps to reinforce the heart against the greater pressure, and wrestlers (who show similar cardiac adaptations to those of strength trainees) indeed show lower levels of ventricular wall stress than age-matched controls [7].

In response to a demand for greater output (i.e. traditional “cardio”), the left ventricular cavity will expand, muscle fibers will be added *in series*, and diastolic function will improve, resulting in some of the common adaptations seen in aerobic athletes such as low resting heart rates [8].

A simplistic interpretation of these facts would suggest that strength training would strictly cause concentric hypertrophy (thicker ventricular walls) due to increased blood pressure, while aerobic training would strictly cause eccentric hypertrophy (widening of the ventricular cavity) due to a demand for increased cardiac output. This is, however, shortsighted, as endurance and resistance training both involve increased blood pressure and higher cardiac output [9]. As we’ll see, even thinking of “strength” and “cardio” training as opposite ends of a spectrum doesn’t quite hit the spot.

## **What About Cardio?**

Now that we know more about the basics of how the heart adapts, we’re in a better position to answer the question that every strength coach will inevitably face: “...but what should I do for cardio?”

A true novice (previously sedentary and starting a novice progression) will experience some aerobic adaptations in the same way that a true novice who starts rowing will see some improvements in strength. This shouldn’t really be a surprise – it’s the novice effect in action. The majority of early training sessions will be spent in warmups, and even the working weights will not require truly maximal efforts, so afterload will be low. Rests will be short (1-4 minutes, maybe even less for early warmups) and heart rate will remain elevated. Total duration and intensity will almost certainly meet or exceed the weekly exercise dose of “150 minutes of moderate physical activity” recommended by the American Heart Association [10]. In short, you’ll huff and puff more than enough to satisfy your doctor, and considering the added benefits of improved strength, it’s a far more productive use of time than the oft-recommended “brisk walking,” which simply cannot provide sufficient stress to elicit continued adaptation beyond the first few walks.

As the linear progression continues, rest periods for working sets will grow longer and afterload will increase as the lifter becomes capable of moving heavier loads and applying more stress to the system. However, cardiac output will remain high during and immediately after working sets, so someone who began completely sedentary and progressed through to the intermediate stage *will still be in better heart health than before they started*. In the same way, a couch potato who becomes a marathoner and then cuts their training volume in half will not somehow become less “fit” than when he began.

Previous aerobic athletes will see a significant loss of capacity from heavy strength training if they completely eliminate aerobic training. Aerobic adaptations fade relatively quickly [11] [12], and aerobic athletes spend even less time at higher heart rates than their novice peers because their heart rate recovers to baseline much more quickly. Still, all hope is not lost. For aerobic athletes trying to maintain or improve performance with concurrent strength training, research suggests that aerobically-trained athletes can dramatically reduce their training volume (by as much as  $\frac{2}{3}$ ) and still keep most of their aerobic adaptations by maintaining or increasing the intensity of their aerobic training sessions [13].

Nervous nellys will tell horror stories about their “friend-of-a-friend who ‘powerlifts’ and gets gassed walking across the parking lot.” When this actually happens, it is almost always the result of unrestricted weight gain, not the consequence of training. More often, it’s merely third-party

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retelling and tall-tale exaggeration. Many heavyweight, world-class powerlifters and strongmen show an impressive mixed (aerobic and anaerobic) capacity. For one example, see Chad Wesley Smith's impressive 1:14.4 500m row [14].

As a personal anecdote, I (Gotcher) was a cross-country runner and mixed-sport athlete from middle school until age 25. My best 5K run time was 16:52 (a 5:32 mile pace for 3.1 miles), and I could consistently run the Navy's fitness test (1.5 miles) in 8:40 or less. At that time, I struggled to deadlift 345#. After beginning strength training, I cut my "cardio" down as much as the Navy would allow. This meant 4 or 5 miles of running and one day of sports a week. After gaining 15 pounds of body weight and adding over 200 pounds to my lifting total, my PRT time had only slowed to 9:20, and my  $VO_{2max}$  remained in the "outstanding" category.

In short, getting stronger is not going to doom you to a life as a fat, gelatinous blob and spell death for your poor heart. If strength training is your first foray into fitness, a correctly executed novice progression will provide plenty of "cardio," and after you've developed a base of strength and muscle, you'll be in a better position to improve your sport and conditioning performance.

## **Metrics and Meaning: Left Ventricular Hypertrophy**

Now that we understand the heart's role in strength training and how it adapts, we can begin to address the concerns of some medical professionals, insurance adjusters, and other nefarious types. The first is the claim that strength training (without additional cardio) actually predisposes you to heart disease due to the concentric left ventricular hypertrophy we described above.

In order to understand where this concern comes from, we should be aware of a medical condition called *hypertrophic cardiomyopathy* (HCM). HCM results from genetic defects in the proteins of the heart, which typically causes excessive thickening (hypertrophy) of the wall between the ventricles (the interventricular septum). Along with fibrosis – a stiffening of the heart – this impairs both systolic and diastolic function. If the wall between the ventricles grows far enough into the left ventricle to block outflow, it can cause sudden cardiac death [15]. It's a serious condition and is the leading cause of sudden death in athletes younger than 35 [16].

The argument goes that because the thickening of the heart walls in HCM is a dangerous pathology, and because strength training thickens these walls (also known as "athlete's heart"), unchecked strength training therefore results in dangerous pathology. Some athletes (~2% of elite athletes in one study) will indeed develop a wall thickness outside of normal values (>12 mm) as a result of their training [17]. However, this process is different in many key ways. First, the thickened muscle does not contain genetically defective proteins as in HCM, so the pump continues to function normally [18]. And 15 mm of wall thickness marks the upper limit of athlete's heart whereas a diseased ventricle can grow to more than 30 mm in thickness [19]. In athlete's heart, the actual size of the left ventricular cavity typically stays the same or even grows larger, and  $VO_{2max}$  improves or stays the same, whereas the opposite occurs in a diseased heart [20].

Not only are they two distinctly different conditions, but the people sounding the siren about the dangers of left ventricular hypertrophy and weightlifting usually fail to provide the whole picture. As it turns out, *the athletes with the thickest heart walls are rowers, cyclists, and swimmers*, not weightlifters and other strength athletes. If concentric hypertrophy were dangerous in and of itself, it would be the endurance athletes who would be at risk. They also fail to mention that weightlifters and other strength athletes *do* show an increase in the size of the left ventricular cavity, a classically 'aerobic' adaptation [21].

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The athlete's heart is not dangerous just because it shares a single feature with HCM. Just like an elite athlete's resting heart rate might be considered dangerously low in a different population, the strength athlete's heart is not only harmless but the inevitable adaptation to stressful, productive training.

### **Heart Attacks, Cardiac Arrest, and Valsalva, Oh My!**

Another challenge to the safety of weightlifting is the possibility of a serious cardiac event, and to understand the argument, we have to understand the scope of the issue. Prolonged interruption of blood flow to the heart (or a segment of it) results in a *myocardial infarction* (also known as a heart attack). These attacks strike 735,000 people in the US each year (approximately .2% of the population), and about 16% of those afflicted will die from the event [22].

*Cardiac arrest* occurs when effective blood flow completely stops due to an abnormal heart rhythm, and is more rare but significantly more serious. In the US, approximately 359,400 people experience cardiac arrest every year, and the likelihood of survival for those receiving emergency medical services is quite low, around 10% [22]. The odds of being discharged from hospital without serious brain damage is even more dismal.

Considering the seriousness of the risk, a great deal of research has focused on the cause of these events. A review of cardiac events in Japan found heart attacks and sudden cardiac death were both significantly more likely to occur on the toilet and during sport/heavy work than at other times [23]. Although there are limitations to this study, it's enough to make us pause. Is the Valsalva maneuver going to cause you to have a heart attack?

### **Why Defecation and Deadlifts Aren't the Same Thing**

Research going back to the 1940s indicates a connection between cardiac events on the toilet and the Valsalva maneuver [24]. For this reason, hospitalized patients in cardiac wards will often be prescribed laxatives to minimize straining. It seems to make sense that if the Valsalva maneuver is responsible for heart attacks on the toilet, when we're not under load, it must certainly put us at risk under the bar. As it turns out, not all things that *seem* to make sense actually do.

First, we have the issue of time. In the gym, even a heavy single rep will rarely take more than 5 seconds to complete, and if the lifter takes a breath or two between reps, most of the cardiac stress will dissipate before the next Valsalva. During defecation, strain periods are often much longer. Halpern et. al did the shitty work of measuring the pressure, duration, and frequency of straining episodes during defecation in normal and constipated subjects. They found that even in normal subjects on a commode (the group that strained the least), each bathroom visit involved an average of 4.6 straining episodes, and 33% of these episodes involved strain lasting 6 seconds or greater. Constipated patients experienced 7.5 straining episodes per visit with 82% lasting at least 6 seconds (some as long as 20-25 seconds!) [25].

Second, we have the added factor of an external load. You might think that adding a load would exacerbate the effects of Valsalva on heart function, but that may not actually be the case depending on the exercise and conditions. A review of the Valsalva maneuver and resistance training found conflicting evidence: most of the studies they investigated showed intra-abdominal pressures were *higher during the Valsalva alone*, without resistance [26].

There is at least a plausible explanation: the rectus abdominis and obliques (the visible *abz*) assist in the Valsalva, but they also flex the spine. Though critical to a heavy lift, they can only contract

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to a certain degree before the cost (the abs causing spinal flexion, working against the extension needed to safely keep the weight over the mid-foot) outweighs the benefit (increased intra-abdominal pressure and spinal stability). In a sneeze, defecation, or other involuntary expulsion, there are no such compromises. In fact, if you're looking for a killer ab workout that leaves you sore for days, drink way too much tequila and spend a half-hour making offerings to the porcelain god before taking an involuntary nap (ask me how I know).

In short, the conditions between lifting weights and bedpan deaths are so distinct that the connection is tenuous and, without significant epidemiological evidence of risk (which we'll discuss later), it's not a substantiated fear.

## **Paradoxical Embolism and Relative Risk**

At a recent seminar, a health professional brought up two possible causes for why the Valsalva might not be worth the risk: paradoxical embolism and the QT interval (QTI). Not only do these each deserve a brief mention and explanation, but they both illustrate fundamental issues with the general conversation about risk in strength training.

Paradoxical embolism occurs when embolic material in the blood returns to the heart and skips directly from the right heart to the left heart (called a right-to-left shunt) without first passing through the lungs. This material is most often a piece of a blood clot, but it could also be leaked fat, air bubbles, amniotic fluid, even small foreign objects like talc and contaminants from injection drug use. From there, it proceeds into the systemic circulation, potentially blocking an artery supplying blood to the brain, a limb, or another organ. Paradoxical embolism is rare, but it might happen if you have an abnormal opening between the right and left chambers of your heart (e.g. *patent foramen ovale*). Because Valsalva increases the pressure in the right heart, it has been suggested in a few case studies that the Valsalva maneuver caused a paradoxical embolism by forcing material across these openings [27].

Being fearful of strength training due to the risk of paradoxical embolism is a good illustration of a greater problem: a failure to understand *relative risk*. When making the recommendation to avoid heavy weight training, you have to consider the likelihood of the undesirable outcome, the likelihood that the intervention will effectively reduce the risk, and the cost of the intervention.

*Likelihood:* Although exact numbers are not known, paradoxical embolism makes up a small proportion of total emboli [28]. The overall likelihood of death from paradoxical embolism in any case is vanishingly small.

*Risk Reduction:* Avoiding weight training is unlikely to *reduce* total risk for two reasons. First, we would still be at risk due to the use of Valsalva in daily living, as we are often required to move heavy loads, defecate, and have sex. Indeed, several case studies found sexual intercourse induced a paradoxical embolism and led to stroke [29], and I don't see many doctors hemming and hawing over the heart risks of hanky-panky. Second, were the embolic material to follow its ordinary path into the lung, it could cause a pulmonary embolism, an event which kills 50,000 to 200,000 people a year in the US [30]. A right-left shunt may change the autopsy report, but not the risk.

*Cost of the Intervention:* More than a quarter of the population has a patent foramen ovale from birth [31]. Telling at least a quarter of the population to avoid strength training or using the Valsalva

based on the data we have would be terrible medical advice considering the benefits of high intensity exercise in general and strength training in particular.

### QT Interval and the Meaning of the Measure

The QT Interval (QTI) is a reference to a portion of a standard electrocardiogram (a measure of the electrical activity of the heart). It measures the time necessary for the electric depolarization and repolarization of the ventricles. It is an important measure in clinical treatment that can be affected by a number of variables, including medication use, electrolyte concentrations, and genetic conditions. A lengthened QT interval is considered a risk factor for certain heart arrhythmias such as *Torsades de Pointes* and ventricular fibrillation (a lethal arrhythmia that the AED at your gym attempts to reverse – *defibrillation*, get it?). Because of this potential risk, the FDA requires all new medicines be tested for their effect on the QT interval.

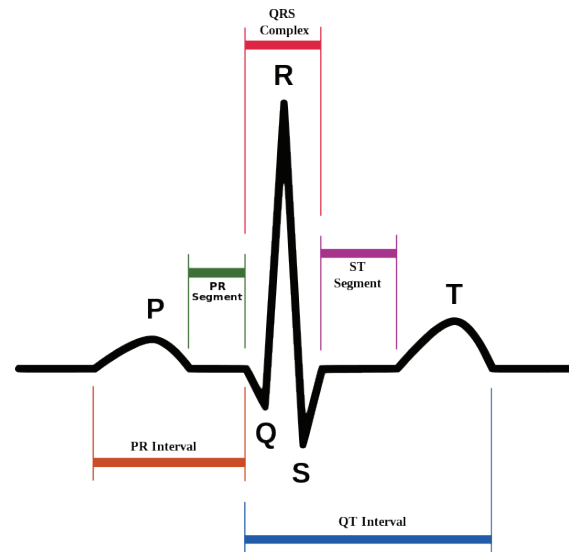
There has also been some interest in “QT dispersion” (QTD) as a potential risk factor, QTD being the difference between the maximum and minimum QT intervals over a period of time. Increased intrathoracic pressure caused by the Valsalva maneuver briefly increases the QT Interval and QTD, leading to breathless pronouncements like this one from Balbay et. al:

*“There are some conditions like constipation, severe coughing spells, nausea, vomiting, and carrying or lifting heavy objects that increase intrathoracic pressure and may increase QT dispersion. Therefore, all these conditions should be treated appropriately and **carrying or lifting heavy objects is forbidden, especially in patients with coronary artery disease**” [32].*

In that study, the authors evaluated the effect of a 10-second Valsalva maneuver on QTD in controls and cardiac patients. They found that the Valsalva held for 10 seconds transiently increased QTD above an *unvalidated* “risky” value of 80 milliseconds in the cardiac patients.

Heavy lifts rarely last 10 seconds, and the recommendation against resistance exercise for all patients with coronary artery disease is vague and overbroad, as the study only investigated those with *angina* (i.e, chest pain due to a loss of blood supply to heart muscle) and not other conditions. Moreover, the study found that among the healthy controls (55 years old on average), QTD remained well below 80 milliseconds even after a 10 second Valsalva.

More importantly, previous investigations which show a possible relationship between QTD and dangerous arrhythmia studied the effect of *extended increases in QTD over long periods*. Even if the metric is a reliable and meaningful risk indicator (and we’re still a little fuzzy on that one), we can’t compare the two. In the same way, it doesn’t make sense to compare the 150 BPM heart rate of someone in mid-marathon and someone lying in bed. One is normal, while the other probably signals a medical emergency.



Note the blue label representing the QT Interval. Created by Anthony Atkielski for the public domain.

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Finally, although the QTD *may* be significant and the QT interval certainly has clinical significance, we're not 100% sure about the chronic risk. In 2004, Montanez et al did a review of the available literature on the QT interval, mortality, and cardiovascular risk, coming to this conclusion:

*“There is no consistent association between prolonged QTc interval and total and cardiovascular mortality or morbidity, except perhaps in patients with prior cardiovascular disease. Even in this subgroup, however, the data are not entirely consistent” [33].*

This brings up a wider concern about health studies in general: “How valuable is this value?” We go to great lengths to find correlations between certain metrics and health risk and then define what is ‘normal.’ Once we’ve defined it, it’s human nature to want to stay in the lines. It runs counter to human nature to say: “It’s outside the range, and that’s okay,” or more often, “Let’s make a lifestyle fix and watch this play out.” Unfortunately, that is often exactly what’s called for, and failing to understand this results in over-prescription and cover-your-ass fear-mongering, leading to worse patient outcomes.

This same cautious, fearful-of-life approach lies behind the newly-popular “movement screens” and, sadly, the knee-jerk recommendations of many doctors to complaints of pain: “Doctor, my arm hurts when I try to lift weights.” “Then stop lifting weights.”

There are certainly risks not worth taking. But if a doctor tells you everything worth doing might send you to an early grave, either get a second opinion or just ignore it and live your life. We’re all ticking time bombs waiting for an errant car, lightning strike, or some other Act Of God to move us onto something else. Why even go through life, short or long, if every exciting achievement, every thrill, every moment that makes our heart race, causes us to pull back in guilt and fear?

## **Count the Bodies**

Epidemiology can tell us a lot about what these risks mean in real terms. If the Valsalva maneuver was a serious risk for cardiac events, you would expect CPR to be a regular occurrence at powerlifting meets (stocked as they often are with plenty of people who look like they might keel over from a heart attack at any minute, lifting or not). This doesn’t appear to be the case.

The available research indicates that there *is* a heightened risk of heart attacks and cardiac arrest during “vigorous exercise,” and without a careful reading, it could appear grim:

*“Although only a minority of cardiac events is [sic] triggered by heavy physical exertion, the relative risk of these events is increased nearly 80-fold when a sedentary individual performs vigorous physical exertion defined as 6 METs or more” [34].*

For perspective, 6 METS is equivalent to a “moderate” aerobics class, backpacking a 5% slope at 3 miles an hour, or cycling at 10 miles an hour [35].

*“...vigorous activity can also acutely and transiently increase the risk of SCA and acute myocardial infarction” [36].*

However, a more careful review reveals an entirely different story. We consistently find three observations reported in the literature:

1) In both healthy and at-risk patients, major cardiac events do occur, but they are exceptionally rare:

*“... primary cardiac arrest during vigorous activity is a rare occurrence in a clinically healthy population, with an annual incidence among exercisers in our population of .55 per 10,000 men...” [36].*



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*“A pooled analysis of 51,000 at-risk patients (those in cardiac rehab programs) found they experienced 1 cardiac arrest in 112,000 exercise hours, one acute heart attack per 300,000 exercise hours, and 1 fatality per 800,000 exercise hours” [38].*

2). Although transient risk is increased, intense exercise is heart-protective overall:

*“... even though intense physical activity may be one of the factors that can precipitate primary cardiac arrest, habitual participation in such activity is associated with an overall reduction in the risk of primary cardiac arrest” [34].*

3). The greatest risk is to be sedentary and to be suddenly stressed by the activities of life or the demands of a newly-started sport.

*“We propose that gradually increasing the level of habitual activity has the greatest potential to render exercise safe while improving all-cause mortality...” [37].*

Not surprisingly, this is how the novice linear progression is designed. We start the lifter at the load where they just begin to slow down, dialing in correct movement and beginning the process of strength development. The lifter progresses up to higher and higher weights gradually as the body adapts, the rate of that progression dictated by factors like age, training advancement, and recovery capacity. As the body adapts to these stresses, the risks of training decrease dramatically:

*“... However, regular physical exertion significantly attenuates these risks, decreasing the risk of MI with vigorous exertion to only 2-fold and sudden death with vigorous exertion to only 10-fold [Down from 80-fold in the sedentary]. Likewise, another potential trigger of MI is sexual activity, with the risk being increased by about 2.5-fold, whereas regular physical exertion seems to completely abolish this slightly increased risk associated with sexual activity” [38].*

When you look at the whole picture, without any evidence for increased health risks among strength trainees, sounding the alarm over the heart-risks of heavy lifting is like shouting “Fire!” when there’s no smoke.

## **So What?**

Before you make the decision on whether or not to strength train, you have to know the benefits and risks. The benefits are considerable:

Progressive strength training improves bone density in both the young and the old in a way that running and traditional light weight training can’t match [39] [40] [41]. It has been shown to improve glycemic control in patients with Type 2 diabetes [42] and improve quality of life and function in old age [43] [44]. Strength and muscle mass are consistently associated with lower overall mortality [45] and, to circle back to the topic of this article, they appear to be cardioprotective (heart-healthy) overall [46] [47]. For the vast majority of people, no magic pills or surgical interventions have the potential to make the kind of lifelong, positive improvements to your health that strength training can.

There are, indeed real risks. In some populations, they may not be worth the benefits. For instance, people with prominent aortic aneurysms or connective tissue diseases like Marfan’s Syndrome are at a significantly higher risk for aortic dissection (a medical emergency where the aorta tears under conditions of high pressure).

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That being said, the only way to be perfectly safe is to do nothing, ever – and we can never entirely predict when disaster will strike. In one collection of case studies where the authors recommend that at-risk individuals avoid heavy strain, two of the five events described occurred during weight training, one during manual labor, two while doing pushups, and *none of the victims showed any serious risk factors prior to the event* [48]. Sometimes, the best we can do is identify our risk factors and proceed with life the best way we can.

If you're in an at-risk population and want to strength train, consider the following steps to train as safely as possible:

1. Talk to your doctor about your condition, your intentions, and your options. Investigate your condition in detail. Be informed.
2. Communicate your concerns and at-risk status to your coach so that, if an event takes place, they can respond quickly and correctly to potentially save your life.
3. The majority of cardiac events are preceded, within a few days, by warning signs, things like unexplained shortness of breath and fatigue, sudden pain or numbness in a limb, and unexplained chest pain [22]. Don't ignore these, and remember that they may occur at any time, not just in the gym.
4. Titrate the weight up slowly, starting at weights lighter than you think you need to, and pay attention to good form. If you give your body time to adapt to increasing stress, you may be surprised how much stress it can adapt to and how strong you can get.
5. Beware of your surroundings. Even the most at-risk populations are far more likely to be injured from a slip, fall, collision, or plate drop at the gym than by a cardiac event [49].

Most importantly, and this is true well beyond the bounds of heart health and weight training: make your decisions based on the best information you have available, not the vague fears of the uninformed.

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